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Orthopaedic Surgeon, Middlesex Hospital and Royal National Orthopaedic Hospital I AM INDEED conscious of the honour of being allowed to give one of these lectures designed to express our respect and reverence for so great a person as John Hunter.

I would have wished, among his wide and valuable researches, to have found some observations on the lower part of the lumbar spine and sacrum, but it seems that he was too occupied discovering and working out other matters of importance. Had he focused his attention on this region, doubtless our present knowledge would be in advance of what it is to-day.

I have chosen this subject of spondylolisthesis for discussion, not because it is necessarily of great clinical significance, but on account of its importance in the study of the mechanics of the lumbo-sacral region, a crucial area in the structure of our design. That spondylolisthesis occurs at all is an event of great interest to the morbid anatomist, but it is a well known fact that many with this condition remain symptom free and indeed ignorant of its presence all their life.

It is interesting to note that in the year 1782, when Herbinaux, a Belgian obstetrician, was publishing the first cases of spondylolisthesis, Hunter was delivering the last of his six famous Croonian lectures at the Royal Society on "Muscular Motion."

## DEFINITION

There is no clear definition of spondylolisthesis, in fact it has an entirely different meaning between one author and another.

Generally speaking in Europe it has indicated an abnormal condition of the interarticular portion of the neural arch which allows ventral displacement of that part of the vertebra anterior to the lesion, thus causing an elongation of its sagittal axis. In the English-speaking world it has generally embraced all conditions in which there is forward displacement of the body of the vertebra, with or without the whole neural arch.

Kilian (1853), the originator of the term, was vague in his description but certainly included a luxation forward of a whole lumbar vertebra. A more comprehensive study of the literature does little to lessen one's confusion of the exact meaning of the term.

The earlier workers based their opinion on very few cases. Only those cases with gross deformity would have been diagnosed clinically and only few could have been examined post mortem, but the descriptions then were in many ways more accurate than the modern conception.

Neugebauer (1888) described the deformity as an elongation and angulation of the sagittal axis and a dislocation forward of the anterior part of the vertebra.

Chiari (1892) had a much broader conception of the etiological factors and distinguished between luxation forward of the whole vertebra due to articular facet deficiency and luxation forward of the anterior part of the vertebra due to a lesion of the neural arch. Meyerding (1932) defined it as a subluxation of a vertebra, usually forward and most commonly at the lumbo-sacral level.

A search for the true nature of the lesion in the pars interarticularis has been the occasion for much research and speculation, and of the various theories advanced the two most widely accepted are the congenital and the post-traumatic, the former has undoubtedly been the most popular. So great has become the controversy among those interested in this subject that the importance of the lesion has overshadowed the significance of spondylolisthesis itself.

With the advent of X-rays and especially of detailed X-rays of high standard, interest has fallen even more upon this lesion of the pars interarticularis.

Colonna (1954) stated that spondylolisthesis was produced as the result of a defect between the inferior and superior articular surfaces of the neural arch.

The narrow crack running through the pars interarticularis without vertebral displacement and known as spondylolysis has been by some workers associated with spondylolisthesis and described as a precursor of this condition.

## INVESTIGATION

This investigation consists of a clinical, radiological and in a few cases operative, study of over two hundred patients with spondylolisthesis, and here I have taken spondylolisthesis in a broad sense to mean not only slipping forward of a vertebral body, but the whole mechanism which permits part or the whole of a vertebra to luxate in such a way. This series does not include lesions other than in the lower lumbar spine nor acute fracture dislocations, nor retro-spondylolisthesis.

In these two hundred and four cases, one hundred and eight only had a break in the pars interarticularis.

In studying this series it has become apparent that they fall into five groups, three common and two uncommon, which briefly can be described by the salient features.

**Group I.**—Luxation at the lumbo-sacral junction due to congenital sacral defect including the articular facets with attenuation of the neural arch.

**Group II.**—Luxation of the anterior part of the vertebra due to attenuation or a break in the pars interarticularis or a combination of both, the facets remaining intact.

