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ORIGINAL ARTICLE

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Morphometry of the lower lumbar vertebrae in patients with and without low back pain

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Abstract The authors made several measurements in the lower lumbar vertebrae of patients with and without low back pain. Our objective was to determine the allometric relationships between different dimensions of the lumbar canal, the effects on these from degenerative disease, and differences between the symptomatic and asymptomatic populations. We compared 119 patients suffering from low back and sciatic pain and 39 subjects without lumbar symptoms as determined by computed tomography (CT). The following measurements were made: sagittal diameter of the canal, interpedicular distance, interarticular distance, and anteroposterior diameter of lateral recess and foramen. With respect to the patients with lumbar pain, the asymptomatic group proved to have wider foramina from L3 to L5 and wider sagittal diameters in S1. The patients with canal stenosis revealed lower figures for all diameters of the central canal, lateral recess of L4, and foramina of L4 and L5. Patients with lumbarization showed smaller diameters of the central canal. *Conclusion*. There is an allometric relationship between the dimensions of the central canals. This relationship is less evident with lateral canals. The patients without lumbar symptoms had wider foramina and sagittal diameters in S1 than those with lumbar symptoms. Of these, patients who developed symptoms of canal stenosis demonstrated smaller diameters in central and lateral canals. Of the developmental anomalies, lumbarization proved to be associated with canal stenosis due to smaller diameters of the central canals.

Keywords Lumbar spine · Computed tomography · Osteometry · Canal stenosis · Abnormalities

Introduction

Many studies point out that the space available in the lumbar canal is the key to the development of discal symptoms [12] but may also be important in other causes of lumbar pain (hypertrophy of bone structures, vertebral displacement, etc.) [8]. The size of the spinal canal is determined by different factors. First of all are genetic factors, the phenotypic expression of which can be altered by direct local injury (trauma, infection, etc.) or repercussions of systemic disturbances (malnutrition, cardiovascular illness, etc.) to the spinal column during its development [1, 6, 26, 27]. Although degenerative alterations have been described in the young [33], these are more frequent when spinal development has ended, and therefore its effects on the dimensions and morphology of the canal could be differentiated with respect to congenital and developmental alterations [15, 28].

The different rates of growth of different dimensions of an organism are referred to as allometric growth. The allometric relationship between two diameters may be calculated by ontogenetic data (measurements on individuals of different ages) or static data (measurements on individuals of similar age but different size) [10]. This possibility moved us to take a series of measurements in the lumbar

regions of 119 adult patients suffering low back pain and sciatica of mechanical origin and in 39 adult patients without prior histories or current lumbar pain.

Our objectives were (1) to determine the allometric relationship between different measurements of the lumbar spinal canal which could indicate how the lumbar region grows or is influenced by developmental or degenerative diseases and (2) to ascertain whether these measurements differed between symptomatic and asymptomatic populations.

Material and methods

Materials

After a revision of clinical histories to exclude cases of tumoral, inflammatory, and traumatic low back pain, a total of 119 patients (76 males and 43 females) suffering mechanical low back pain and sciatica were selected. Mean age, weight, and height were 41.9±1.23 years, 73.6±1.30 kg, and 167.9±0.89 cm, respectively.

Radiological study consisted of simple anteroposterior and lateral radiographs as well as computed tomography (CT) of the lumbar spine. The CT scans included continuous slices from the pedicle of L3 to the foraminal level of S1 performed with patients in the supine position using a Sytec 3000 apparatus (General Electric, Milwaukee, Wis., USA), with slice thickness of 3 mm or 5 mm at 120 kV and 100 mA/s. The slices were made with the gantry inclined parallel to the intervertebral disc. When the inclination was inadequate for radiological measurements, image reformation was performed [13]. When transitional vertebra was suspected, plain film of the dorsal spine was used for correct identification of the vertebral level. We began counting at T1, considered to be the vertebra articulated with a normal first rib. After T12 was located, lumbar vertebrae could be counted easily in a caudal direction.

