Acta orthop. Scandinav. 37, 367–374. 1966.

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HYPERALGESIA OF THE LUMBAR NERVE ROOTS IN SCIATICA

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Received 7.II.66

Normal peripheral nerves, including the spinal nerve roots, react to compression by loss of function (1). If the compression occurs rapidly, possibly with mechanical deformation and consequent damage to the nerve structure, paraesthesia, radiating and tingling sensations and more or less pronounced pain will result. It is surprising how moderate the pain caused by pressure or a blow on a nerve trunk, such as the ulnar or fibular nerve, can be. Continuous moderate pressure will usually produce only a loss of function, with no pain at all.

In 1946 *Lindahl* (8) demonstrated that symptoms of impaired function of the lumbar nerve roots such as paraesis, areflexia and reduced sensibility occurred in tuberculous spondylitis of the lumbar spine, and that these symptoms were unaccompanied by pain. In cases of tumours of the spinal canal, pain is a common initial symptom, but in at least 30 per cent of the cases of benign tumours there is no pain, and neurologic symptoms of loss of function, mostly paresis, usually appear first (2, 3, 11). It is thus evident that pressure on the spinal nerve roots, as in the peripheral nerves, can be quite devoid of pain.

It is now widely considered that the radiating pain in sciatica it is due to pressure on the nerve roots, usually caused by herniated disks. This pressure may be described as continuous, intermittent or abrasive in type. Since pressure on a nerve root does not necessarily involve pain it would seem reasonable to suppose that the sciatica must be due to some other cause besides. Evidence for this is found in the great range of intensity of the pain in sciatica, even in the absence of herniated disks, and likewise in the wide variation in the sensitivity of the nerve roots to pain on pressure and manipulation during operations for sciatica.

Since disk herniation was "discovered" two main explanations of sciatica have been advanced, namely a mechanical effect, the pain in a nerve root being elicited by pressure on it, and, secondly, inflammation of the nerve. In 1947 *Lindahl* (8) proposed that sciatica could occur through a combination of these two factors; in some cases inflammatory changes, unaccompanied by external pressure, would suffice as the explanation, while in the case of disk herniation the associated mechanical pressure would, of course, extend and intensify the pain and evoke symptoms of functional loss, but could not be the sole cause of the pain.

To study the incidence of pathological changes in the nerve roots, *Lindahl & Rexed* in 1951 took biopsy specimens from the posterior spinal nerve roots at operations on cases of sciatica (10). In 7 out of 10 cases, pathological, usually inflammatory, processes were found in the nerve roots; some of the patients had disk herniation and others not.

In the subsequent discussion on the cause of the pain, *Kelly* (4–7) suggested that the theory of mechanical pressure did not fit in with a number of clinical, experimental and physiqlogical facts. Sharply criticizing Kelly's views, *Smyth & Wright* (14) maintained that the pressure of a herniated disk could alone account for sciatica. In operations for disk herniation they placed nylon threads round the nerve roots and left them in place for a time after the operation. When the pain was relieved after removal of a herniated disk the threads were pulled so that the nerve roots were subjected to pressure; the typical pain was again elicited.

To examine more closely the sensitivity to pain from the lumbar spinal nerves the author has subjected various lumbar nerve roots to a rapid increase in pressure by an epidural injection of isotonic saline in connection with back operations.

MATERIAL AND METHOD

The experiments were performed on 20 patients operated on for disk herniation and on 4 patients where a posterior fusion was performed for chronic back pains but not sciatica. The patients were anaesthetized with a local infiltration anaesthesia adjacent to the spinous process and vertebral arches. After exposing these on both sides, the epidural space was punctured with 0.7 mm thick needle, care being taken to avoid coming into contact with the nerve roots and passing intradurally. As a rule, the 3 lower lumbar intervertebral spaces on each side were punctured. When the needle was in position 10 ml of isotonic saline was injected as fast as possible so that a rapid increase in hydrostatic pressure was obtained around the needle. The

patient was told that an injection was about to be given and was asked to say whether it caused any pain and, if so, where. After the pain had ceased—usually after a minute or so—the next space was punctured in the same way. In the various experiments the space where the clinical findings or myelography indicated that the herniated disk would be located was injected alternately first and last. The clinical syndrome and surgical findings are given in Table 1.