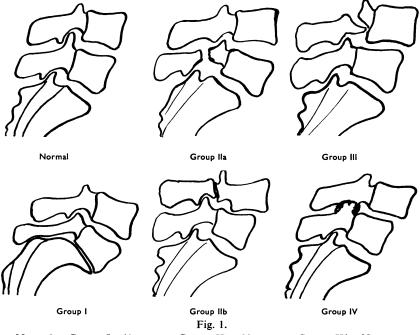
Group III.—Luxation due to an acute fracture of the neural arch.

Group IV.—Luxation due to degenerative joint changes causing facet deficiency.

Group V.—Luxation due to bone disease.

### **Group I**

This group shows essentially a luxation forward of the last lumbar vertebra due to a defect in the development of the upper part of the sacrum. It is described by some to be comparatively rare and by some



Normal; Group I—41 cases; Group IIa—88 cases; Group IIb—22 cases; Group III—3 cases; Group IV—46 cases.

is not included as spondylolisthesis at all. Yet many illustrations of so-called true spondylolisthesis are of this type. The luxation is partly due to a facet defect and partly due to elongation of the neural arch. In the process of slipping it grinds its way over the top of the sacrum with a glacier-like action, its inferior facets becoming worn and grooved and its neural arch attenuated and sometimes broken. At the outset the spinous process slips forward with the rest of the vertebra, but later it stabilises itself on the fibrous roof of the sacrum and the vertebral body, continuing to slip, leaves it behind. The lumbo-sacral disc becomes useless and degenerate and eventual stabilisation of the body is often achieved by anterior buttressing from, or fusion with, the body of the sacrum.

In this group occurs the grossest degree of displacement. Sometimes the degree of slip is greater on one side so that scoliosis occurs. Occasionally so precarious has become the position of the vertebra above that it too may slip on the displaced one below.

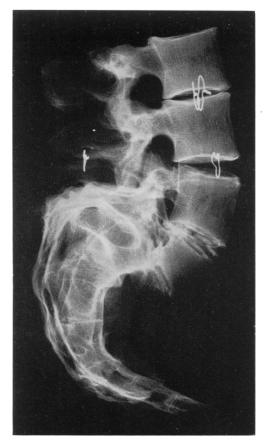


Fig. 2.

Group I.—X-ray of a specimen from the Pathological Museum at University College Hospital. It shows marked slipping, a continuous and attenuated neural arch circumscribing the top of the sacrum to which it has fused.

A defect in the development of the neural arches of the sacrum is always present, varying in degree and character. The neural arches of the lumbar spine are less frequently affected but in both regions it is the mid vertebra which is most likely to be normal. As a result the superior facets of the sacrum are an insufficient mechanical barrier to the forward thrust of the lowest lumbar vertebra whose inferior facets gradually slip either in between or over the top of the sacral facets. See also

Harris (1951). Radiologically this defect in the sacrum is often difficult to see owing to the superimposed vertebral bodies and the abnormal position of the downward displaced spine of the last lumbar vertebra



Fig. 3. Group I.—Spondylolisthesis with scoliosis. Spina bifida of the sacrum is indicated by absence of sacral spinous processes.

closely resembling a sacral spinous process. The upper part of the sacrum may be inclined forward to an abnormal degree giving an increased lumbo-sacral angle and the appearance of a "caving-in" of the lumbo-sacral area as a whole.

In this group of 41 cases, eight had a break in the neural arch.

At operation the following points are noted :--

- (1) Slipping forward of the fifth lumbar spine.
- (2) A fibrous roof to the sacrum replacing a defect in the neural arches.
- (3) Two smooth or grooved mounds of bone—the deficient sacral facets which have allowed the inferior facets of the last lumbar vertebra to slip over to between them.
- (4) Generally a stable fifth lumbar spine closely hugging the sacrum and often indenting its fibrous roof.
- (5) Removal of the neural arch may reveal the 1st sacral roots, or even the cauda equina, compressed.

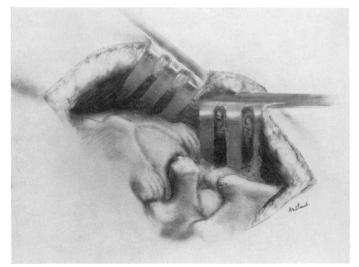


Fig. 4.

Group I.—Operative exposure showing a receding last lumbar spine, defective and grooved sacral facets and defective sacral neural arch.

