# LUMBAR SPINAL STENOSIS AND DIABETES

## OUTCOME OF SURGICAL DECOMPRESSION

GIANLUCA CINOTTI, FRANCO POSTACCHINI, JAMES N. WEINSTEIN

*From the Universities of Rome and Modena, Italy and the University of Iowa Hospitals and Clinics, USA*

**We reviewed 25 diabetic (mean age 68 years) and 25 non-diabetic patients (mean age 71 years) who had undergone decompression for lumbar spinal stenosis at a mean of 3.4 years after operation to determine whether diabetes affected the outcome of surgery.**

**The preoperative symptoms were similar in the two groups except that an abrupt onset of symptoms, the presence of night pain and the absence of any posture-related pain relief were recorded only by diabetic patients. Nerve-conduction velocity was slowed in 80% of the diabetic and in 25% of the non-diabetic patients. Peripheral vascular deficiency was diagnosed in 20% of patients with diabetes and in 4% of non-diabetics.**

**The outcome of surgery was similarly successful in the two groups. Mistaken preoperative diagnosis was the cause of failure in three diabetic patients, two with diabetic neuropathy and one with diabetic angiopathy.**

*J Bone Joint Surg [Br]* 1994; 76-B:215–9.
*Received 12 June 1993; Accepted after revision 19 August 1993*

The long-term complications of diabetes mellitus include peripheral angiopathy and neuropathy. The former is responsible for the high incidence of intermittent claudication in diabetic patients (Kreines et al 1985) and the latter for several distinct syndromes characterised by pain and sensory or motor deficits in the legs. These symptoms closely mimic those of lumbar stenosis and there may be a risk of inappropriate surgical intervention in patients with both diabetes *and* spinal stenosis. Furthermore, in the presence of diabetes, a poor surgical outcome might be expected. Diabetic patients have a high incidence of comorbidity which is known to correlate with a poor outcome after lumbar spinal decompression (Katz et al 1991). Moreover, hyperglycaemia can affect the pain threshold (Lee and McCarty 1992) and diabetic microangiopathy can damage the periradicular circulation, the disturbance of which is thought to cause the intermittent claudication in patients with lumbar stenosis (Blau and Logue 1961; Evans 1964).

The outcome of surgery in patients with lumbar stenosis and diabetes has not been investigated. We compared the results of decompressive surgery for lumbar stenosis in diabetic and non-diabetic patients, to identify the clinical features associated with a poor outcome.

## PATIENTS AND METHODS

We analysed two groups of patients operated on for lumbar spinal stenosis; those in group A all had diabetes and those in group B did not.

For inclusion in the study patients had to have clinical symptoms of lumbar stenosis not improved by conservative treatment for more than three months with evidence from a myelogram and a CT scan and/or MRI that there was central or lateral stenosis at one or more levels. Those with diabetes had to have type-I or type-II disease requiring insulin or oral medication which should have started at least three years before the operation. Diabetic patients have, on average, a higher rate of comorbidity than non-diabetics and we therefore selected those non-diabetic patients who also had a high rate of comorbidity in an attempt to match the groups.

There were 25 patients in each group; their mean age at the time of operation was 68 years in group A (37 to 84) and 71 years in group B (58 to 84). There were 14 men and 11 women in group A and 13 men and 12 women in group B.

Their preoperative histories and the results of objective examinations were independently assessed by one of us (GC) by reviewing the medical records. Symptoms of diabetic neuropathy, such as night pain, contact discomfort and sudden loss of weight, were recorded as well as the mode of onset of the symptoms

G. Cinotti, Orthopaedic Surgeon
I Orthopaedic Clinic, University of Rome 'La Sapienza', Piazzale Aldo Moro 5, 00185 Rome, Italy.

F. Postacchini, Professor of Orthopaedic Surgery
Orthopaedic Clinic, University of Modena, Largo del Pozzo 71, 41100 Modena, Italy.

J. N. Weinstein, DO, Professor and Director
Spine Diagnostic and Treatment Center, Department of Orthopaedic Surgery, University of Iowa Hospitals and Clinics, 200 Hawkins Drive, 1201-1 RCP Iowa City, Iowa 52242-1088, USA.

Correspondence should be sent to Dr G. Cinotti.


0301-620X/94/2734 $2.00

and whether they were posture-related. Intermittent claudication was measured by the distance that the patient could walk without symptoms in the legs.

Preoperatively, we examined each patient for nerve-root tension signs, range of spinal motion, motor and sensory deficits and activity of the deep tendon reflexes. Preoperative EMG and nerve-conduction studies were done in 20 patients of group A and in 16 of group B.

We recorded the type of diabetes (I or II), the time interval between its diagnosis and the spinal operation, and the presence (or suspicion) of diabetic neuropathy.

Comorbidity was measured on the Cumulative Illness Rating Scale (Linn, Linn and Gurel 1968), three classes being distinguished on the basis of the total score (Katz et al 1991).

The patients were examined at one to six months following surgery and after an average of 3.4 years (2 to 7.3). At each attendance they answered a questionnaire to assess pain and function. The replies were compared with the objective evaluation and any discrepancies between the two were noted. The clinical outcome was rated as excellent if there was complete relief of leg pain and full resumption of normal activities. It was good if there was occasional leg pain, but normal activities were possible without medication; fair if there was moderate pain requiring medication, or mild restriction of normal activities; and poor if there was little or no improvement after surgery or if normal activities were seriously restricted.