The healthy control group was composed of 39 persons (19 males and 20 females), none of whom had any history of back pain resulting in time off from work or need of medical treatment. Mean age, weight, and height were 37.9±2.04 years, 71.3±3.29 kg, and 164.3±2.53 cm, respectively. The CT study was conducted in the same manner as with patients.

Comparisons were made between symptomatic and asymptomatic patients, both sex-pooled and separated.

Radiological classification

Alterations of the discovertebral complex

Changes in the discovertebral complex noted were posterior disc hernia and disc degeneration. In the former, focal bulging of the disc with or without compression of the nerve structures was considered (Fig. 1). Sequestrated fragments were also included. The latter [31] were estimated from the presence of osteophytes, narrowed disc space, vacuum phenomenon, or diffuse bulging disc. Bulge was defined as a diffuse nonfocal extension of nonosseous material more than 2.5 mm beyond the normal disc space [35] (Fig. 2).

Alterations of the vertebral arch

Spondylolysis resulted in defects in the intervertebral arch at the level of the isthmus.

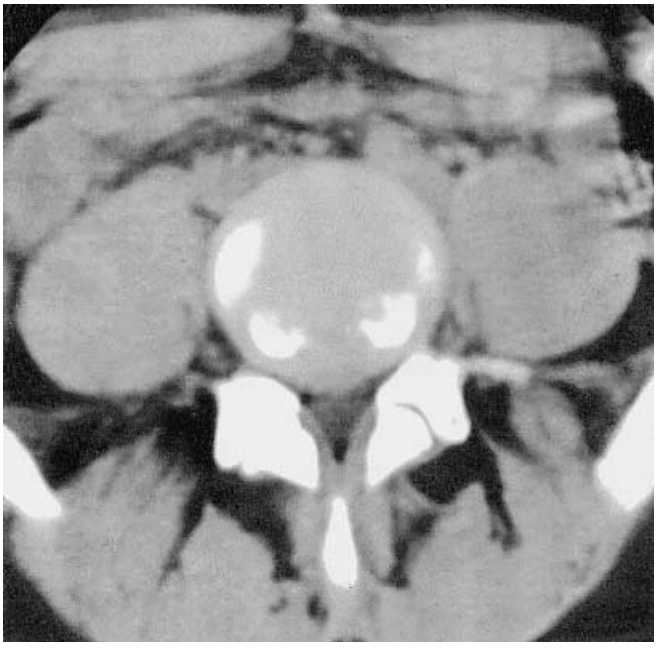

Fig. 1 Herniated disk in an asymptomatic female of 27 years

Fig. 2 Bulging disk in an asymptomatic male of 24 years

Alterations of the posterior articulations

Facet degeneration was considered due to hypertrophic changes or osteophyte formation, periarticular calcification, articular narrowing of the joint space, vacuum phenomenon, or subchondral erosion. No attempt was made to grade the severity of the degenerative changes (Fig. 3).

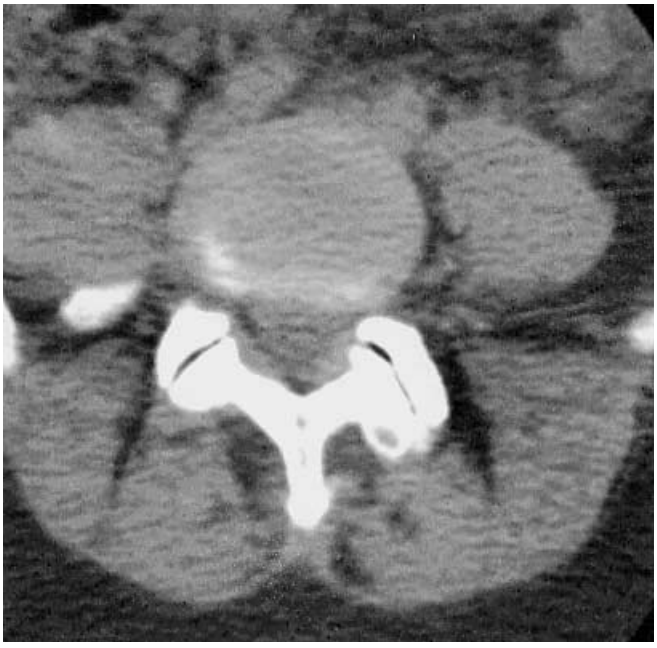

Fig. 3 Facet hypertrophy and articular vacuum phenomenon in an asymptomatic female of 54 years

