The functional relevance of neurological recovery 20 years or more after lumbar discectomy

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There are relatively few publications that evaluate neurological recovery after lumbar disc herniation. Outcome is usually measured by pain scores and indicators of function, such as the Oswestry Disability Index\(^1\) or the Roland-Morris questionnaire.\(^2\)

The study by Mariconda et al.\(^3\) in this issue is welcomed as an excellent attempt to determine the late outcome of patients who have a neurological deficit at the time of lumbar discectomy and the impact of a persistent deficit on function and the quality of life. Although they were able to trace less than 50% of their patients, those seen were followed up for more than 20 years. Most of the motor recovery would be expected to occur during the first three years, so the real value of the long follow-up is to evaluate the impact of persistent weakness on overall function.

In keeping with other studies,\(^4\)\(^-\)\(^10\) sensory deficit was excluded because it cannot be measured as objectively as motor loss, but it is a pity that the authors chose to exclude patients with complete myotomal paralysis, as the outcome in this group might have been of greater interest and the implications for late disability and short form-36 scores would have been more profound.

The authors found that the motor paresis recovers in 75% of cases, and that the reflexes revert to normal in 60%. These findings are in accordance with those of an earlier study by Postacchini et al.\(^4\) and in that of Jönsson and Strömqvist.\(^5\) Those with a persistent motor deficit will have little long-term disability or impaired quality of life as long as their pain has been relieved. These findings provide a new perspective for clinicians treating patients with sciatica and neurological dysfunction and should serve to make surgeons less concerned about minor neurological deficits which are still evident after the conclusion of treatment.

As a consequence, patients can also be given reassuring advice about the long-term prognosis. Implicit in the title of the paper is the suggestion that surgical removal of the disc material was responsible for the neurological recovery, and spinal surgeons certainly consider motor deficit to be an important indication for operative treatment. However, it must be remembered that lumbar disc herniation is a self-limiting condition and that surgical intervention is only a single event in the natural process of healing. Discectomy should therefore be viewed as one factor in determining the clinical outcome and neurological recovery of the patient. Surgeons all experience the satisfaction of seeing neurological improvement early after operation, but they also see untreated patients undergo spontaneous resolution of weakness and numbness. The natural history of massive disc herniation is of resolution.\(^11\) In support of the assertion that neurological deficit requires decompression, a small clinical study on patients undergoing discectomy by Takahashi, Shima and Porter.\(^12\) showed that the severity of neurological deficit correlated well with the pressures measured by a pressure transducer placed between the nerve and the herniated disc, but the levels recorded did not correlate with limitation of straight leg raising.

Although there is little in the recent literature concerning recovery of nerve function, there are some good historical studies reporting the incidence of neurological compromise resulting from lumbar disc herniation. Spangfort\(^6\) undertook a computer analysis of 2504 patients treated by discectomy and found that there was a diminished ankle reflex in 30%, weakness of dorsiflexion of the ankle or toe in 30%, a combination of the two in 17%, and in only 21% was there no detectable motor deficit. Only 4% had alteration of the
knee reflex, indicating that involvement of higher lumbar roots is relatively uncommon. Andersson and Carlsson, after excluding cases of cauda equina syndrome, found evidence of footdrop in 65 patients following 372 operations for lumbar disc herniation over a period of ten years. This persisted in 50%, and the outcome was not related to rate of onset, the time interval to operation or the age of the patient. Hakelius studied weakness of dorsiflexion in 66 patients following operation and 119 treated conservatively. He found similar ratios of normal strength, partial weakness and profound weakness in the two groups. He concluded that discectomy provided relief of pain, but did not make any significant difference to recovery of nerve function.

The authors of the current paper have quoted the study by Weber of patients with sciatica due to lumbar disc herniation which showed a clear benefit for surgery over conservative treatment at one year, with little difference in the outcome on assessment after four and ten years. However, they have not mentioned the equally important, earlier, prospective randomised study by Weber, which involved the use of strain gauges to measure changes in muscle power in 128 patients in groups treated conservatively and by operation, comparing them prior to treatment and at one year afterwards. Motor recovery was the same for each group, with no improvement in outcome in those treated by operation.

Jonsson and Strömqvist studied severe weakness of the extensor hallucis longus in a series of 187 patients treated by lumbar discectomy and found an incidence of 11%, of which 70% to 75% showed recovery (one-third complete and two-thirds partial) after operation.

Osterman et al in a small randomised controlled trial, showed no benefit of surgery over conservative treatment in patients with sciatica and no pressing need for operative treatment. Millisdotter, Strömqvist and Jönsson, in a study of 71 patients with L4-5 or L5-S1 disc herniations, found that proximal and distal weakness was common, that the degree of motor deficit correlated with postoperative pain, and that muscle strength improved over a period of a year.

In all the quoted studies cases of cauda equina syndrome were excluded.

Incorporating the current study into the evidence base, the state of our knowledge about the effects of lumbar disc lesions on nerve function can be summarised as follows:

1) Mild motor paresis is very common in lumbar disc herniation, occurring in approximately 50% of cases;

2) Mild to moderate paresis will recover after surgery in 75% of cases, and most of this will occur during the first year;

3) Even where cases of mild to moderate weakness fail to improve, there is little detriment to late function and quality of life;

4) Marked extensor weakness, including complete footdrop, occurs in 5% to 10% of cases, and there is a potential for recovery in approximately half, with or without treatment;

5) In those with severe extensor weakness, especially painless footdrop, discectomy does not improve the outcome;

6) After discectomy, pain relief occurs first, followed by recovery of motor function and finally by improvement in sensation. Persistence of minor dermatomal sensory deficit is common and usually trivial;

7) Except for the cauda equina syndrome and with clearly evolving motor weakness, unremitting radicular pain (sciatica and cruralgia) must be considered to be the main indication for discectomy.

References