An excellent illustration of such a case appears in Capener's article (1932). It shows the sacral defect and the receding and closely fitting fifth lumbar spine. But he says "With one morphological defect, it would not be surprising to find other anomalies and this is in fact true in spina bifida. While I do not believe that spina bifida bears any causal relation to spondylolisthesis, I find that these cases having wide gaps in the laminae of the fifth lumbar and sacrum do show much more severe forward displacement."

The latter statement has been confirmed in these cases but on the other hand it seems very probable that spina bifida is a cause of spondylolisthesis.

Lane (1893) operated on such a case and gives an excellent description in the *Lancet* of that year.

"A woman aged 35 was admitted to Guy's Hospital; she worked for a master who had delusions that he was always playing cricket. He carried a heavy stick and in belief that he was hitting the ball, beat the furniture and other objects including the patient's back.

The patient had noticed increasing weakness of the legs over the last six years and increasing deformity of her spine; she had been unable to walk for the last year.

Examination showed deformity of the back and it was noticed that the fifth spine was difficult to feel. There was loss of sensation in both legs and the knee and ankle jerks absent.

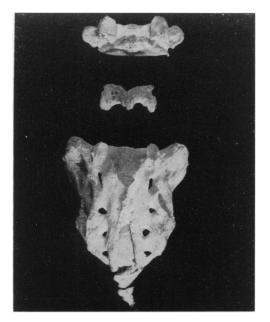


Fig. 5.

Group IIa.—Spina bifida occulta, intact sacral facets and a separated posterior neural arch of lumbar.

An operation was performed for suspected tuberculous caries. The spinous process of the fifth lumbar was found to be placed in the upper part of the sacral canal, quite in front of its normal position. When it was removed it was seen to have compressed the dura mater sheath of the cauda equina. The fifth lumbar nerve root seemed to be compressed between the lamina and the sacrum. The superior margin of the sacrum stood out prominently and the posterior surface of the body of the fifth lumbar vertebra could be felt about three-quarters of an inch in front of its normal position. There was no solution of continuity of the neural arch though at both sides it was deeply channelled by the articular processes "

## **Group II**

This group is the most common and it differs essentially from Group I in that there is no deficiency of the facets. The inferior articular facet remains in its correct anatomical relationship to the superior facet below. Something happens in the pars interarticularis to allow the body to slip away from the posterior part of the neural arch, either an attenuation of the bone, as in Group I, or a break, and frequently both. The characteristics rather differ according to the level of the slip and the age of the patient.



Fig. 6.

Group III.—Left: X-ray immediately after accident shows break of pars interarticularis and pedicle of fourth lumbar vertebra, but no luxation. Right: X-ray three months later shows a healing fracture and luxation of the fourth body.

At the lumbo-sacral level the average age is lower, it is more likely to be associated with spina bifida occulta and there is often attenuation of the bone. At the higher level the lesion resembles more a fatigue fracture with sclerosis and new bone formation, the degree of slip is moderate and there is disc degeneration. These two types, variations of the same group, have been termed Groups IIa and IIb. The bony lesion over the whole group is in the pars interarticularis, the facets, the pedicle and the posterior part of the neural arch remaining normal. The lesion is a fracture secondary to attenuation, fatigue, or both.

At operation the posterior part of the neural arch is found prominent posteriorly and loose, whereas the one above has slipped forward.

## **Group III**

This is a small group due to immediate traumatic fracture of the neural arch which later allows forward luxation. Immediate fracture dislocations have not been included in this group, as this displacement could hardly be described as slipping, but really the distinction is only a matter of degree. It is a difficult group to isolate from Group II and only three obvious cases have been selected. These show, in one case, a fracture running down into the pedicle with pictures taken before and after slipping. The other two show other fractures of the neural arch and subsequent attempted healing. All three had a dramatic history of injury.

### **Group IV**

This group almost invariably occurs at the lumbar 4/5 level and always in middle-aged or old people with severe osteoarthritis of the articular facets.

The original lesion occurring earlier in life is a soft tissue instability and in spite of the additional strain the pars interarticularis holds, with the result that there is severe wear and tear at the interarticular joint. When these degenerative conditions become severe enough facet deficiency results and the inferior articular facets of the vertebra above luxate through the superior ones of that below.