Table 1. Clinical syndrome and findings at operation in the cases of sciatica tested.

Syndrome	No.	Positive findings	Negative findings
S_1	9	7	2
L_5	7	5	2
L_4	1	1	0
Mixed	3	1	2
Total	20	14	6

RESULTS

As, with few exceptions, the patients' reaction was remarkably uniform a detailed account of all 24 cases would appear to be superfluous. In connection with the injections, all the patients with typical root syndrome stated that they felt a pain in the usual area *irrespective of where the injection was made.*

The intensity of pain was apparently related to the pre-operative severity of the symptoms. When the injection was given at a distance from the root the pain was slightly milder than when it was given just over the root, and in some cases the pain on injection at a distance of 3 spaces was very weak. In none of the cases was there pain on the sound side, where one half of the injections were given.

In cases of back pains without sciatica the injection sometimes gave rise to mild diffuse back pains, and in two cases to weak radiating pains on the same side as the injection. Usually there was no pain.

In the 3 cases of atypical sciatic pain, implying radiation only in the thigh and lower leg but not the foot, or where more than one root appeared to be involved, the injection also elicited the characteristic pains, but the component reproduced depended on the level of injection. In these cases, too, no pain was felt on the sound side. In a number of leading questions 10 of the patients were asked whether there was not now pain in the sound leg, and the injection was repeated on the sound side with a needle of larger calibre (1.0 mm), at a faster rate and with

a larger volume (20 ml). In 4 of these cases the patient reported a slight sensation in the sound leg but said that this could hardly be described as pain, and could by no means be compared with the pain felt simultaneously in the usual region.

The patients recorded pain sensations that were alike in cases with and without herniated disk.

DISCUSSION

It would seem obvious from these experiments that a normal lumbar nerve root does not react with an appreciable pain sensation to an increase in diffuse hydrostatic pressure produced in its vicinity, an increase in pressure that, as was seen, resembled closely that which may be produced *via* the veins in coughing or blowing the nose. It is also evident that the nerve root or roots that are responsible for the sciatic pain are abnormally sensitive to such pressure. That such a marked variation in the sensitivity of the nerve roots also accompanies mechanical deformation during operative insensitivity of the sound nerve roots has, however, not received much attention in the discussions on the cause of sciatic pain. It is also evident from these experiments that "hyperalgesia" of the nerve roots occurs in cases of sciatic with and without herniated disk.

It might be asserted that in the cases of negative findings at operation, disk herniation may have been present but overlooked, perhaps because it was located lateral to the intervertebral space. While this cannot, of course, be proved wrong, it must be borne in mind that all 6 such subjects were relieved of pain after the operation, and at a follow-up examination 6 months later they still had no pain in the legs; there is here no evidence that the pain was due to pressure from a possible residual herniated disk.

It is a familiar experience from all large operation series that the results at a follow-up of the "negative" operations are good but not quite as good as after the "positive" operations. *Senning & Sjöquist* (13) reported 50 per cent satisfactory results after negative explorations, and *Warris* (15) 71 per cent. This would suggest that the operation for disk herniation can have a therapeutic effect that is not associated with the actual removal of the offending disk, and in fact cannot always be attributed to the relief of pressure in any other way (for instance, by laminectomy). This therapeutic effect might well, it

seems, be due to an influence on the hyperalgesic state of the nerve—"a nonspecific irritation therapy".

We have now the following facts: (1) Pressure on normal nerves does usually not produce pain; (2) in sciatica the nerve roots are hypersensitive to pressure, to which they react with pain; (3) disk herniation or any other source of pressure is not always found in sciatica; (4) the surgical treatment for sciatica includes a factor that is unassociated with removal of the offending disk but which still banishes pain in at least 50 per cent of the cases.