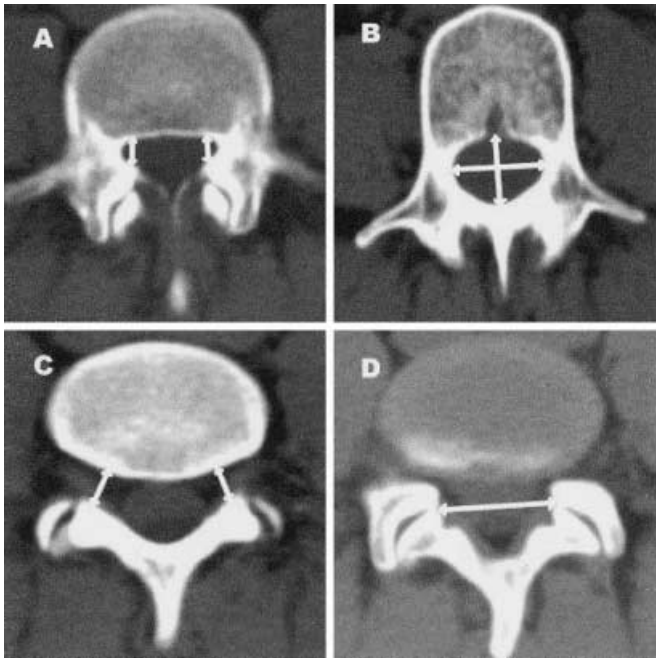

Fig. 4 Schematic representation of radiological measures. **A** lateral recess, **B** sagittal and interpedicular diameters, **C** foramen, **D** interarticular distance

Alterations of the vertebral canal

In this group, only stenosis of the bony canal was included. Stenosis of the central canal was shown in patients with sagittal diameters lower than 12 mm in the presence of signs of nerve compression (motor or sensorial alterations). With regard to the interarticular diameter, values of less than 15 mm in patients with symptoms of nerve compression were considered pathological [5, 9, 16]. Stenosis of the lateral canal was diagnosed when the nerve root was found to be trapped in the bony margins of the lateral recess or foramen with clinical symptoms or signs attributable to this root [20, 22].

Transitional anomaly

After vertebral recount, transitional anomaly was classified as lumbarization of S1 or sacralization of L5. In the former, we ascertained 12 thoracic vertebrae and six lumbar vertebrae. The lower vertebra was considered as an S1 fully or partially detached from the sacrum. In the latter, only four lumbar vertebrae were identified, because the theoretical L5s were partially or fully attached to the sacrum or articulated to it by the transverse processes.

Radiographic measurements

All measurements were performed at the CT screen, positioning the cursor on the suitable reference point using a trackball. Window width and level were 1000 H and 300 H, respectively. The radiographic measurements were divided into two groups (Fig. 4).

Measurements of the central canal

Interarticular distance (IAD) was measured at the level of the disc slice joining the internal borders of the facet articulations. Interpedicular distance (IPD) was considered as the distance between midlines of the internal sides of the pedicles. Sagittal diameter was determined in the middle pedicular slice, from the entrance of the basivertebral vein to the midpoint of the anterior portion of the lamina.

Measurements of the lateral canal

The lateral canal was measured bilaterally at each level. For statistical treatment, the mean of the right and left sides was used. The lateral recess was measured in the slice at the level of the upper vertebral platform as the distance between the posterior edge of the vertebral body and the anterior part of the articular facet.

