Fractures du rachis thoraco-lombaires traitées orthopédiquement chez le sujet de moins de 18 ans : évolution dans le plan coronal et sagittal : à propos de 48 cas
Audrey Angelliaume

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THESE

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FRACTURES DU RACHIS THORACO-LOMBAIRE TRAITEES ORTHOPEDIQUEMENT CHEZ LE SUJET DE MOINS DE 18 ANS. EVOLUTION DANS LES PLANS CORONAL ET SAGITTAL. A PROPOS DE 48 CAS.

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Introduction

Les fractures du rachis thoraco-lombaire sont rares en pédiatrie, moins de 4% des fractures de l’enfant (1-4) ; elles peuvent survenir de la naissance jusqu’à la fin de la croissance. Elles intéressent un squelette en croissance comprenant des zones cartilagineuses vertébrales non ossifiées, qui sont des zones de fragilité chez l’enfant. La part variable des différents éléments constituant la vertèbre (cartilage, os, moelle hématopoïétique) se modifie et les modes de faillite mécanique sont spécifiques à chaque âge pour un traumatisme équivalent. Les zones de vulnérabilité sont les cartilages neuro-centraux chez le tout petit, les cartilages de croissance des plateaux vertébraux chez l’enfant d’âge moyen et la jonction listel marginal-corps vertébral chez l’adolescent.

La croissance vertébrale est bien connue. Chaque corps vertébral compte deux zones de croissance : les physes des plateaux vertébraux qui permettent la croissance en hauteur du corps vertébral et les cartilages neuro-centraux qui unissent le corps vertébral aux pédicules et déterminent la taille du canal rachidien. La poursuite de la croissance du corps vertébral longitudinalement et dans le sens antéropostérieur dépend de points d’ossification secondaires qui apparaissent après la naissance. Cette croissance osseuse est responsable du phénomène de remodelage, spécifique à l’enfant, qui permet la « correction » de la déformation fracturaire après un traumatisme de la colonne vertébrale. La croissance rachidienne se prolonge durant la deuxième décennie de vie, avec un pic majeur au moment de la puberté, c’est pourquoi le traitement orthopédique des fractures du rachis thoraco-lombaire peut être proposé jusqu’à la fin de la croissance. Ce remodelage osseux peut être spectaculaire, en particulier dans le plan sagittal, cependant il demeure très difficile à anticiper. Plus encore, la déformation dans le plan coronal et son évolution, soumise aux mêmes capacités de remodelage, sont peu étudiées, et laissent une zone d’ombre importante sur les fractures tassements vertébrale générant une déformation dans le plan coronal.

A cela s’ajoute le risque de lésion des zones de croissance osseuses et le phénomène d’épiphyseiodèse post-traumatique qui pourrait également intervenir dans l’évolution des courbures post-traumatiques. L’épiphyseiodèse asymétrique dans le plan coronal, ou antérieure dans le plan sagittal, rendrait impossible le remodelage osseux dû à la croissance et, entraînerait des troubles de la statique rachidienne à type de scoliose dans le plan coronal et d’hypercypose dans le plan sagittal. Ainsi, il est admis qu’il existe un risque de cyphose et/ou de scoliose après une fracture du rachis thoraco-lombaire chez l’enfant et l’adolescent (1, 2, 5, 6).

Bien que la littérature se soit intéressée aux fractures du rachis thoraco-lombaire chez l’enfant, il y a peu d’articles qui rapportent un suivi à long terme dans le plan coronal et dans le plan sagittal de ces fractures traitées orthopédiquement en période de croissance.
Ce travail avait pour objectif initial l’étude des déformations post-traumatiques dans le plan coronal et leurs évolutions. Le risque « scoliogène » des fractures tassements traitées orthopédiquement devait être caractérisé avec, si possible, la mise en évidence de facteurs de risque en rapport (âge, maturité osseuse, niveau lésionnel, nombre de fractures…).

L’ensemble des données recueillies a permis l’analyse dans le plan coronal et dans le plan sagittal des déformations post-traumatiques et de leurs évolutions respectives. Si l’évolution dans le plan frontal et dans le plan sagittal semblent a priori liée, les auteurs ont choisi, dans un souci de clarté, de diviser l’analyse en trois parties, sous forme de trois articles scientifiques qui ont pour objectif de répondre aux questions suivantes :

- Quelle est l’évolution des déformations vertébrales post-traumatiques en période de croissance dans le plan frontal lors d’un traitement orthopédique ?
- Quel est l’impact des déformations vertébrales post-traumatiques en période de croissance sur l’équilibre sagittal du rachis lors d’un traitement orthopédique ?
- Quel est le lien entre la déformation initiale de la ou les vertèbre(s) fracturée(s) et la déformation finale de la colonne vertébrale ?

Les trois articles sont rapportés ad integrum, comme soumis aux éditeurs, au prix de certaines redondances, obligatoires dans la mesures où il s’agit de la même série de patients. Ils ont été délibérément laissés en anglais afin d’éviter des biais de traduction.
References

Part I: Post-trauma coronal spinal deformity after conservative treatment of thoracolumbar spinal fracture in children and adolescents.

Long-term results in 48 patients.

Introduction

Spine injuries in children are relatively rare and represent a small percentage of overall injuries to children (range 0.3-4%) (1-6), they are less common than in adults because of the greater mobility and elasticity of the pediatric spine and the smaller mass of the child’s body. The incidence of pediatric spine injuries has been reported to be 2-5% of all spine injuries (1) and occur in the thoracic or lumbar spine in 20-60% of injuries (4). Conservative treatment remains the most common treatment because children and adolescents have strong bones with excellent healing potential and usually reconstitute loss of vertebral height. Child spinal injuries are determined by the presence of growth plates. There are three groups of cartilage involved in vertebral growth, which allow the three dimensional growth of the vertebrae: the cartilage of the vertebral endplate, the neurocentral cartilage, and the ring apophysis. The cartilage of the vertebral endplate is responsible for the growth in height, like the growth plate of a long bone. In this way a traumatic separation of the vertebral endplate can account for abnormal growth because of premature epiphyseal fusion, in the manner described by Salter and Harris in long bones. This is one hypothesis to explain coronal spinal deformity after vertebral fracture. Post-trauma scoliosis are well known (1, 2, 7, 8) but, to our knowledge, there is no report which focuses on the coronal plane analysis after vertebral fracture. The aim of the present study was to evaluate radiological and clinical findings of coronal spinal balance, after conservative treatment of spinal fracture.

Materiel and Methods

A tricentric (2 hospitals, 3 departments of surgery) retrospective study was performed between 1996 and 2014. A computer search was initiated to find all patients under 18 years old who were hospitalized because of lumbar and/or thoracic vertebra fracture. Patients who underwent surgery during the first year after the accident, patients with neurological deficiency and patients with pre-existing scoliosis or spinal malformation were excluded. Fractures were classified according to AO spine fracture classification. All patients’ demographic data are summarized in table 1 (p.46).

Radiological measurements. All angular measurements were calculated by the method of Cobb (9) with Surgimap software and, were done by a single person to decrease measurement bias. On anteroposterior (AP) radiographs, local and regional Cobb angles (figure 1) were measured to quantify the deformity of each fractured vertebra and the deformity between vertebrae at the upper and lower limits of the curve respectively. The difference between final and initial local Cobb angles was called local deformity (LD); and the same difference
regarding regional Cobb angles was called regional deformity (RD). All measurements were done at the moment of the accident, authors using full spine radiograph with brace or cast and at last follow up with another full spine radiograph without contention; all radiograph were done in standing position. Scoliosis was defined according to the Scoliosis Research Society (SRS) by a Cobb angle above 10° and, vertebra rotation was asserted by Nash and Moe method.

**Analysis.** Several analyses were done according to the initial Risser grade, the number of vertebrae fractured (single fracture versus several stepped fractures) and the level of injury (thoracic versus lumbar). The first analysis was made in each group to characterize LD and RD. The second analysis was made to compare subgroups between them. The third analysis was to search for a correlation between sagittal and coronal deformity.

**Telephone interview.** The authors attempted to contact each patient included in the study. Patients were asked about their pain, their professional and physical activities, their consumption of painkillers, and the consequences of back pain in their daily activities.