This evidence suggests the following conclusions: (1) Symptoms of loss of function in sciatica are due to compression of one or more nerve roots. (2) The mechanical component in sciatica is usually extremely definite (movements, loading and function aggravate the pain; removal of a herniated disk results in improvement). (3) The pain in sciatica cannot be ascribed solely to mechanical pressure; there must be also a pathological alteration of one or more nerve roots, which results in hyperalgesia. (4) The severity of the sciatic pain is related mainly to the severity of the hyperalgesia. Most cases of sciatica are mild, and then a herniated disk is neither sought nor found. (5) In sciatica the nerve root environment can be anatomically normal but mechanical compression of the nerve roots aggravates the pain, and symptoms of functional loss may then be elicited. (6) In hyperalgesia the normal physiological contact with a normal anatomic environment can suffice to account for aggravation of the pain by mechanical factors.

This explanation of sciatic pain is not, of course, proven in every respect, but it would seem to be more consistent with our present knowledge of sciatict than the theory relying on mechanical compression as the sole cause of the pain.

Smyth & Wright's (14) experiments with nylon threads have not proved that pressure is the sole cause of sciatica. Their patients obviously had hyperalgesic nerve roots, the traction on which elicited pain. Normal nerve roots would definitely not have reacted in the same way. What, then, is the cause of the "hypothetical hyperalgesia", and why is there reason to consider it at all when the role of mechanical pressure is still thought to be the major one?

It is, of course, obvious that our power to eliminate entirely any mechanical effect on the lumbar nerve roots is limited on both theoretical and practical grounds. Every orthopaedic department has a not inconsiderable group of patients whose sciatica has not been relieved. The discovery of the cause of the hyperalgesia and some way of treating

it would constitute a therapeutic advance that would benefit not only the cases that have resisted our present therapy but also many who have benefited from an operation but who might have been helped more easily without surgery.

The cause of the hyperalgesia remains to be found. In an earlier paper *Lindahl* (9) showed that the negative pressure in the epidural space normally present is absent in sciatica. *Lindahl & Rexed* (10), among others, have shown that the nerve roots are the site of pathological changes. In some cases the cause may lie with infection. A discussion of this possibility and appropriate references are to be found in the paper by *Lindahl & Rexed.*

It would seem conceivable also that an inflammatory process in the epidural space can result from outward penetration of disk tissue, and perhaps also of metabolic products, from the nucleus and annulus. Evidence pointing in this direction has been reported by *Olsson* (12), who has shown that such an inflammatory reaction was a more or less regular phenomenon in disk hernia in the dog. Such an inflammatory reaction, with subsequent hyperalgesia of all the nerves in the region, might be a common cause not only of sciatica but also of the much more common lumbago.

From a practical standpoint this approach to sciatica is not so far removed from the conventional one, and it is perhaps the disk that is in one way or another the most important factor.

SUMMARY

Various theories on the cause of sciatic pain are discussed. There are two prevalent views on the cause, namely mechanical pressure on the nerve roots and an inflammatory process.

With the object of throwing more light on this problem the author has examined the sensitivity of the lumbar nerve roots to hydrostatic pressure in connection with back operations by rapid injection of physiological saline epidurally. In cases of sciatica with or without disk herniation, one, or possibly more, nerve roots reacted abnormally to pressure, and the typical pain was elicited, while for the other nerve roots, like those in cases without sciatica, there was no pain reaction at all.

It is suggested that in sciatica there is almost invariably hyperalgesia of the nerve roots, and that this is a more or less necessary condition for the sciatic pain. It is recognised that a mechanical factor is of prime

importance, but this is not inconsistent with the hyperalgesia being an equally important factor in the explanation of the disease.

RESUME

Différentes théories sur la cause des douleurs sciatiques sont discutées. Il y a deux opinions qui prévalent, à savoir la pression mécanique sur les racines nerveuses et un processus inflammatoire.

Dans le but d'éclaircir ce problème, l'auteur a examiné le sensibilité des racines du nerf lombaire à une pression hydraulique en relation avec des opérations du dos par une injection rapide d'eau salée physiologique épiduralement. Dans les cas de sciatique avec ou sans hernie discale, une ou peut-être plusieurs racines nerveuses réagissent anormalement à la pression et la douleur typique est provoquée alors que pour d'autres racines nerveuses, comme celles dans les cas sans sciatique, il n'y a aucune réaction douloureuse.

