

THE POST-LAMINECTOMY SYNDROME

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INTRODUCTION

The post-laminectomy syndrome is defined as a term encompassing all persistent severe symptoms following operation on a lumbar disc¹. It is thought to be due both to instability of the motion segment and adhesions in the spinal canal.

Spangfort² reviewed the results of 2504 laminectomies, selected for the study because they had clear-cut signs of nerve root entrapment. He found that complete relief of both leg and back pain post-operatively occurred in only 60% of cases. Failure to relieve pain in these cases of nerve root entrapment was attributed to one or more of the following:

- The exploration having been carried out at the wrong level
- A second disc prolapse having been overlooked
- The nerve root continuing to be compressed by the posterior intervertebral joints
- Spinal stenosis causing pressure on the nerve root
- Rarely, because of an extraforaminal lateral disc herniation³.

Wilkinson⁴ prefers the term "failed back syndrome" because the syndrome is not always preceded by an incorrectly performed operative procedure but can also occur following correctly performed surgery.

Should we, therefore, look beyond surgery for other perpetuating causes of the residual pain? Why does pain continue post-operatively? Is this pain neurogenic or musculoskeletal? Can we treat this condition effectively?

Theoretical assumptions

As far back as 1983 Travell and Simons⁵ suggested that myofascial trigger points which have become activated pre-operatively are likely to remain active post-operatively, and are therefore likely to continue to cause pain long after the nerve root has been decompressed satisfactorily. To test this assumption I have assessed the presence of trigger points and the patterns of pain pre- and post-operatively in patients treated in a surgical spinal unit. Pre-operatively, trigger points producing pain were identified but deactivation of these trigger points did not have a lasting effect. Post-operatively the trigger points were found to be situated in the same distribution and deactivation over several treatment sessions provided lasting relief of pain.

The purpose of this article is not to discuss the research as such, but to examine theoretical assumptions regarding the mechanisms of pre- and post-operative pain which may explain the results of the study.

Pre-operative back pain

The development of disc herniation at the level of the lower lumbar segments is assumed to set the pain process in operation. Altered segmental function provokes protective spasm in the muscles surrounding the joints, resulting in segmental imbalance locally. This results in compensatory reaction and adaptation in other segments so that finally the whole spinal system is reflexly involved in the activation of trigger points⁶. A cycle of strain and resultant pain is set up which magnifies and reinforces this mechanism as the local pathology progresses. Secondary muscle and fascial shortening occurs which is responsible for generalised muscle stiffness and restriction of joint movement and, in turn,

SUMMARY

Pre-operative patterns of pain and altered function have been found to persist post-operatively following disc surgery. This article explores theoretical concepts which may provide a rationale for successful post-operative treatment.

restrictive involvement of joint structures. It is this low back stiffness, in addition to pain, which causes the patient to develop abnormal patterns of posture and gait.

Pre-operative leg pain

Simultaneous with the mechanisms described above, entrapment of the emerging nerve root by the disc prolapse gives rise to radicular pain in segmental distribution in the leg. The muscles supplied by this nerve root respond by going into spasm. As in the case of local back pain, prolonged spasm leads to shortening and to the development of trigger points which in turn generate their own patterns of referred pain.

Post-operative pain

The two causes of the post-laminectomy syndrome have been stated to be motion segment instability and the presence of adhesions in the spinal canal.

It is possible that motion segment instability is not only due to expulsion or operative removal of the intervertebral disc, but also to imbalances in the intrinsic musculature due to the pre-operative development of the cycle of altered segmental function as described above. This would explain the persistence of abnormal patterns of posture and gait post-operatively.

Adhesions are known to develop in the operative area as a result of fibrous organisation of bleeding and exudate. These adhesions involve the dura and nerve roots by restricting their movement within the spinal canal and the intervertebral foramina, leading to adverse neural tension. Such adhesions may also produce compression of the nerve root post-operatively, resulting in pain. It is striking that the somatic pattern of distribution down the leg post-operatively so closely mimics that of the original radicular pain.

Adverse neural tension can also be generated by restrictions at mechanical interfaces occurring as a result of the altered segmental function and subsequent muscle spasm and myofascial shortening. Even minimal pressure compromises the blood supply to the nerve and affects the function of the nervi nervorum supplying the nerve itself. Increasing pressure affects axonal flow and nerve conduction.

Treatment considerations

From the above it is apparent that the causes of post-operative pain may be multiple, involving not only instability of the motion segment and adhesion formation, but also the perpetuation of altered segmental functions and trigger points activated pre-operatively. Treatment has to be directed at all the involved structures – joint, muscle, fascia and nerve – and experience has shown that better results are obtained if such treatment is instituted immediately post-operatively.

The aims of treatment are to relieve back and leg pain and to restore maximum function. Since shortening and stiffness may be

the major causes of post-laminectomy pain, treatment must be directed at releasing the structures involved.

Conservative measures

Drugs, a corset and bed-rest are appropriate in the early stages and are beneficial in restricting the inflammatory process and thereby relieving immediate post-operative pain.

The joints

Passive intervertebral joint mobilisation aims to mobilise adherent structures within the central canal as well as in adjacent lateral canals. Specific techniques for the lower lumbar motion segments are directed initially at relieving pain, utilising small amplitude movements, and progress to deeper grades to reach the end of available range and treat stiffness.

Where indicated, generalised and localised mobilising techniques are applied to joints at other levels of the axial skeleton in order to treat restrictions which have resulted from compensatory mechanisms.

The soft tissues

By releasing the restrictions in soft tissues surrounding the affected vertebral segments and locating and releasing the offending trigger points in the leg, pain can be relieved. The resultant muscle lengthening spontaneously facilitates strengthening of the weakened muscles and aids in restoring normal segmental function. After release of trigger points, joint mobilisation and myofascial stretching techniques are used to restore normal mobility. Muscle strengthening is directed towards stabilisation of the lumbar motion segments in the normal physiological lordosis by activation of the intrinsic musculature.

Following surgery, neural mobilisation is of particular importance in restoring spinal and limb mobility. Restriction of neural movement occurs not only in the spinal canal and adjacent structures, due to adhesions, but also at the mechanical interfaces within muscles and ligaments and at bony attachments due to the altered function and restricted movement pre-operatively. Mobilisation of the nervous system plays an important part in influ-

encing the tension and movement of neural structures, thus improving vascular dynamics, axonal transport systems and the mechanics of neural fibres and nervous connective tissue. Improved blood supply to the nerve itself may play an important part in restoring normal nerve conduction and relieving pain.

Compensatory mechanisms

Compensatory postural mechanisms may not in themselves be symptomatic, but will prevent normal alignment and function of the lumbar spine, leading to faulty posture, uneven weight distribution, altered patterns of movement and gait and secondary decreased mobility of the related soft tissue structures. These compensatory mechanisms may persist after removal of the original cause and, unless tackled, will in themselves perpetuate pain. Successful long term results depend upon effective postural re-education.

Conclusion

The successful management of post-laminectomy syndrome is a challenge to the physiotherapist, both in her evaluation and differentiation of the underlying causes and in the effective treatment of the involved structures. Only early and intensive treatment of all structures involved will achieve a successful result.

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