**Statistical analysis.** Data were presented as mean and range. A Student t-test was performed to compare series. A difference of $P<0.05$ was regarded as a statistical significant difference. A Pearson test was used to look for a correlation between two series.

**Figure 1: Measurements of local and regional Cobb angle.**

![Image of measurements](image)

**Results**

The computer search found 320 patients in center 1 (2 departments of surgery) and 41 in center 2. After application of exclusion criteria and verification of patients’ files, 48 patients were included, 23 males and 25 females (figure 2, p.50), with a total of 84 vertebrae. There were 43 thoracic fractures (T4-T12) and 41 lumbar fractures (L1-L5) (Table 2, p.49); 89.6% are A1 fracture according to Magerl classification. All fractures were treated conservatively,
85.4% had a brace and 14.6% underwent reduction of the fracture and a cast. The mean age at the inclusion was 12.3 years old (range 2.6-17.1). Mean follow-up was 49 months (range 12-210) and 62% of cases had a Risser grade 4 or 5 at the end of the follow-up.

**Coronal deformity**

*Evolution of local deformity*

Mean initial local Cobb angle (ILCA) was 3.05° (range 0;19) and mean final local Cobb angle (FLCA) was 2.6° (range 0;10). LD decreased by -0.3° but without any statistical difference between FLCA and ILCA.

*Evolution of regional deformity*

Mean initial regional Cobb angle (IRCA) was 5.9° (range 1;14) and mean regional final Cobb angle (FRCA) was 8.4° (range 1;34). RD increased by +2.5° with a statistical (p=0.01) difference between FRCA and IRCA.

**Final scoliosis**

At the end of the follow-up, 11 patients (23%) had a scoliosis with a Cobb angle above 10° and associated rotation (figure 3); according to Nash and Moe method, rotation was characterized as + in 5 patients and as ++ in 6. Ten individuals had a Cobb angle at 20° or less and one had a Cobb angle of 20°. None of these 11 patients presented an IRCA above 10° on the initial AP radiograph.
Predictive factors of coronal deformity

The following statistical analyses were performed making subgroups to try to find predictive factors of coronal deformity.

Subgroup results: skeletal maturity, initial Risser grade

Patients were divided into two groups, group 1 with patients who had a Risser grade of 0, 1 or 2 and group 2 who had a Risser grade of 3, 4 or 5. Initially, both groups were comparable regarding the following data: age, gender, mechanism of injury, follow-up. In group 2, FRCA (mean=9.9°, range 1:19) was statistically higher that the IRCA (mean=5.8°, range 1:13) (p=0.03). On the contrary, there was no statistical difference between IRCA and FRCA in group 1. There was also no statistical difference between ILCA and FLCA in each group.
**Subgroup results: single fracture versus several staged fractures**

Two groups were made regarding the number of fractures, group U, with a single vertebra fracture and group S, with several staged fractures. Initially, both groups were comparable regarding the following data: age, gender, initial Risser grade, mechanism of injury and follow-up. In group U, FRCA (mean = 9.0°, range 1;34) was statistically higher than IRCA (mean=5.4°, range 1;14) (p=0.02). There was no statistical difference regarding IRCA and FRCA in group S. There was also no statistical difference between ILCA and FLCA in each group.

**Subgroup results: level of injury, thoracic versus lumbar**

Patients were divided into two groups regarding the level of injury, group T, thoracic vertebra fracture and group L, lumbar vertebra fracture. Initially, both groups were comparable regarding the following data: age, gender, mechanism of injury and follow up. But, there was a statistical difference between the two groups regarding the initial Risser grade (p=0.003); in group T 83.3% were Risser 0, 1 or 2 whereas in group L there were only 50% of Risser 0, 1 or 2. In group L, FRCA (mean=10.6°, range 1;34) was statistically higher than IRCA (mean=5.9°, range 1;13) (p=0.007). The same analysis in group T did not find any statistical difference. There was also no statistical difference between ILCA and FLCA in each group.

**Analyses between subgroups (Table 3)**
<table>
<thead>
<tr>
<th></th>
<th>ILCA</th>
<th>p</th>
<th>FLCA</th>
<th>p</th>
<th>IRCA</th>
<th>p</th>
<th>FRCA</th>
<th>p</th>
<th>LD</th>
<th>p</th>
<th>RD</th>
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<td><strong>Group 1:</strong></td>
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<tr>
<td>Risser 0, 1, 2</td>
<td>mean=3.2</td>
<td>p&gt;0.05</td>
<td>mean=2.2</td>
<td>p&gt;0.05</td>
<td>mean=6.7</td>
<td>p&gt;0.05</td>
<td>mean=7.9</td>
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<tr>
<td>Risser 3, 4, 5</td>
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<td>mean=5.8</td>
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<td>mean=9.9</td>
<td></td>
<td>mean=0.9</td>
<td></td>
<td>mean=3.8</td>
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<td>range 0;8</td>
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<td>range 1;13</td>
<td></td>
<td>range 1;19</td>
<td></td>
<td>range -4;5</td>
<td></td>
<td>range -4;12</td>
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<td>mean=2.2</td>
<td>p&gt;0.05</td>
<td>mean=5.6</td>
<td>p&gt;0.05</td>
<td>mean=6.2</td>
<td>p&gt;0.05</td>
<td>mean=-1.3</td>
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<td>range 1;14</td>
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<td>range 1;9</td>
<td></td>
<td>range-18;5</td>
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<td>range -12;9</td>
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<td>p&gt;0.05</td>
<td>mean=6</td>
<td>p&gt;0.05</td>
<td>mean=10.6</td>
<td>L&gt;T</td>
<td>mean=0.9</td>
<td>L&gt;T</td>
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<td>L&gt;T</td>
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<td>range 1;13</td>
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<td>range 1;34</td>
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<td></td>
<td>p=0.003</td>
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<tr>
<td><strong>Single fracture</strong></td>
<td>mean=3.5</td>
<td>p&gt;0.05</td>
<td>mean=3.1</td>
<td>p&gt;0.05</td>
<td>mean=5.3</td>
<td>p&gt;0.05</td>
<td>mean=9.0</td>
<td>p&gt;0.05</td>
<td>mean=0.7</td>
<td>p&gt;0.05</td>
<td>mean=3.6</td>
<td>p&gt;0.05</td>
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<td>(U)</td>
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<td>range 0;10</td>
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<td>range 1;34</td>
<td></td>
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<td>range -3;28</td>
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<tr>
<td><strong>Several fractures (S)</strong></td>
<td>mean=2.4</td>
<td>p&gt;0.05</td>
<td>mean=1.9</td>
<td>p&gt;0.05</td>
<td>mean=6.5</td>
<td>p&gt;0.05</td>
<td>mean=7.4</td>
<td>p&gt;0.05</td>
<td>mean=-1.1</td>
<td>p&gt;0.05</td>
<td>mean=0.9</td>
<td>p&gt;0.05</td>
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<td></td>
<td>range 0;8</td>
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<td>range 1;13</td>
<td></td>
<td>range 0;19</td>
<td></td>
<td>range -18;5</td>
<td></td>
<td>range -12;12</td>
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</table>
Correlation with sagittal deformity
Our analyses reported no statistical correlation between coronal and sagittal deformity at the last follow-up, nor in thoracic (r=-0.2) or lumbar area (r=0.05).

Others analyses

Clinical findings
Thirty-two patients were contacted by telephone, 81% reported back pain; but 87.5% revealed that back pain did not disturb their daily life and did not require any medication. Only one patient had stopped his professional activity, 31 of the 32 patients (97%) continued their professional or student life as before.

Mechanism of injury
The mechanism of injury was sport accidents in 50%, motor vehicle accidents (MVA) in 26% and falls (accidents or suicide attempts) in the last 23%.

Associated lesions
There were 42% of associated injuries in our series. Associated skull and long bone fractures were present in 33% of all cases, abdominal and/or thoracic injuries in 18.75%.