Il est suggéré que dans la sciatique il y a presque invariablement une hyperalgésie des racines et que c'est une condition plus ou moins nécessaire à la douleur sciatique. Il est reconnu qu'un facteur mécanique est d'une importance primordiale, mais cela n'est pas contradictoire avec l'hyperalgésie comme un facteur d'une importance égale dans l'explication de cette maladie.

ZUSAMMENFASSUNG

Verschiedene Theorien über die Ursache von Ischiasschmerzen werden besprochen. Es bestehen zwei vorherrschende Ansichten über die Ursache, nämlich die des mechanischen Druckes auf die Nervenwurzeln und die eines Entzündungsprozesses.

Mit der Absicht mehr Klarheit in dieses Problem zu bringen, hat der Verfasser die Empfindlichkeit der Lendennervenwurzeln gegenüber hydrostatischem Druck im Zusammenhang mit Rückenoperationen mittels rascher, epiduraler Injektionen von physiologischer Kochsalzlösung untersucht. In Fällen von Ischias mit oder ohne Diskushernia reagierte eine oder möglicherweise mehrere Nervenwurzeln abnormal auf Druck und der typische Schmerz wurde hervorgerufen, während die anderen Nervenwurzeln, ebenso wie jene in Fällen ohne Ischias keinerlei Schmerzreaktion aufwiesen.

Es wird angenommen, dass bei der Ischias fast immer eine Hyperalgesie der Nervenwurzeln vorhanden ist und dass dies eine mehr oder weniger notwendige Bedingung für den Ischiasschmerz ist. Man er-

kennt, das der mechanische Faktor äusserst wichtig ist, aber das schliesst nich aus, dass die Hyperalgesie einen ebenso wichtiger Faktor zur Erklärung der Erkrankung darstellt.

REFERENCES

1. Aird, R. & Pfaffmann, C. (1947) Pressure stimulation of peripheral nerves. *Proc. Soc. exp. Biol. Med.* **66**, 130–139.
2. Broanger, B. (1953) Spinal neurinoma, a clinical study comprising 44 cases. *Acta psychiat. et neurol.*, Suppl. 85.
3. Elsberg, C. A. (1925) Tumors of the Spinal Cord. Paul B. Hoeber, New York.
4. Kelly, M. (1948) Brachial neuralgia. *Brit. med. J.* **4547**, 416.
5. Kelly, M. (1948) Cervical root & irritation. *Lancet* **154**, 384–387.
6. Kelly, M. (1956) Is pain due to pressure on nerves? *Neurology* **6**, 32–38.
7. Kelly, M. (1946) Interstitial neuritis and pressure theory of pain. *Med. J. Austr.* **33**, 325–333.
8. Lindahl, O. (1947) Om ischiassjukdomens patogenes. *Svensk Läkartidning* **51**, 2389–2393.
9. Lindahl, O. (1952) The pressure in the epidural space in operated cases of sciatica. *Acta orthop. scand.* **22**, 232–236.
10. Lindahl, O. & Rexed, B. (1951) Histologic changes in spinal nerve roots of operated cases of sciatica. *Acta orthop. scand.* **20**, 215–225.
11. Oddson, B. (1947) Spinal Meningioma. Munksgaard, Copenhagen.
12. Olsson, S.-E. (1951) On disc protrusion in dog. *Acta orthop. scand.*, Suppl. 8.
13. Senning, A. & Sjöqvist, O. (1947) Senresultaten vid diskbråck. En efterundersökning av 400 opererade fall. *Nord. Med.* **34**:**2**, 1128–1129.
14. Smyth, M. & Wright, V. (1958) Sciatica and the intervertebral disc. *J. Bone Jt Surg.* **40–A**, 1401–1418.
15. Waris, W. (1948) Lumbar disc herniation. *Acta chir. scand.*, Suppl. 140.