The anteroposterior diameter of the foramina was determined at the subpedicular slice at the level of the dorsal ganglia as the distance between the posterolateral edge of the vertebral body and the anterior part of the facets [21].

Statistical treatment

All measurements were made by a musculoskeletal radiologist unaware of final clinical diagnosis, to avoid interobserver errors [3]. To estimate intraobserver error, we randomly chose 30 patients, and the same examiner repeated the measurements on successive days. This measurement error was calculated as the square root of the sum of the differences between each two measurements, squared, and divided by 60. The reliability of the measurements was determined by the coefficient of intraclass correlation [15].

For the sake of consistency, the radiological findings were estimated by a neuroradiologist. A subset of 30 patients were reexamined on different days to estimate the intraobserver agreement. This was measured by kappa values [7].

The influence of the different radiological alterations on the measurements was determined by analysis of covariance, using

one of the radiological alterations as a fixed factor, while one of the measurements was the dependent variable, introducing age, weight, and height as covariables.

The allometric relationship between different measurements of the canal was calculated using Pearson's correlation analysis.

Results

Frequency of radiological alterations presented

Table 1 lists the frequency of radiological alterations found in the 119 patients and 39 healthy control subjects. Table 2 shows the statistical association between the different radiological abnormalities.

Measurement error

The intraobserver error for radiological measurements ranged from 1.6% for the lateral recess of L4 and 3.5% for the interpedicular distance of S1. The intraclass correlation of intraobserver measurements was lower for the lateral recess of S1 (0.681) and greater for the sagittal diameter of L5 (0.982).

Kappa values for the intraobserver variability of radiological abnormalities ranged from 0.762 for facet pathology to 0.849 for bulging disk.

Table 1 Frequency of patients with a given radiological alteration

Pathology	Normal		Low back patients	
	(*n*)	(%)	(*n*)	(%)
Herniated disc	7	17.9	64	53.7
Disc degeneration	18	46.1	97	81.5
Disc bulge	14	35.8	53	44.5
Spondylolysis	1	2.5	15	12.6
Facet degeneration	11	28.2	62	52.1
Canal stenosis	0	0	14	17.7
Transitional vertebra				
Lumbarization	0	0	10	8.4
Sacralization	4	10.2	12	10.0

Table 2 Statistical association between different radiological abnormalities. *HD* herniated disc, *DD* disc degeneration, *FD* facet degeneration, *CS* canal stenosis, *SS* spondylolytic spondylolisthesis, *S* sacralization, *L* lumbarization. **P*<0.05, ***P*<0.001

	HD	DD	FD	CS	SS	S
DD	50					
FD	26	66**				
CS	6	14	11*			
SS	4	12	10	0		
S	6	10	9	0	0	
L	4	9	5	5**	1	0

Correlations between different measurements (allometric relationship)

Except for S1, there was a positive association between the sagittal diameters of the central canal and the transverse diameters (range 0.24–0.83).

Lateral canal measurements were highly and positively correlated, but their relationships with the central canal measurements were less clear: lateral recess measurements were not related to any interarticular diameters. They correlated only with the sagittal diameters of L3 and L5 (range 0.30–0.46) and to a lesser degree with interpedicular diameters (range 0.10–0.41).

The dimensions of the foramina of L3 were not related to any of the central canal diameters. Dimensions of the foramina of L4 and L5 correlated with some of the sagittal and transverse diameters of L3 to L5 (range 0.06–0.46).

Correlations with age, weight, and height

No correlation was found between subject height and weight and the radiological measurements of their lumbar spines. Age correlated negatively with foraminal measurements at L3 (–0.40), L4 (–0.33), and L5 (–0.25).

Differences between the symptomatic and asymptomatic population

Differences were found mainly at the lateral canal and are listed in Table 3. Table 4 indicates the measurements that remained statistically different, separated by sex.

Measurements in the different radiological findings

The patients with canal stenosis presented smaller diameters of their central canals, lateral recesses of L5, and foramina of L4 and L5. The patients with transitional vertebrae showed different trends according to the type of transitional anomaly.