Discussion
The present series of 84 vertebrae confirms that conservative treatment of wedge compression fractures without neurological lesion ensures stabilization with moderate residual deformity. It confirms that patients with the most skeletal immaturity have greater bone remodeling power. But, in 11 cases restoration of height is inconclusive. Prevalence of scoliosis in our series is 23% whereas prevalence of idiopathic scoliosis (IS) is 0.47-15.3% (10-13), decreasing to 0.2-3% for a Cobb angle superior to 30° (11, 14). This result suggests that a fracture of the thoracolumbar spine is a risk factor of scoliosis. Other publications find scoliosis after conservative treatment of spinal fractures. Parisini et al. (1), in a series of 44 patients report four (9%) burst fractures (one T5, two L1 and one L3) treated conservatively with a mean final Cobb angle of 20°. Karlsson et al. (5) have two individuals (8.3%), in a series of 24 patients, who developed a scoliosis with 11° and 13° Cobb angles. Gnanenthiran et al. (15) find only one (2%) scoliosis, in a series with 50 patients, with a Cobb angle of 14°. The authors note that, in our series and in literature, post-traumatic scoliosis has small Cobb angles. The hypothesis to explain the incapacity to reconstitute loss of vertebral height in immature patients is the post-traumatic epiphysiodesis, as it may be encountered in long bones (16).

An interesting result in the analysis of the whole series is the improvement of local deformity whereas regional deformity worsens. The phenomenon of epiphysiodesis does not explain this result while the Hueter and Volkmann principle could. They describe how increased pressure on the end plate of bone retards growth (Hueter) and conversely, reduced pressure accelerates growth (Volkmann). Indeed, we can imagine that initial LD takes time to
heal and during this time an asymmetric growth occurs worsening the RD. Once the deformity reaches certain proportions, the effect of gravity on lever arm created continues to be a major deforming force. Using this principle, scoliotic curves have been reproduced on animal studies. Braun et al. (17) create an idiopathic type of deformity in goats by applying forces across the spine and find wedging of the vertebrae similar to that seen in scoliosis in humans. Similarly, Mente (18, 19) and Stokes (20, 21) in separate studies on rat tail models, not only create scoliosis, but also succeed in correcting it reversing the forces. Another explanation is the involvement of the intervertebral disc in the curve increase. Schlösser et al. (22) study the contribution of VB and intervertebral discs to three-dimensional (3-D) spinal deformity in adolescent idiopathic scoliosis (AIS); they conclude that the discs contribute more to 3-D deformity than the bony structure, because of the decreased stiffness of intervertebral fibrocartilage as compared to bony vertebrae. Modi et al. (23) in a series of 150 adolescent AIS conclude that wedging in disc and body increase with progression of the scoliosis and disc wedging is more profound in the lumbar area. This could explain why, in our series, lumbar fractures have a higher deformity rate than thoracic fractures. But, all of these studies are done on patients with AIS, not with post-trauma spinal deformity. Moreover, literature has varying results regarding disc lesion after VB fracture. Karlsson et al. (5) report no decreased in disc height at the fracture level but just a modification of nucleus pulposus intensity on the T2-weighted MRI. Whereas Kertulla et al. (24) find 57% of disc degeneration with a strong association between endplate damage and adjacent intervertebral disc degeneration. The hypothesis of intervertebral disc involvement in post-trauma spinal deformity has to be studied further to know its role in final spinal deformity.

The analysis regarding the level of injury shows that lumbar fractures are more responsible for coronal deformity than thoracic fractures; this could be explained by the fact that lumbar vertebrae are more mobile than thoracic vertebrae which are attached to the rib, making a rigid assembly less sensitive to deformity. Literature seems to report the same tendency as our series (1).

Our analysis according to initial Risser grades underscores that the group with a Risser grade 3 or above has FRCA higher than IRCA whereas there is no difference in the group with a Risser grade of 2 or below. This confirms that healing potential allows the restoration of the loss of vertebral height in immature patients. There are other publications which find that the cut-off point of a Risser grade 2 allows good restoration of vertebral wedging (2, 25).

The difference in group with a single fracture where FRCA is higher than IRCA is not found in the group with several fractures. Local deformity is higher in group with a single fracture than in group with several fractures. Authors make the hypothesis that when there is just one fracture, the deformity is more important whereas when several vertebrae are involved the energy of the trauma is absorbed in a greater area, making the deformity is smaller. Indeed, even if there is no statistical difference regarding ILCA between the two groups, ILCA is higher in group with unique fracture than in group with several fractures.

In our study, there is no correlation between coronal and sagittal deformity at the last follow-up; these results confirm that coronal analysis must be taken into account when the
treatment of vertebral fracture is decided. Indeed, to be concerned only with sagittal plane does not seem sufficient and satisfactory.

The weakness in our study is the retrospective design responsible for a heavy loss of information and patients. The strength of this study is the follow-up and the radiographic evaluation by the same individual at both baseline and at follow-up.

Our series finds a high rate of back pain, but a functional satisfying result, because patients do not feel limited in their daily life and need no pain-relief medication. We find varying values in literature: 12% to 57% of back pain after conservative treatment (1, 5, 24, 26). The rate of back pain in adolescents without back injury is high too: 7% to 70% (27-29) depending on the definition of pain and study design; this makes the interpretation of our rate difficult because back pain is multifactorial and it is difficult to identify the precise origin. Initially, authors wanted to search a correlation between coronal deformity and back pain but, because of the high rate found, it was not possible to conclude.

Our series finds the same common mechanisms of injury as in literature (2, 6, 30-32): sports accidents, MVA, pedestrian-vehicle accidents, and falls with varying proportions according to publications.

Values concerning associated lesions are similar with those of literature: 42% to 65% of patients have associated injuries with 5% to 50% of skull or long bone fractures and 20% to 37% of visceral (thoracic and/or abdominal) injures (3, 7, 32).

Conclusion

There is a higher prevalence of scoliosis in the population which sustains thoracolumbar spinal fractures. Risser grade 3 or above, lumbar fractures and single fractures seem to be responsible for more severe coronal deformation.
References

Part II: Thoracolumbar spinal fracture treated conservatively in children and adolescents, outcomes in sagittal plane
Long term follow up in 48 patients.

Introduction

Spine injuries in children are relatively rare and represent a small percentage of overall injuries in children (range 0.3-4%)(1-6), there are less common than in adults because of the greater mobility and elasticity of the pediatric spine and the smaller mass of the child’s body. The incidence of the pediatric spine injuries has been reported to be 2-5% of all spine injuries (1) and occur in the thoracic or lumbar spine from 20% to 60%(4). Conservative treatment remains the most common because children and adolescents have strong bones with excellent healing potential and usually reconstitute loss of vertebral height. Although spine injuries in children have received an increasing coverage in the literature, there are, to our knowledge, few reports which focusing on the post-trauma sagittal balance whereas it is associated with pain, neurologic symptoms and poor quality of life. Adjacent anatomical regions of the spine and sacro-pelvis are interdependent (7), and their relationship results in a stable and balanced posture (8, 9). Therefore, it would be logical that post-trauma local kyphosis (LK) bring hyperlordosis and maybe modify pelvic parameters as adaptive behaviors, as shown in aging spine. The aim of the present study is to confirm that vertebral fractures are responsible for LK and to investigate the sagittal balance looking at compensation mechanisms as increased lumbar lordosis and modification of pelvic parameters.

Materials and Methods

A tricentric (2 hospitals, 3 departments of surgery) retrospective study was performed between 1996 and 2014. An informatics search was initiated to find all patients under 18 years old who were hospitalized because of lumbar and/or thoracic vertebra fracture. Patients who underwent surgery during the first year after the accident, patients with neurologic deficiency and patient with pre-existing scoliosis or spinal malformation were excluded. Fractures were classified according to Magerl classification. All patients’ demographic data are summarized in table 1 (p.46).

**Radiological measurements (figure 1 and 2).** All angular measurements were calculated by a modification of the Cobb technique (10) with Surgimap software, there were done by a single person to decrease measurements bias. On lateral plain radiographs, LK was measured to quantify the deformity of each fractured vertebra; thoracic kyphosis (T4-T12) and L1-L5 lumbar lordosis were measured to quantify the regional deformity (RD) after thoracic and lumbar fracture respectively. The L3-L5 lumbar lordosis and pelvic parameters (pelvic tilt (PT), sacral slope (SS), pelvic incidence (PI)) were measured to quantify adaptive spinal and pelvic behaviors. At last follow up, the C7 plumbline was measured to characterize the global
sagittal balance. The measurement was assumed positive if directed forwards and negative if directed backwards. All measurements were done at the moment of the accident, authors using full spine radiograph with brace or cast and at last follow up with another full spine radiograph without contention; all radiograph were done in standing position.