This is seldom possible at the lumbo-sacral level because the superior articular facets of the sacrum face almost directly backwards, but at the 4/5 level they are turned inward more and this allows such a set of circumstances to occur.

This type was first described by Junghanns (1930) as pseudospondylolisthesis. He remarks on the peculiar fact that it always occurs at the 4/5 level and that there is no lesion in the pars interarticularis and marked osteoarthritic changes of the intervertebral facets. He does not offer any definite cause for the luxation but thinks it may be due primarily to an abnormally obtuse angle of the pedicle with the rest of the neural arch. This type has been described in detail by McNab (1950) who reported 22 cases. He upholds the explanation that there is an increase in the angle between the pedicle and the inferior articular facet. This has not been confirmed in the present series.

Five cases have been operated on and in every one the facets of the vertebra above have slipped between the facets below owing to severe degeneration of the joint surfaces. In no case was there a break in the neural arch.

#### Group V

A small group in which instability is due to bone disease. There were four cases in this series, two from Paget's disease, one from tuberculosis, and one an achondroplastic.

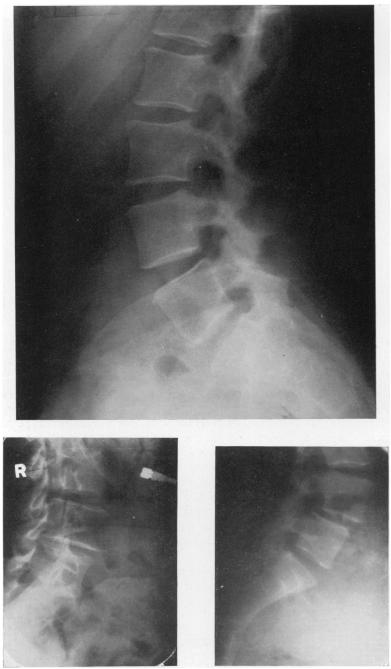


Fig. 7.

Group IV.—A lady whose lumbar spine was X-rayed for backache in 1940, again in 1948 (above) and at both times no abnormality seen. X-ray taken in 1953 (below) shows moderate slip at lumbar 4/5 level and the oblique view shows arthritic change in the joint and an intact neural arch.

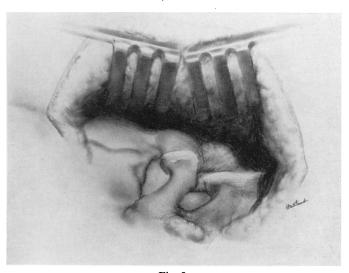


Fig. 8. Group IV.—Operative exposure shows a receding fourth spinous process and neural arch with enlarged osteoarthritic joints.

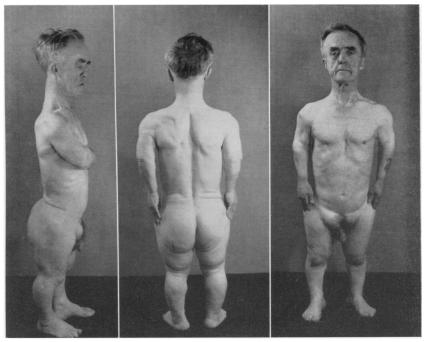


Fig. 9. Group V.—An achondroplastic with spondylolisthesis.

## Nerve Root and Cauda Equina Compression

Many cases of spondylolisthesis remain symptomless and it is impossible to give an accurate estimate of the percentage of all cases that do experience pain. It seems likely that pain arises either from the ligaments, joint capsules and other mesodermal structures or from the nerve roots themselves.

In this series 29 had objective signs of nerve root involvement and one had a complete lesion of the cauda equina.

These figures show the relatively high percentage of nerve root involvement at the lumbar 4/5 level compared to the lumbo-sacral level. That is understandable when the difference of the nature of the lesion is considered. At the lower level there is widening of the spinal canal and the root is less likely to be interfered with by changes around the lesion of the pars interarticularis and around the intervertebral joint.