The patients with lumbarization revealed reduced central canal diameter, IAD ($P<0.01$) and IPD distances ($P=0.004$–$P<0.001$) in all levels, and to a lesser degree sagittal dimensions of L4 ($P<0.001$), L5, and S1 ($P<0.05$).

The patients with sacralization also presented alterations in the transverse diameters of their central canals but showed a larger IPD distance in L5 ($P<0.05$) and IAD in L3-4 ($P<0.001$) and L4-5 ($P<0.05$). Sagittal diameters were smaller in S1 ($P<0.05$).

Table 3 Differences in millimeters between the patients with low back and sciatic pain and the asymptomatic population. *SAG* sagittal, *IAD* interarticular distance, *IPD* interpedicular distance, *LR* lateral recess, *FOR* foramen, *NS* not significant

	Low back pain	Asymptomatic	Significance
SAG L3	15.0±0.25	15.8±0.28	NS
SAG L4	15.8±0.25	16.3±0.37	NS
SAG L5	17.1±0.33	17.4±0.40	NS
SAG S1	16.6±0.33	18.0±0.46	$P<0.01$
IAD L3-4	19.7±0.33	19.9±0.38	NS
IAD L4-5	22.2±0.40	22.3±0.47	NS
IAD L5-S1	25.8±0.45	27.0±0.53	NS
IPD L3	22.6±0.53	23.5±0.30	NS
IPD L4	23.5±0.32	23.7±0.35	NS
IPD L5	26.5±0.35	27.1±0.38	NS
IPD S1	30.3±0.34	31.2±0.40	NS
LR L4	5.9±0.26	6.3±0.22	NS
LR L5	5.2±0.10	5.5±0.16	NS
LR S1	5.3±0.10	5.6±0.14	NS
FOR L3	8.9±0.20	10.3±0.25	$P<0.01$
FOR L4	8.5±0.13	9.9±0.19	$P<0.01$
FOR L5	8.3±0.19	8.8±0.31	NS

Table 4 Radiological measurements that remain statistically different analyzed by sex. *SAG* sagittal, *FOR* foramen, *NS* not significant

	Men	Women
SAG S1	$P=0.002$	$P=0.093$
FOR L3	$P<0.001$	$P=0.073$
FOR L4	$P<0.001$	$P=0.036$
FOR L5	NS	$P=0.048$

Discussion

A major problem in the management of degenerative lumbar disease is the difficulty in correlating many radiological abnormalities with patients' clinical symptoms. This is also true for measurements of the vertebral canal [4, 11, 34].

This difficulty prompted us to perform a comparative morphometric study of patients with low back and sciatic pain and healthy controls with no history of back troubles. This study involved measurements of the central and lateral canals.

Central canal

Measurements of the central canal included transverse and sagittal diameters. Although ontogenetic differences concur in their formation [19], these dimensions show an allometric relationship, and variation of one diameter influenced the value of the others. The cases of canal stenosis show this finding clearly, because all central canal diameters were consistently lower. Nevertheless, it is well known that sagittal diameters stop increasing in early childhood, while transverse diameters grow until adulthood [17, 24]. If the increase in sagittal diameter is visibly disturbed in an early phase of life, the allometric relationship explains that even when the damaging agent disappear, other diameters in the vertebral canal will also be hampered from reattaining normal values.

Except for the sagittal diameter of S1, the dimensions of the central canals showed no differences between the symptomatic and asymptomatic populations. This tendency is consistent with the wide range of values reported in the literature that demonstrate overlap between symptomatic and asymptomatic populations [3, 9, 15, 21, 29]. Also, most clinical symptoms result from injuries to the soft tissue components of the spine, while bony canal measurements remain within the normal value range [30]. The capacity of the bone central canal alone is usually not an important clue to the development of symptoms.