**Analysis.** First analyses were done to search an evolution between time at fracture and last follow up looking at all the measurements. Second analyses were done according to the initial Risser grade to know if evolution was different according to skeletal maturity. Last analyses were done according to the initial LK to search if higher initial LK was responsible for higher RD and modification of pelvic parameters and C7 plumbline.

**Telephone interview.** Authors attempted to contact each patient included in the study. Patients were asked about their pain, their professional and sport activities, and their painkillers’ consumption.

**Statistical analysis.** Data are presented as mean and range. A Student t test was performed to compare series. A difference of P<0.05 was regarded as a statistical significant difference.

**Figure 4:** Measurements of local kyphosis (LK), T4-T12 kyphosis and L1-L5 lordosis and C7 plumbline.

**Figure 5:** Measurements of L3-L5 lordosis and all pelvic parameters (PI, PT and SS).
Results

The informatics search found 320 patients in center 1 (2 departments of surgery) and 41 in center 2. After application of exclusion criteria and verification of patients’ files, 48 patients were included (figure 2, p.50), 23 males and 25 females. There were a total of 84 vertebrae, 43 thoracic fractures (T4-T12) and 41 lumbar fractures (L1-L5) (Table 2, p.49); 89.6% are A1 fracture according to Magerl classification. All fractures were treated conservatively, 85.4% had a brace and 14.6% underwent reduction of the fracture and a cast. Mean age at the inclusion was 12.3 years old (range 2.6-17.1). Mean follow up was 49 months (range 12-210) and, 62% had a Risser grade 4 or 5 at the end of the follow up.

Sagittal deformity, global analysis: thoracic fracture (T1-T12) (Table 3), lumbar fracture (L1-L5) (Table 4).
Initially, the two groups were comparable regarding the following data: age, gender, initial Risser grade, mechanism of injury and follow up. These analyses confirmed the remodeling power of young person and reported a statistically increased lordosis (L1-L5 and L3-L5) after lumbar fracture.

Table 3: Evolution of sagittal parameters between time at accident and last follow up, thoracic fracture.

<table>
<thead>
<tr>
<th></th>
<th>Local Kyphosis</th>
<th>Thoracic Kyphosis</th>
<th>L1-L5 Lordosis</th>
<th>Pelvic Tilt</th>
<th>Sacral Slope</th>
<th>Pelvic Incidence</th>
<th>C7 Plumb line (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial (I)</td>
<td>11.6 (1;22)</td>
<td>35.8 (14;65)</td>
<td>34.9 (21;56)</td>
<td>10.1 (1;22)</td>
<td>37 (18;65)</td>
<td>47.9 (32;88)</td>
<td>-2.5 (-38.6;38.1)</td>
</tr>
<tr>
<td>Final (F)</td>
<td>8.2 (0;18)</td>
<td>39.6 (14;66)</td>
<td>34.2 (20;41)</td>
<td>9 (1;22)</td>
<td>36.1 (15;57)</td>
<td>46.3 (31;69)</td>
<td>-3.6 (-36.1;49)</td>
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<td>p</td>
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<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
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Table 4: Evolution of sagittal parameters between time at accident and last follow up, lumbar fracture.

<table>
<thead>
<tr>
<th></th>
<th>Local Kyphosis</th>
<th>L1-L5 Lordosis</th>
<th>L3-L5 Lordosis</th>
<th>Pelvic Tilt</th>
<th>Sacral Slope</th>
<th>Pelvic Incidence</th>
<th>C7 Plumb line (mm)</th>
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<td>Initial (I)</td>
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<td>27,3 (14;43)</td>
<td>28,6 (16;44)</td>
<td>12,3 (1;28)</td>
<td>36,4 (24;47)</td>
<td>49,2 (37;66)</td>
<td>-6,8 (-53,9;40,6)</td>
</tr>
<tr>
<td>Final (F)</td>
<td>8,7 (0,23)</td>
<td>37,6 (17;64)</td>
<td>37,6 (27;56)</td>
<td>9,5 (1;30)</td>
<td>40,1 (28;56)</td>
<td>46,9 (20;62)</td>
<td>-3,8 (-18,25;12)</td>
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<td>(p)</td>
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Sagittal deformity: Analyses according to skeletal maturity, initial Risser grade (Tables 5, 6, 7 and 8). Initially, both groups were comparable regarding the following data: age, gender, mechanism of injury and follow-up. Results are the same than in the analysis of the whole series: it confirmed the remodeling power of young person and found an increased lordosis after lumbar fracture.

All analyses report no modification of C7 plumbline and pelvic parameters at follow up except the increased SS after lumbar fracture in group with Risser grade at 2 or less.

Table 5: Results of subgroup Risser grade 0, 1 and 2, thoracic fractures.

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<th>Local Kyphosis</th>
<th>Kyphosis</th>
<th>L1-L5 Lordosis</th>
<th>L1-L5 Lordosis</th>
<th>Pelvic Tilt</th>
<th>Sacral Slope</th>
<th>Pelvic Incidence</th>
<th>C7 Plumb line (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial (I)</td>
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<td>34,5(14;60)</td>
<td>35,9 (25;56)</td>
<td>10,3 (1;28)</td>
<td>38,1 (31;65)</td>
<td>47,1 (34;88)</td>
<td>-1,2 (-38,6;38,1)</td>
<td></td>
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<tr>
<td>Final (F)</td>
<td>7,9 (1;18)</td>
<td>38,3(14;66)</td>
<td>34,6 (26;41)</td>
<td>6,8 (1;16)</td>
<td>36,6 (17;47)</td>
<td>45,8 (31;62)</td>
<td>-3,4 (-45,4;49)</td>
<td></td>
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<td>(p)</td>
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<td>(I&gt;F)</td>
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\(p>0,05\)          |                |           |                |               |             |              |                  |                   |
Table 6: Results of subgroup Risser grade 3, 4 and 5, thoracic fractures.

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<thead>
<tr>
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<th>Local Kyphosis</th>
<th>Kyphosis</th>
<th>L1-L5 Lordosis</th>
<th>Pelvic Tilt</th>
<th>Sacral Slope</th>
<th>Pelvic Incidence</th>
<th>C7 Plumb line (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Initial (I)</strong></td>
<td>9,8 (6;20)</td>
<td>41,3 (28;65)</td>
<td>30,7 (21;44)</td>
<td>10,5 (3;18)</td>
<td>34 (18;56)</td>
<td>49,8 (22;65)</td>
<td>-8,65 (-11,4;-5,9)</td>
</tr>
<tr>
<td><strong>Final (F)</strong></td>
<td>9,3 (5;17)</td>
<td>43 (37;53)</td>
<td>32 (20;39)</td>
<td>11,3 (2;17)</td>
<td>33,7 (15;57)</td>
<td>48,7 (30;69)</td>
<td>-4,6 (-31,5;19,3)</td>
</tr>
<tr>
<td><strong>p</strong></td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
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</table>

Comparison according to initial LK (Tables 9 and 10). Initial LK was statistically different whereas at last follow up there was no difference regarding final RD, PP and C7 plumbline.

Table 7: Results of subgroup Risser grade 0, 1 and 2, lumbar fractures.