Group	Level	Number of Cases	Number with Nerve Root Signs	Percentage		
1 $2a$ $b$ $3$ $4$ $5$	Lumbo-sacral Lumbo-sacral L. 4/5 L. 4/5	41 88 22 3 46 4	5 1 (cauda equina) 7 12 	$ \begin{array}{r} 12.2 \\ 2.4 \\ 5.6 \\ 31.8 \\ 26.1 \\ \end{array} $		

NERVE ROOT INVOLVEMENT IN SPONDYLOLISTHESIS

## The Cause of Spondylolisthesis

It is reasonable to suppose, when studying the anatomy of this area, that the lower lumbar vertebrae have a great potential tendency to slip forward, especially when the lumbosacral angle is increased by abnormal forward curving of the sacrum. The most obvious block to forward slipping is the hooking of one neural arch behind another and the greater the lordosis the more will be the hold of the articular facets and the more will be the weight taken by them. Any lesion of the bone which occurs between the inferior articular facet and the body would allow forward slipping and because a lesion so often occurs here, it has been thought, perhaps wrongly, to be the cause of spondylolisthesis.

This bony block of one facet behind another, is, it seems, insufficient to hold the forward thrust of a vertebra perched at such a precarious angle without the binding strength of strong soft tissues. A continual but slight over-tilting forward during stress would throw great strain on the inter-locking facets and tend to precipitate any latent developmental defect. This type of instability could be caused by deficiency of :--

- (1) The lumbo-dorsal fascia.
- (2) The intervertebral disc.

Having described the different types of spondylolisthesis, the main object remaining is to discuss the etiology and to show that the typical break of the neural arch is not a congenital defect nor is it the cause of spondylolisthesis but like spondylolisthesis itself, a complication of instability.

## The Neural Arch

In Group I there is generally no lesion of the pars interarticularis and in Group IV never such a lesion. Yet in Group I the degree of slipping is greater than in any other group. A close study of the morbid anatomy at operation reveals that the slipping is due to facet deficiency, in the former due to congenital defect and in the latter due to severe degenerative changes. This conception is borne out by careful study of the X-rays.

In Group II—the largest group—and occurring at any of the low lumbar levels—there is no such deficiency of the articular facets; they remain in their correct anatomical relationship. The obvious assumption is that the lesion which so often occurs in the neural arch, in this group, is the cause of the instability and subsequent slipping. "How can there be a slip without some bone lesion between the inferior articular facet and the vertebral body?" is a reasonable question but in this group of a 110 cases, there were 10 without a break in the neural arch.

Many think the defect between the facets to be congenital, due to a double centre of ossification. In the latter half of the last century various workers stated that they had observed a double centre of ossification in the neural arch and some were even of the opinion that it was an atavistic deformity reminiscent of the separation of the vertebral body and arch found in certain reptiles.

Willis (1923) examined several foetuses and came to the conclusion that the minute isthmus seen in the centre of ossification was due to a nutrient vessel. Friberg (1939) substantiated this work and showed how ossification in one centre started very early on in foetal life in the region of the pars interarticularis, to assure as it were priority of strength to a would-be harassed area.

Various authorities including Professor Wood Jones (1953) have stated that there is no embryological or anatomical basis to believe that the neural arch is ever developed from two centres.

If the lesion was congenital and the cause of slipping :---

- (1) It would be present in at least all cases where the facets were intact—it is not in 10 cases of this series.
- (2) The two bone ends would be rounded and smooth but they are not, they are jagged and hypertrophied.
- (3) The bone adjoining the lesion would be normal, but it is attenuated or thickened, sclerotic and rough. Sections taken from operation cases show that the bone adjoining the lesion is markedly sclerotic compared to a normal neural arch.



Fig. 10. The site of the break in the neural arch is indicated by ragged bone projection.



# Fig. 11.

X-ray of a specimen from St. Mary's Hospital. It shows a forward slip of the body of three-quarters of an inch, yet the lesion in the pars inter-articularis, hardly seen in this view, has only one millimetre separation of its edges. Luxation is due mostly to attenuation of the bone.

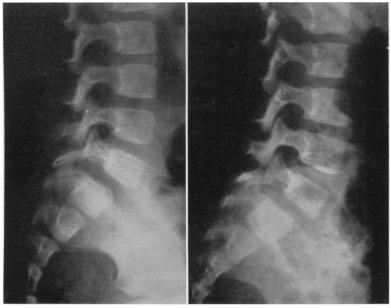


Fig. 12.