Transitional vertebral anomalies represent ontogenetic alterations which cause changes to the central canals that have not been described in the literature. The trend differs with the cases of sacralization and lumbarization. In sacralization, the IPD widens at L5, compatible with sacral assimilation of the last lumbar vertebra, which behaves as an S1 and shows the greatest IPD diameter. In lumbarization, the diameters of the central canal are narrow, increasing the probability of canal stenosis. In fact, five of ten patients with lumbarization presented with clinical canal stenosis ($P<0.001$). It is as though the longitudinal elongation of the canal involved a certain loss of capacity for caudal widening, mainly of the transverse diameters, which, as is well known, grow from infancy to adulthood. Therefore, as opposed to congenital stenosis of the sagittal diameter, which occurs in the first years of life, the effect on the canal in lumbarization depends on mechanical and anatomical alterations which hinder widening of the canal throughout the entire growth period. This finding, previously unpublished in humans, has been reported in dogs [23].

Lateral canal

Foramina were wider in the asymptomatic population. This finding has also been reported for the cervical spine of asymptomatic individuals [14]. Many features suggest that these differences may be acquired rather than determined by congenital alterations. At birth, the vertebral canal is dome-shaped. Lateral canals are formed during the entire growth phase, being susceptible to local or general disturbances [2, 25]. From our results, we deduce that the capacity of the foramina is important in the development of lumbar symptoms. The lateral canal contains nerve roots, the main tissue capable of producing leg pain, while dural tissue in the central canal is less sensitive [18]. Also, the extrathecal intraspinal nerve roots are more fixed than the roots inside the thecal sac, being therefore more susceptible to injury [32].

The role played by the reduction in the sagittal diameter of S1 in symptomatic patients is not well understood. It is well known that the caudal vertebral column matures later [27], and our work has demonstrated that the sagittal diameter of S1 is the most independent dimension, along with those of the lateral canal. Therefore, their growth may be influenced by a damaging agent for a longer period of time.

In summary, a narrow canal due to severe disturbance of its growth during early phases of life could be the origin of what we know as congenital narrow canal with mainly small sagittal diameters. A less severe disturbance or injury during later phases of growth would not have repercussions on the lumbar sagittal dimensions but could affect lateral canal development while it is still growing, thus making patients more susceptible to low back pain or sciatica throughout life. The opposite would apply to asymptomatic patients who, with normal canal dimensions, can tolerate degenerative alterations to the spine without clinical repercussions. Our study suggests that small foramina may be an important clue to the development of low back pain and sciatica, as is the narrow central canal in congenital canal stenosis. Among the developmental anomalies, lumbarization proved to be associated with canal stenosis due to small transverse diameters of the central canal.