<table>
<thead>
<tr>
<th></th>
<th>Local Kyphosis</th>
<th>L1-L5 Lordosis</th>
<th>L3-L5 Lordosis</th>
<th>Pelvic Tilt</th>
<th>Sacral Slope</th>
<th>Pelvic Incidence</th>
<th>C7 Plumb line (mm)</th>
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<tbody>
<tr>
<td><strong>Initial (I)</strong></td>
<td>13,5 (4;28)</td>
<td>26,8 (14;43)</td>
<td>26,6 (16;39)</td>
<td>10,3 (1;28)</td>
<td>34,9 (24;42)</td>
<td>46,2 (36;66)</td>
<td>-7,8 (-18,2;-2,1)</td>
</tr>
<tr>
<td><strong>Final (F)</strong></td>
<td>10,2 (1;23)</td>
<td>35,1 (17;52)</td>
<td>38,9 (29;56)</td>
<td>6,8 (1;16)</td>
<td>42,3 (28;56)</td>
<td>47,8 (20;62)</td>
<td>-0,1 (-5;5,3)</td>
</tr>
<tr>
<td><strong>p</strong></td>
<td>p&gt;0,05</td>
<td>F&gt;I p=0,05</td>
<td>F&gt;I p=0,002</td>
<td>p&gt;0,05</td>
<td>F&gt;I p=0,02</td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
</tr>
</tbody>
</table>

Table 8: Results of subgroup Risser grade 3, 4 and 5, lumbar fractures.

<table>
<thead>
<tr>
<th></th>
<th>Local Kyphosis</th>
<th>L1-L5 Lordosis</th>
<th>L3-L5 Lordosis</th>
<th>Pelvic Tilt</th>
<th>Sacral Slope</th>
<th>Pelvic Incidence</th>
<th>C7 Plumb line (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Initial (I)</strong></td>
<td>8,6 (1;29)</td>
<td>28,4 (25;31)</td>
<td>28,7 (22;44)</td>
<td>16,4 (3;24)</td>
<td>39 (28;47)</td>
<td>55,2 (39;65)</td>
<td>-5,7 (-53,9;40,6)</td>
</tr>
<tr>
<td><strong>Final (F)</strong></td>
<td>6,9 (1;17)</td>
<td>42,3 (26;64)</td>
<td>34,8 (27;46)</td>
<td>13,7 (6;30)</td>
<td>36,5 (30;41)</td>
<td>46,7 (35;61)</td>
<td>-7,5 (-18,2;12)</td>
</tr>
<tr>
<td><strong>p</strong></td>
<td>p&gt;0,05</td>
<td>F&gt;I p=0,05</td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
<td>p&gt;0,05</td>
</tr>
</tbody>
</table>
Tables 9: Comparison according to initial LK, thoracic fracture.

<table>
<thead>
<tr>
<th></th>
<th>LK</th>
<th>FK</th>
<th>Final PI</th>
<th>Final PT</th>
<th>Final SS</th>
<th>Final C7 plumpline (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>group 1</td>
<td>mean=7</td>
<td>mean=41</td>
<td>mean=46,6</td>
<td>mean=10</td>
<td>mean=36,6</td>
<td>mean=-3,2</td>
</tr>
<tr>
<td></td>
<td>range 1;10</td>
<td>range 26;53</td>
<td>range 31;68</td>
<td>range 1;17</td>
<td>range 17;57</td>
<td>range -45,4;29</td>
</tr>
<tr>
<td>group 2</td>
<td>mean=15,7</td>
<td>mean=40,4</td>
<td>mean=46</td>
<td>mean=8,8</td>
<td>mean=37,8</td>
<td>mean=-4,2</td>
</tr>
<tr>
<td></td>
<td>range 12;22</td>
<td>range 24;65</td>
<td>range 30;61</td>
<td>range 1;22</td>
<td>range 29;47</td>
<td>range -36,1;49</td>
</tr>
</tbody>
</table>

| p     | p<0,05 | p>0,05 | p>0,05 | p>0,05 | p>0,05 | p>0,05 |

Tables 10: Comparison according to initial LK, lumbar fracture.

<table>
<thead>
<tr>
<th></th>
<th>LK</th>
<th>FL</th>
<th>Final PI</th>
<th>Final PT</th>
<th>Final SS</th>
<th>Final C7 plumpline (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>group 1</td>
<td>mean=5,5</td>
<td>mean=37,3</td>
<td>mean=51,3</td>
<td>mean=10</td>
<td>mean=39,7</td>
<td>mean=-5,4</td>
</tr>
<tr>
<td></td>
<td>range 1;9</td>
<td>range 27;50</td>
<td>range 35;62</td>
<td>range 1;22</td>
<td>range 30;3</td>
<td>range -15;5,3</td>
</tr>
<tr>
<td>group 2</td>
<td>mean=16,6</td>
<td>mean=37,8</td>
<td>mean=43,1</td>
<td>mean=9,1</td>
<td>mean=39,8</td>
<td>mean=-1,9</td>
</tr>
<tr>
<td></td>
<td>range 10;29</td>
<td>range 29;56</td>
<td>range 20;56</td>
<td>range 2;30</td>
<td>range 28;56</td>
<td>range -18,3;12</td>
</tr>
</tbody>
</table>

| p     | p<0,05 | p>0,05 | p>0,05 | p>0,05 | p>0,05 | p>0,05 |

Other analyses

Clinical findings
Thirty-two patients were contacted by telephone in which 81% reported back pain; but 87.5% revealed that back pain did not disturb their daily life and did not require any medication. Only one patient had stopped his professional activity, 31 of the 32 patients (97%) continued their professional or student life as before.

Mechanism of injury
The mechanism of injury was 50% sport accidents, 26% motor vehicle accidents (MVA) and 23% falls (accidents or suicide attempts).

Associated lesions
There were 42% associated injuries in our series. Associated skull and long bone fractures were present in 33% of all cases, abdominal and/or thoracic injuries in 18.75%.
Discussion

All analyses report no sagittal imbalance at follow up. Measurements of both C7 plumbline and pelvic parameters show not difference between the moment of the fracture and the last follow up. Our results confirm that young patients have a great growth potential to correct post trauma LK and therefore, that conservative treatment is a treatment of choice of wedge compression thoracolumbar fractures.

The weakness in our study is the retrospective design responsible for heavy loss of information and patients. Measurement of kyphosis suffers from quality of radiograph because of pulmonary interpositions; to decrease the bias authors choose to measure T4-T12 kyphosis. The strength of this study is the follow-up, the radiographic evaluation by the same individual at both baseline and follow-up. Authors do not use absolute values because there are various values of kyphosis and lordosis in the general population (11); but these values are stable during childhood (9, 12, 13) allowing comparison between initial and final values. Moreover, thoracic kyphosis is similar for males and females until 40 years old (14) and this is why our analyses are not done according to gender. Authors choose to use L3-L5 lumbar lordosis to search hyperlordosis as a compensation mechanism of post-fracture LK. Indeed, it was not possible to use the L1-L5 lordosis because, in our series, there are too many L1 and L2 fractures with a loss of anterior vertebral height which underestimates lordosis. Moreover, 70% of L1-L5 lumbar lordosis is below L3 (15).

Thoracic fractures are responsible for eight degrees of LK at the end without modification of regional kyphosis, lordosis, pelvic parameters and C7 plumbline. This means that a LK at 8° or less in thoracic spine has no consequence on sagittal spinopelvic balance in young person.

Results regarding lumbar fractures reveal that LK decreases but nine degrees remain. Moreover, the difference between initial and final LK is statistically significant in the thoracic area whereas it is not in the lumbar one. Authors make the hypothesis that healing potential is higher in thoracic spine because compressive forces are smaller than in lumbar spine. Indeed, lumbar vertebrae support the entire weight of the body; Roaf (16) has proposed a law to explain that. Studying Scheuermann’s disease he describes a vicious cycle regarding the progression of kyphosis. According to it, a minimal wedging of the vertebrae would produce abnormal compressive force in the vertebral end plate, which would increase the wedging, as per the Huter and Volkman’s law, and thus produce further abnormal forces.

The nine degrees of LK which remain in lumbar area are not responsible for loss of lordosis because L1-L5 lordosis is equal at L1-L3 lordosis. Measurement of LK reflects the wedging of bony structure whereas L1-L5 and L3-L5 lordosis include both bony and soft structures. This result suggests that other structures than bone are involved in spinal adaptive behavior. Intervertebral discs have probably a role in the evolution of spinal curve, like in scoliosis and adult spine, but it is not described in pediatric population. Modi et al. (17) in a series of 150 adolescent idiopathic scoliosis (AIS) conclude that disc wedging and body are increasing with progression on scoliosis and, disc wedging is more profound in lumbar area.
This result could explain why in our series lumbar fractures have a significant increase in lordosis whereas thoracic curvatures are stable. Schlösser et al. (18) study the contribution of VB and intervertebral discs to the three-dimensional (3-D) spinal deformity in AIS; they conclude that discs contribute more to 3-D deformity than the bony structure, because of the decreased stiffness of intervertebral fibrocartilage as compared to bony vertebrae. Analyses according to the values of initial LK confirm that other structures than bone are involved in the final RD because no differences are found regarding final RD, pelvic parameters and C7 plumbline between the two groups whereas initially the two groups have a statistical difference concerning the initial bony LD.