X-rays of the same child's spine (*left*) aged four; (*right*) aged six. A crack in the neural arch has appeared in the second picture.

- (4) The degree of slipping of the body would be similar to the distance of separation at the break. An illustration shows a specimen from the Pathological Museum of St. Mary's Hospital. There is a slip of the body of half an inch and although there is a fissure across both sides of the neural arch there is only minimal separation indicating that it is the attenuation of the bone that has allowed luxation forward.
- (5) The lesion would be present at the first sign of slipping, but in young children the slip has been noticed to appear before the break.
- (6) The anterior part quietly slipping away from the rest would leave the spinous process firmly anchored to the vertebra below, but at operation it is found to be quite loose, bound neither to the spinous process above nor below.
- (7) There has recently been published a valuable piece of work by Stewart (1953) on the age incidence of neural arch defects.

He examined nearly 800 skeletons which had been collected in Alaska, North and South of the Yukon River and brought to the National Museum in Washington. He found not only that there was a high percentage of neural arch defects but also that the percentage rose constantly with age.

Age	No. of Cases	Cases with Defect	Percentage	
Under 6	23	1	4.3	
,, 12 18	26 69	3	11·5 14·5	
,, 24	77	13	16.9	
,, 30	122 227	21	17·2 33·9	
,, 40 Over 40	242	82	33.9	
Total	786	207	26.3	

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Stewart-examination of Alaskan skeletons for neural arch defect.

This overall percentage of 26.3 is more than four times greater than found generally elsewhere. He found also that the percentage in adult skeletons coming from the more frozen and rocky areas of Alaska was as high as 47.6 per cent.

The rise of incidence with age contra-indicates a congenital defect and the most ready explanation is that these defects are caused by instability

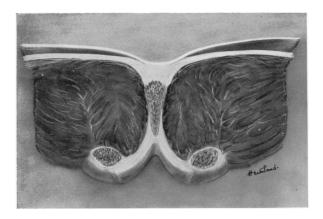


Fig. 13.

A drawing of a transverse section of the erector spinae and adjoining structures. The attachment of the two posterior layers of the lumbo-dorsal fascia to the spinous process forming the supra-spinous ligament.

due to soft tissue injury arising from frequent falls on to the buttocks on rock and ice. Stewart points out that the eskimo works standing up, with the spine flexed forward to allow the hands to reach the ground. This attitude for working in the presence of instability would be prone to give rise to fatigue fracture.

(8) And finally many workers have searched for defects in foetal and stillborn skeletons but none has been found and yet it is common knowledge that there is an overall percentage in adults of at least 4 to 6 per cent.

Although there is no direct scientific proof here to establish the nature of the break, it may be agreed that this indirect evidence is significant. It seems that the bone breaks in an area which takes great strain when instability is present. Bone in the young, like hot metal, is attenuated by longitudinal and up and down strain, but in the adult it breaks by fatigue and is not elongated.

## The Lumbo-Dorsal Fascia

The lumbo-dorsal fascia is a powerful structure anatomically neglected and functionally ill-understood. The posterior layer sweeps over the erector spinae towards the spinous processes and downwards toward the sacrum and where it meets from either side it forms a tough median raphe—the supraspinous ligament. This layer is no ordinary sheet of fascia; it consists of a posterior thinner layer and an anterior thick layer composed of dense bundles of fibrous tissue. The supraspinous ligament binds the spinous processes together, rations the range of up and down movement and anchors them to the median ridge of the sacrum. In the congenitally deficient the ridge of the sacrum is absent and the supraspinous ligament poorly developed so that the mechanism of stabilisation is upset. A potential weakness exists, easily upset by trauma or strain, especially in flexion. This deficiency is protected by a posture of hyperlordosis.

## The Intervertebral Disc

The intervetebral disc acts as a cushion to the seating of the vertebral body. It prevents forward tilting of the vertebra as a whole and possible luxation at the intervertebral joints. The annulus fibrosus binds strongly one vertebral body to another, assisting also in the prevention of forward luxation.

## The Hereditary Factor

Evidence has accumulated to suggest that spondylolisthesis is familial and Friberg (1939) has made a great contribution to this particular subject. He has reported a family of which sixty-one blood relatives were examined and of these no less than fifteen had spondylolisthesis and one spondylolysis. So significant a number in the members of one family can be used as a strong argument in favour of the congenital nature of the lesion of the pars interarticularis. On the other hand it is known that spina bifida occulta is a familial condition and it can equally be argued that the lesion in the pars interarticularis is acquired and secondary to congenital deficiency elsewhere.