References

1. Afshani E, Kuhn J (1991) Common causes of low back pain in children. Radiographics 11:269–291
2. Atilla B,Yazici M, Kopuz C, Baris S, Balcik C (1997) The shape of the lumbar vertebral canal in newborns. Spine 22:2469–2472
3. Beers GJ, Carter AP, Leiter BE, Tilak SP, Shah RR (1984) Interobserver discrepancies in distance measurements from lumbar spine CT scans. AJNR Am J Neuroradiol 5:787–790
4. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW (1990) Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. J Bone Joint Surg Am 72:403–408
5. Burton CV, Heitoff KB, Kirkaldy-Willis W, Ray CD (1979) Computed tomographic scanning and the lumbar spine. Part II: clinical considerations. Spine 4:356–369
6. Clark GA, Panjabi MM, Wetzel FT (1985) Can infant malnutrition cause adult vertebral stenosis? Spine 10:165–170
7. Cohen J (1960) A coefficient of agreement for nominal scales. Educ Psychol Meas XX:37–46
8. Edwards WC, LaRocca SH (1985) The developmental segmental sagittal diameter in combined cervical and lumbar spondylosis. Spine 10:42–49
9. Epstein BA, Epstein JA, Lavine L (1964) The effect of anatomic variations in the vertebrae and spinal canal on cauda equina and nerve root syndromes. AJR A J Roentgenol 91:1055–1066
10. Futuyma DJ (1986) Evolutionary biology. Sinauer, Sunderland, pp 412–419
11. Hadelman S (1990) Failure of the pathology model to predict back pain. Spine 15:718–724
12. Heliövaara M, Vanharanta H, Korpi J (1986) Herniated lumbar disc syndrome and vertebral canals. Spine 11: 433–435
13. Hirschy JC, Leue WM, Berninger WH, Hamilton RH, Abbott GF (1981) CT of the lumbosacral spine: importance of tomographic planes parallel to vertebral end plate. AJR Am J Roentgenol 126:47–52
14. Humphreys SC, Hodges SD, Patwardhan A, Eck JC, Covington LA, Sartori M (1998) The natural history of the cervical foramen in symptomatic and asymptomatic individuals aged 20–60 years as measured by magnetic resonance imaging. A descriptive approach. Spine 23:2180–2184
15. Hurme M, Alaranta H, Aalto T, Knuts LR, Vanharanta H (1989) Lumbar spinal canal size of sciatica patients. Acta Radiol 30:353–357
16. Jönsson B, Strömqvist B (1993) Symptoms and signs in degeneration of the lumbar spine. J Bone Joint Surg Br 75: 381–385
17. Knutsson F (1961) Growth and differentiation of the postnatal vertebra. Acta Radiol 55:401–408
18. Kulisch SD, Ulstrom CL, Michael CJ (1991) The tissue origin of low back pain and sciatica. Orthop Clin North Am 22:181–187
19. Larsen JL (1985) The posterior surface of the lumbar vertebral bodies. Spine 10:50–58
20. Lassale B (1987) Anatomie du recessus lateral ou gouttiere radiculaire lombaire. Acta Orthop Belg 53:137–142
21. Leiviskä T, Videman T, Nurminen T, Group JDG (1985) Radiographic versus direct measurements of the spinal canal at lumbar vertebrae L3-L5 and their relations to age and body stature. Acta Radiol 26:403–411
22. Mikhael MA, Ciric I, Tarkington JA, Vick NA (1981) Neuroradiological evaluation of lateral recess syndrome. Radiology 140:97–107
23. Morgan JP (1999) Transitional lumbosacral vertebral anomaly in the dog: a radiographic study. J Small Anim Pract 40:167–172
24. Papp T, Porter RW, Aspden RM (1994).The growth of the lumbar vertebral canal. Spine 19:2770–2773
25. Papp T, Porter RW, Aspden RM (1995) Trefoil configuration and developmental stenosis of the lumbar vertebral canal. J Bone Joint Surg Br 77: 469–472
26. Porter RW, Oakshot G (1994) Spinal stenosis and health status. Spine 19: 901–903
27. Porter RW, Pavitt D (1987) The vertebral canal: I. Nutrition and development, an archeological study. Spine 12: 901–906
28. Porter RW, Hibbert CS, Wicks M (1980) Backache and the lumbar spinal canal. Spine 5:99–105
29. Postacchini F, Ripani M, Carpano S (1983) Morphometry of the lumbar vertebrae. Clin Orthop 172:296–303
30. Rauschning W (1993) Pathoanatomy of lumbar disc degeneration and stenosis. Acta Orthop Scand Suppl 64:3–12
31. Resnick D (1985) Degenerative diseases of the vertebral column. Radiology 166:3–14
32. Spencer DL, Irwin GS, Miller JAA (1983) Anatomy and significance of fixation of the lumbosacral nerve roots in sciatica. Spine 8:672–679
33. Tertti MO, Salminen JJ, Paajanen HEK, Terho PH, Kormano MJ (1991) Low-back pain and disk degeneration in children: a case-control MR imaging study. Radiology 180:503–507
34. Torgerson WR, Dotter WE (1976) Comparative roentgenographic study of the asymptomatic and symptomatic lumbar spine. J Bone Joint Surg Am 58:850–853
35. Yu S, Haughton VM, Sether LA, Wagner M (1988) Anulus fibrosus in bulging intervertebral disks. Radiology 169: 761–763