It remains less than ten degrees in both thoracic and lumbar area but regional curvature is modified only in lumbar one. To explain this, authors suggest that lumbar spine is more mobile than thoracic because of the ribcage. The greater mobility of lumbar vertebrae makes them probably more sensitive to deformity.

Our analyses find no modification in pelvic parameters at follow up except the SS in lumbar fracture when children have a Risser grade 2 or less. The absence of pelvic parameters’ modification, particularly the PT, means that adaptive mechanisms after vertebral fracture in children only concern vertebral column and not pelvis. The increased SS in lumbar fracture is explained by the increased lumbar lordosis. Surprisingly this result is only found in patients with Risser grade 2 or less. Our results regarding sagittal balance (PP, PI, SS, Thoracic kyphosis, Lumbar lordosis) are similar to those in the literature in general population (7, 19).

Our series find a high rate of back pain, but a functional satisfying result, because patients do not feel limited in their daily life and need no pain medication. There are varying values in literature, 12% to 57% of back pain after conservative treatment (1, 5, 20, 21). The rate of back pain in adolescents without back injury is high too: 7% to 70% (21-23) depends on the definition of pain and study design; that’s makes difficult the interpretation of our rate because back pain is multifactorial and it’s difficult to identify the precise origin. Initially, authors wanted to search a correlation between sagittal deformity and back pain but, because of the high rate found, it was not possible to conclude.

Our series find the same common mechanism of injury as in the literature (2, 6, 25-27): sport accident, MVA, pedestrian-vehicle accidents, and falls with varying proportions according to publications.

Values concerning associated lesion are similar with those of the literature: 42% to 65% of patients have associated injuries with 5% to 50% of skull or long bones fracture and 20% to 37% of visceral (thoracic and/or abdominal) injuries (3, 26, 28).
Conclusion

Our study confirms that young patients have a great growth potential to correct post trauma LK and report no sagittal imbalance at follow up. Lumbar fractures seem to be responsible for more adaptive responses than thoracic ones. Our results suggest that other structures than bone are involved in spinal adaptive behavior; intervertebral discs have probably a role in the evolution of sagittal balance.
References


**Part III: Is vertebral post-fracture local deformity a predictive factor for final spinal regional deformity?**

Study in coronal and sagittal plane.

**Introduction**

Spine injuries in children are relatively rare and represent a small percentage of overall injuries in children (range 0.3-4%) (1-6), there are less common than in adults because of the greater mobility and elasticity of the pediatric spine and the smaller mass of the child’s body. The incidence of pediatric spine injuries has been reported to be 2-5% of all spine injuries (1). They occur in thoracic or lumbar spine from 20% to 60% of cases (4). There is an ongoing discussion about the causal role of skeletal growth process in the etio-pathogenesis of spinal deformity after fractures. A fractured vertebra could be rebuilt like long bones’ fractures due to skeletal remodeling during growth, resulting in a normalized spine alignment. Or fracture during growth could result in an increased local kyphosis and/or scoliosis with subsequent spinal imbalance later in life (1, 2, 7, 8). Looking at the local kyphosis is part of the set of parameters to decide the appropriate treatment for a vertebral fracture. So it would be logical to look at the local deformity (LD) also in coronal plane to decide the kind of treatment. It would mean that local spinal deformity after vertebra fracture is responsible for the final spinal alignment. To our knowledge, there is only few articles in the literature that searched correlation between local post-trauma deformity and final regional deformity (RD) and it was always in sagittal plane and not in coronal. The purpose of this study is to establish if there is a correlation between the initial LD and the final RD in both coronal and sagittal planes studying vertebral index (VI) and Cobb angle.

**Materials and Methods**

A tricentric (2 hospitals, 3 departments of surgery) retrospective study was performed between 1996 and 2014. An informatics research was initiated to find all patients under 18 years old who were hospitalized because of lumbar and/or thoracic vertebral fracture. Patients who underwent a surgery during the first year after the accident, patients with neurologic deficiency and patient with pre-existing scoliosis or spinal malformation were excluded. Fractures were classified according to Magerl classification. All patients’ demographic data are summarized in table 1 (p.46).

**Radiological measurements.** For each fracture the following measurements were done; on anteroposterior (AP) radiograph, both Cobb angle (9) and the two lateral heights of each fractured vertebral body (VB) was measured (figure 6). Cobb angle was used to quantify the RD between vertebrae at the upper and lower limits of the curve. On lateral view, RD, thoracic kyphosis or lumbar lordosis, were measured, according to the level of the fracture,
thoracic or lumbar respectively, and anterior and posterior heights of each fractured VB (figured 7). All measurements were done at the moment of the accident, authors using full spine radiograph with brace or cast and at last follow up with another full spine radiograph without contention; all radiograph were done in standing position. There were done by a single person to decrease measurements bias, and Surgimap software was used. With these measurements, the authors defined two VI to reflect the bony LD. One in coronal plane, coronal VI (CVI) = smallest lateral height of VB/highest lateral height of VB, and one in sagittal plane: sagittal VI (SVI) = anterior height of VB/posterior height of VB.

**Analysis.** Several analyses were done to search a correlation between VI and final RD. First, thoracic and lumbar fractures were separated into two groups; then, these groups were divided into three equal subgroups according to the value of VI. In each two groups and six subgroups a correlation between VI and Cobb angle was searched and a comparison between RD in subgroups was made to search if VI was a predictive factor for RD.

**Statistical analysis.** Data are presented as mean and range. A Pearson test was used to search a correlation between VI and RD. A Student t test was performed to compare subgroups. A difference of P<0.05 was regarded as a statistical significant difference.

**Figure 6: Measurements on AP radiograph: Cobb angle and the two lateral heights of fractured VB.**
Figure 7: Measurements on lateral view: lordosis and anterior and posterior heights of fractured VB.

Results

The computer search found 320 patients in center 1 (2 departments of surgery) and 41 in center 2. After application of exclusion criteria and verification of patients’ files, 48 patients were included, 23 males and 25 females (figure 2, p.50), with a total of 84 vertebrae. There were 43 thoracic fractures (T4-T12) and 41 lumbar fractures (L1-L5) (Table 2, p.49); 89.6% are A1 fracture according to Magerl classification. All fractures were treated conservatively, 85.4% had a brace and 14.6% underwent reduction of the fracture and a cast. Mean age at inclusion was 12.3 years old (range 2.6-17.1). Mean follow-up was 49 months (range 12-210) and 62% had a Risser grade 4 or 5 at the end of the follow-up.

Analyses with two groups: thoracic and lumbar spinal fracture

Coronal plane. (Table 11) Results reported that smallest was the LD, the smallest was the RD in both thoracic and lumbar area.
### Table 11: Results for thoracic and lumbar fractures in coronal plane.

<table>
<thead>
<tr>
<th></th>
<th>Thoracic RD</th>
<th>Lumbar RD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean=5.9</td>
<td>mean=10.2</td>
</tr>
<tr>
<td></td>
<td>range 1;19</td>
<td>range 0;34</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Thoracic</th>
<th>Lumbar</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVI</td>
<td>mean=0.931</td>
<td>mean=0.769</td>
</tr>
<tr>
<td></td>
<td>range 0.786;1</td>
<td>range 0.667;0.981</td>
</tr>
<tr>
<td></td>
<td>r=-0.03</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td></td>
<td>r=-0.32</td>
</tr>
</tbody>
</table>

**Sagittal plane.** (Table 12) Contrary to the coronal plane, the smallest was the LD, the highest was the RD in both lumbar and thoracic area. Correlation was stronger in lumbar area than in thoracic area, in both coronal and sagittal plane.