Harris (1951) reported the incidence of spondylolisthesis in parent and child and suggests that "If this is not evidence of a congenital origin, it suggests a familial weakness which permits the defect to occur." In this series no extensive investigation has been done but two or more cases have occurred in five instances among siblings. Recently a boy aged ten was sent for consultation for the abnormal posture of his back. He had increased antero-posterior curves and hirsutes over the lower lumbar area. Radiological examination showed deficient neural arch ossification in all five sacral segments and all lumbar segments except the third. There was spondylolisthesis of lumbar five but no break in the pars interarticularis. His brother aged eight came with him and radiological examination showed the neural arches of the sacrum defective and the pars interarticularis of lumbar five attenuated but likewise no sign of a break.

Friberg (1939) reports the case of a girl aged eleven months and here undoubtedly the radiograph shows lesions of the pars interarticularis of at least five vertebrae. These lesions are clean cut, regular with normal bone on either side, quite unlike the usual picture of spondylolisthesis. Can this be a rare phenomena, a true congenital lesion of the interarticular portion of the neural arch ?

Whatever the causes of spondylolisthesis are, and there are many, that at the lumbo-sacral level seems most prone to develop during the few years following walking. At the toddling age, the spine is being adjusted to the upright position, the lumbar region is held in hyperlordosis, the erector spinae contracted and the abdominal muscles relaxed. From this position the spine is insulted frequently by falls on the buttocks with the legs stretched out in front and the lumbar region flexed. Frequent shocks of this nature would be liable to precipitate any latent weakness in the lumbo-dorsal fascia, supraspinous ligament or other posterior spinal ligaments. Instability will result, leaving the last lumbar vertebra insecure. The facets may then become deficient, the arch attenuated and later a break may occur, the classical lesion of the pars interarticularis.

#### SUMMARY

A series of cases of spondylolisthesis has been reviewed and by their nature have been divided into five different groups :----

Group			I	IIa	IIb	ш	IV	v
No. of Cases	••		41	88	22	3	46	4
Average age in years at first examination			26.1	34.9	46.2		65.2	
Spina Bifida Occulta Percentage	•••		41 100	43 48·8	2 9·1		2 4·4	
Break in Pars Interarticularis Percentage	 	•••	8 19·5	78 88·6	22 100		0 0	_

**Group I.**—Slipping occurring at the lumbo-sacral level in the young, often of severe degree. Instability is caused by congenital deficiency of the neural arches of the sacrum and of the posterior ligaments of the spine.

Joint luxation, attenuation of the neural arch and occasionally a secondary break result.

Group II.—Slipping occurring at any lower lumbar level from instability due to congenital or post-traumatic posterior ligament deficiency. In the young the neural arch attenuates and often breaks in the pars interarticularis; in adults a fatigue fracture occurs and is often associated with disc degeneration. The former occurs more often at the lumbo-sacral level, the latter at the lumbar 4/5 level, but in both cases the facets remain intact.

Group III.—Slipping due to instability caused by an acute fracture of the neural arch.

Group IV.—Long-standing soft tissue instability causes degenerative changes in the intervertebral joints. Late in life this may result in facet insufficiency and secondary subluxation forward.

Group V.—Instability caused by bone disease.

## IN CONCLUSION

Spondylolisthesis is due to lumbar instability caused by :---

- (1) Congenital absence of the sacral ridge associated with deficiency of the median raphe of the lumbo-dorsal fascia and other posterior spinal ligaments.
- (2) Congenital or acquired facet deficiency.
- (3) Acquired deficiency of the lumbo-dorsal fascia, other posterior spinal ligaments and the intervertebral disc.

The characteristic lesion of the pars interarticularis is by no means always present. It is secondary to instability and caused by attenuation or fatigue or a combination of both. It is not the cause of spondylolisthesis although its presence may permit an additional degree of slip.

I wish to thank those who have assisted me in the Orthopaedic and Backache Clinics, and the Staff of the Photographic and Secretarial Departments for their kind help.

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