### Table 12: Results for thoracic and lumbar fractures in sagittal plane.

<table>
<thead>
<tr>
<th></th>
<th>Kyphosis</th>
<th>Lordosis RD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean=39.5</td>
<td>mean=38.1</td>
</tr>
<tr>
<td></td>
<td>range 10;58</td>
<td>range 15;66</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Thoracic</th>
<th>Lumbar</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVI</td>
<td>mean=0.760</td>
<td>mean=0.769</td>
</tr>
<tr>
<td></td>
<td>range 0.533;0.921</td>
<td>range 0.478;0.949</td>
</tr>
<tr>
<td></td>
<td>r=0.20</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td></td>
<td>r=0.31</td>
</tr>
</tbody>
</table>

**Analyses with subgroups according to VI values**

**Coronal plane.** (Table 13 and 14) Results reported that smallest was the LD, the smallest was the RD in both lumbar and thoracic area. However, LD and RD seemed to be correlated only in three of six subgroups, two in thoracic area (r=-0.56 and r=-0.25) and one in lumbar area (r=-0.44).
Table 13: Results for the subgroup of thoracic fractures, results in coronaI plane.

<table>
<thead>
<tr>
<th></th>
<th>Thoracic RD</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean=5,4</td>
<td>mean=5,9</td>
<td>mean=5,3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>range 1;19</td>
<td>range 0;19</td>
<td>range 0-12</td>
<td></td>
</tr>
<tr>
<td>CVI</td>
<td>0,786-0,917</td>
<td>r=-0,10</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>0,921-0,969</td>
<td>X</td>
<td>r=-0,56</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>0,972-1</td>
<td>X</td>
<td>X</td>
<td>r=-0,25</td>
</tr>
</tbody>
</table>

Table 14: Results for the subgroup of lumbar fractures, results in coronaI plane.

<table>
<thead>
<tr>
<th></th>
<th>Lumbar RD</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean=13,2</td>
<td>mean=8,3</td>
<td>mean=7,8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>range 2-34</td>
<td>range 2-16</td>
<td>range 0-16</td>
<td></td>
</tr>
<tr>
<td>CVI</td>
<td>0,667-0,896</td>
<td>r=-0,16</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>0,900-0,944</td>
<td>X</td>
<td>r=-0,08</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>0,946-0,981</td>
<td>X</td>
<td>X</td>
<td>r=-0,44</td>
</tr>
</tbody>
</table>

Sagittal plane. (Table 15 and 16) In thoracic area there were discordant results, the more significant reported that smallest was the LD, the highest was the RD. The same results were found in lumbar area with a satisfying correlation (r>0.3).

Table 15: Results for the subgroup of lumbar fractures, results in sagittal plane.

<table>
<thead>
<tr>
<th></th>
<th>Kyphosis</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean=36,4</td>
<td>mean=38,4</td>
<td>mean=43,5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>range 10;56</td>
<td>range 14;58</td>
<td>range 32;53</td>
<td></td>
</tr>
<tr>
<td>SVI</td>
<td>0,533-0,718</td>
<td>r=-0,18</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>0,720-0,788</td>
<td>X</td>
<td>r=0,39</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>0,800-0,921</td>
<td>X</td>
<td>X</td>
<td>r=-0,11</td>
</tr>
</tbody>
</table>

Table 16: Results for the subgroup of thoracic fractures, results in sagittal plane.

<table>
<thead>
<tr>
<th></th>
<th>Lordosis</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean=38,4</td>
<td>mean=41,5</td>
<td>mean=39,3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>range 19-66</td>
<td>range 29-64</td>
<td>range 30-59</td>
<td></td>
</tr>
<tr>
<td>SVI</td>
<td>0,478-0,714</td>
<td>r=0,38</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>0,737-0,837</td>
<td>X</td>
<td>r=0,42</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>0,837-0,949</td>
<td>X</td>
<td>X</td>
<td>r=0,67</td>
</tr>
</tbody>
</table>
Comparison of RD according to VI values. (Tables 17, 18, 19 and 20)

There was no statistical difference between RD in subgroups according to the value of bony LD.

Table 17: Comparisons of RD according to VI value, thoracic fractures in coronal plane.

<table>
<thead>
<tr>
<th>Thoracic RD</th>
<th>mean=5.4 range 1;19</th>
<th>mean=5.9 range 0;19</th>
<th>mean=5.3 range 0-12</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean=5.4 range 1;19</td>
<td>X</td>
<td>P&gt;0.05</td>
<td>P&gt;0.05</td>
</tr>
<tr>
<td>mean=5.9 range 0;19</td>
<td>X</td>
<td>X</td>
<td>P&gt;0.05</td>
</tr>
</tbody>
</table>

Table 18: Comparisons of RD according to VI value, lumbar fractures in coronal plane.

<table>
<thead>
<tr>
<th>Lumbar RD</th>
<th>mean=13.2 range 2-34</th>
<th>mean=8.3 range 2-16</th>
<th>mean=7.8 range 0-16</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean=13.2 range 2-34</td>
<td>X</td>
<td>P&gt;0.05</td>
<td>P&gt;0.05</td>
</tr>
<tr>
<td>mean=8.3 range 2-16</td>
<td>X</td>
<td>X</td>
<td>P&gt;0.05</td>
</tr>
</tbody>
</table>

Table 19: Comparisons of RD according to VI value, thoracic fractures in sagittal plane.

<table>
<thead>
<tr>
<th>Kyphosis</th>
<th>mean=36.4 range 10;56</th>
<th>mean=38.4 range 14;58</th>
<th>mean=43.5 range 32;53</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean=36.4 range 10;56</td>
<td>X</td>
<td>p&gt;0.05</td>
<td>p&gt;0.05</td>
</tr>
<tr>
<td>mean=38.4 range 14;58</td>
<td>X</td>
<td>X</td>
<td>p&gt;0.05</td>
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</table>
Table 20: Comparisons of RD according to VI values, lumbar fractures in sagittal plane.

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<th>mean=41,5</th>
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<td>range 19-66</td>
<td>range 29-64</td>
<td>range 30-59</td>
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<td>range 19-66</td>
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<tr>
<td>mean=41,5</td>
<td>X</td>
<td>X</td>
<td>p&gt;0,05</td>
</tr>
<tr>
<td>range 29-64</td>
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Discussion

Our results report the same progression of LD and RD in coronal plane while in sagittal plane, LD and RD have an opposite evolution. Indeed, when LD decreases, RD continues to increase.

Results in coronal plane could be explained by the Hueter and Volkmann principle. They describe how increased pressure on end plate of bone retards its growth (Hueter) and how, conversely, reduced pressure accelerates it (Volkmann). It would mean that vertebral LD is responsible for an asymmetric growth which causes RD during development.

Authors propose involvement of intervertebral discs to explain that LD and RD do not have the same direction in sagittal plane, as in case of scoliosis. Modi et al. (11) in a series of 150 adolescent idiopathic scoliosis (AIS) conclude that wedging in disc and body are increasing with progression of scoliosis and that disc wedging is more profound in lumbar area. This result could explain why correlation is stronger in lumbar area than in thoracic one. Schloesser et al. (12) study the contribution of VB and intervertebral discs to three-dimensional (3-D) spinal deformity in AIS; they conclude that discs contribute more to 3-D deformity than bony structure, because of the decreased stiffness of intervertebral fibrocartilage as compared to bony vertebrae.

Authors explain in the difference for results between sagittal and coronal planes because of soft structures and anatomy. Indeed, all posterior muscles and ligaments such as interspinal ligament, can offset local kyphosis. In the same manner, facet joints’ sagittal position opposes an anterior sagittal imbalance. All these structures can balance the LD in sagittal plane but not in coronal one.

Other authors use a VI in sagittal plane between anterior and posterior body height because it strictly evaluates the modeling potential of the fracture. Gaca and al. (10) define an anterior-to-posterior VB ratio on sagittal tomographies’ reformations. They conclude that a ratio less than 0.893 should raise the possibility of vertebra trauma more than a normal variant. It is the case for the majority of our VI. Karlsson and al. (5) use the same ratio and report a reduction of wedge shape of the fractured vertebra in young patients. However, in
these studies the relation with final spinal deformity is not presented while it is the problem which is responsible for spinal imbalance, pain and loss of quality of life. There is, to our knowledge, no report with a coronal VI in literature.

The weaknesses in our study are the retrospective design responsible for a heavy loss of information and patients and the difficulty of radiographic measurements. The VI introduced in this study needs to be validated using a large number of patients. The strength of our study is the follow-up and the radiographic evaluation by the same individual both at baseline and follow-up.

Even if parts of LD are correlated with RD, comparisons between RD according to value of VI report no argument to use the VI value as a predictive factor of RD. It means that there are probably other factors than bony LD involved in determining final RD after a vertebral fracture.

**Conclusion**

After a thoracolumbar fracture, the evolution of LD and RD seems to be correlated; LD and RD follow the same direction in coronal plane but have an opposite evolution in sagittal plane. But, the initial deformation of VB cannot predict the final curvature of spine both in coronal and sagittal.
References

Conclusion

Notre travail suggère que les déformations rachidiennes post-traumatiques sont le résultat à la fois d’atteinte des structures adjacentes, les disques intervertébraux entre autres et, de phénomènes adaptatifs répondant au loi de Hueter et Volkman dans le plan coronal et, de Roaf (1) dans le plan sagittal. En effet, dans le plan coronal comme dans le plan sagittal, on assiste à une diminution de la déformation locale vertébrale entre le jour de la fracture et la fin du suivi alors que les courbures régionales augmentent sur cette même période. En outre, nos analyses statistiques ont mis en évidence des résultats variables concernant la corrélation entre la déformation osseuse initiale et la déformation régionale finale. En effet, les résultats sont différents selon que l’on soit dans le plan coronal ou dans le plan sagittal et, la corrélation est plus forte à l’étage lombaire qu’à l’étage thoracique. Dans le plan coronal, la déformation initiale locale et la déformation régionale évolue dans la même direction; alors que dans le plan sagittal, la déformation initiale locale diminue quand la déformation régionale augmente. Ces résultats laissent penser que les courbures finales sont au moins autant le résultat de phénomènes adaptatifs de l’ensemble des structures de la colonne vertébrale que directement liées à l’importance du tassemment initial du corps vertébral.

L’amélioration de la déformation du corps vertébral entre le jour de la fracture et la fin du suivi est retrouvée dans tous les résultats, quel que soit le plan étudié, le niveau fracturé, le stade initial de Risser et le nombre de fractures. On retrouve cette amélioration même chez les patients qui présentent les plus grandes déformations régionales en fin de suivi. Ainsi, ce résultat ne plaide pas en faveur de l’hypothèse qui ferait des épiphysiodèses localisées le point de départ des troubles de la statique rachidienne post-traumatique comme cela est proposé dans la littérature (2).

Par ailleurs, notre travail confirme qu’il existe un plus grand potentiel de remodelage osseux chez les patients qui ont un stade de Risser inférieur ou égal à 2 (3, 4) par rapport aux patients aux patients avec une maturité osseuse plus avancée.

L’étude dans le plan coronal suggère que les fractures du rachis thoracique et lombaire sont un facteur de risque de scoliose puisqu’on retrouve une prévalence supérieure à celle de la population générale. Le stade de Risser supérieur ou égal à 3, les fractures lombaires et les fractures uniques sont être responsables de plus de déformations. Les patients ayant un Risser supérieur ou égal à 3 ont un remodelage osseux faible ce qui explique qu’ils ne sont pas en mesure de corriger suffisamment la déformation fracturaire ; ainsi, une déformation régionale s’installe selon les lois de Hueter et Volkman. Pour expliquer que les fractures lombaires entraînent plus de déformations que les thoraciques, les auteurs ont émis l’hypothèse que le caractère mobile des vertèbres lombaires par rapport aux thoraciques les rend plus susceptibles d’entrainer des déformations régionales. Enfin, les fractures uniques présentent une déformation initiale locale plus importante que les fractures étagées. Bien que cette différence ne soit pas significative, les auteurs émettent l’hypothèse que cette déformation locale plus importante témoigne de lésions sévères à la fois osseuses et des parties molles ce qui expliquerait une déformation régionale finale plus sévère. En effet, à mécanismes
équivalents, lors d’une fracture unique la vertèbre est plus déformée que si la force de compression est répartie sur plusieurs étages.

Dans le plan sagittal, notre travail confirme les grandes capacités de compensation du rachis pédiatrique en raison de son extrême souplesse. En effet, sans surprise, on ne retrouve pas de déséquilibre sagittal malgré la persistance à terme de 8 et 9° de cyphose locale, respectivement, en thoracique et en lombaire. Par ailleurs, les résultats rapportent une adaptation quasi-exclusive dans la colonne vertébrale sans mise en jeu du pelvis comme on peut le voir chez l’adulte.

L’ensemble de notre travail confirme que le traitement conservateur pour les fractures de type A1 de la classification de Magerl est un traitement de choix. En effet, dans le plan coronal malgré un taux de scoliose élevé (11 patients soit 23%), les angles de Cobb restent faible (10 des 11 patients ont un angle de Cobb inférieur à 20°). Dans le plan sagittal, on ne retrouve pas de déséquilibre rachidien et les valeurs de cyphoses locales en fin de suivi restent tolérables (8.4° en moyenne). En outre, les résultats cliniques sont rassurants. Malgré un taux élevé de douleurs rachidiennes (81%), 97% des patients n’ont pas de retentissement de ces douleurs sur leur vie quotidienne et ne prennent pas d’antalgiques. Au regard du taux de douleurs lombaires, il n’a pas été possible de rechercher de corrélation entre la douleur et l’importance des déformations rachidiennes. De plus, la littérature rapportent des taux de douleurs rachidiennes très variables, jusqu’à 70%, chez l’enfant et l’adolescent sans antécédent de fracture (5-7) ce qui rend l’interprétation de nos résultats très difficile et ce d’autant que les douleurs rachidiennes sont multifactorielles.

Notre travail rétrospectif avec tous les biais et pertes d’informations qui en découlent nécessite d’être confirmé par une étude prospective avec un suivi à long terme. Un suivi très prolongé aurait comme intérêt principal de savoir si ces patients font l’objet à l’âge adulte de chirurgies extensives de la colonne vertébrale pour des troubles de la statique rachidienne dans le plan coronal et/ou sagittal afin de juger de la qualité de la prise en charge initiale.
Références


Annexes communes:

**Table 1: Demographic data.**

MVA: Motor Vehicle Accident, SA: Sports Accident
<table>
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<th>Patient number</th>
<th>Age (month)</th>
<th>Gender</th>
<th>Injury date</th>
<th>Risser's grade</th>
<th>Y-cartilage</th>
<th>Mechanism of injury</th>
<th>Level of injury</th>
<th>Fracture</th>
<th>Osteoarticular associated lesions</th>
<th>Visceral associated lesion</th>
<th>Total follow up (month)</th>
<th>Risser's grade at follow up</th>
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<td>25/06/2001</td>
<td>1</td>
<td>closed</td>
<td>fall</td>
<td>L1</td>
<td>A1</td>
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<td>30/03/1993</td>
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<td>30/01/1998</td>
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<td>A1</td>
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<td>26/02/2000</td>
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<td>A1</td>
<td>rib fractures</td>
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Table 2: Level injury.

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<th>T6</th>
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<th>T8</th>
<th>T9</th>
<th>T10</th>
<th>T11</th>
<th>T12</th>
<th>L1</th>
<th>L2</th>
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Figure 2: Flow chart.

Computer search Center 1
n=320

Exclusion:
- Surgical treatment n= 35
- Incomplete data n= 156
- Files not found n= 51
- Neurological deficiency n= 32
- Pre-existing deformity n= 8

Patients included
n=38

Computer search Center 2
n=41

Exclusion:
- Surgical treatment n=2
- Incomplete data n=24
- Files not found n=5

Patients included
n=10

Patients included
n=48

Exclusion:
- Surgical treatment n=35
- Incomplete data n=156
- Files not found n=51
- Neurological deficiency n=32
- Pre-existing deformity n=8