Statistical analysis was performed by the chi-squared test.

## RESULTS

**Preoperative findings.** The preoperative clinical and electrophysiological findings in the two groups are summarised in Tables I and II.

Five patients (20%) in group A experienced night pain, two (8%) reported an abrupt onset of symptoms and four (16%) experienced no relief from pain with change of posture. These three symptoms were not reported by any of the patients in group B. Both the patients with an abrupt onset of symptoms and two of the four who had no postural pain relief had poor results from surgery.

Intermittent claudication occurred at a walking distance of one block in seven patients in group A and in nine in group B and at more than one to five blocks in 14 in group A and 10 in group B. Nerve-conduction velocities were slowed in 16 patients (80%) in group A and 4 (25%) in group B. EMG showed denervation or an increase in spontaneous activity in the paraspinal and/or lower limb muscles in 17 patients (85%) in group A and 14 (88%) in group B.

The diabetes was type I in four patients and type II in 21. Its duration at the time of surgery was 3 to 10 years in 12 patients, 11 to 20 years in five, and more than 20 years in eight. Preoperatively, peripheral neuropathy was suspected to be an additional source of symptoms in three patients who complained of severe symmetrical dysaesthesia in the legs.

**Clinical outcome.** There were 4 (16%) excellent, 14 (56%) good, 5 (20%) fair and 2 (8%) poor results in group A and 5 (20%) excellent, 15 (60%) good, 3 (12%) fair and 2 (8%) poor results in group B. No significant differences were found between the clinical results in the two groups ($p > 0.05$). Considerable improvement was reported by 18 (72%) patients in group A and by 20 (80%) patients in group B.

Neither the duration of diabetes before surgery nor its type correlated with the final outcome (Table III). All three patients in whom diabetic neuropathy had been thought to be a concurrent cause of symptoms, reported

**Table I.** Preoperative clinical findings in the two groups of patients

| Preoperative symptoms and signs | Group A (diabetic) (n = 25) | | Group B (non-diabetic) (n = 25) | |
|---|---|---|---|---|
| | Number | *Percentage* | Number | *Percentage* |
| Time interval (yr)* | | | | |
| < 1 | 15 | *60* | 18 | *72* |
| 1 to 3 | 6 | *24* | 6 | *24* |
| > 3 | 4 | *16* | 1 | *4* |
| Night pain | 5 | *20* | 0 | |
| Abrupt onset | 2 | *8* | 0 | |
| No postural relief† | 4 | *16* | 0 | |
| Claudication distance | | | | |
| 1 block | 7 | *28* | 9 | *36* |
| > 1 to 5 blocks | 14 | *56* | 10 | *40* |
| Positive root tension tests | 5 | *20* | 6 | *24* |
| Motor deficits | 11 | *44* | 10 | *40* |
| Peripheral vascular disease | 5 | *20* | 1 | *4* |
| Comorbidity rate | | | | |
| < 2 points | 5 | *20* | 7 | *28* |
| 3 to 5 points | 6 | *24* | 9 | *36* |
| > 6 points | 14 | *56* | 9 | *36* |

* interval between onset of symptoms and operation
† symptoms not posture-dependent

**Table II.** Preoperative electrophysiological findings in the two groups of patients

| Electrophysiological studies | Group A (diabetic) (n = 20 of 25) | | Group B (non-diabetic) (n = 16 of 25) | |
|---|---|---|---|---|
| | Number | *Percentage* | Number | *Percentage* |
| Nerve conduction | | | | |
| Slow | 16 | *80* | 4 | *25* |
| Normal | 4 | *20* | 12 | *75* |
| EMG | | | | |
| Denervation | 17 | *85* | 14 | *88* |
| Normal | 3 | *15* | 2 | *13* |
| Electrophysiological diagnosis | | | | |
| Radiculopathy | 2 | *10* | 14 | *88* |
| Polyneuropathy | 11 | *55* | 0 | *0* |
| Mixed* | 7 | *35* | 2 | *13* |

* EMG signs of radiculopathy and polyneuropathy

**Table III.** Relationship of the outcome of surgery to the duration of diabetes before operation

| Surgical outcome | 3–10 yr (n = 10) | 11–20 yr (n = 5) | > 20 yr (n = 8) |
|---|---|---|---|
| Excellent | 2 | 0 | 2 |
| Good | 5 | 3 | 6 |
| Fair | 3 | 2 | 0 |
| Poor | 2 | 0 | 0 |

satisfactory results. Two had complete relief of leg symptoms and the third reported relief of leg pain but persistence of paraesthesiae. One patient developed bilateral femoral amyotrophy two months after operation. She experienced the sudden onset of progressive weakness in the thighs with no sensory loss. The strength gradually improved and four years later she was asymptomatic.

High comorbidity correlated with a poor outcome in both groups, but not significantly ($p > 0.05$).

Of the seven patients in group A who had unsatisfactory results, four had improvement soon after surgery but subsequently regressed. Of these four, two had recurrence of preoperative symptoms three and five years after surgery, one developed severe back pain and one experienced complete relief of preoperative symptoms on the left side, but began to complain of pain and weakness in the contralateral limb, although the MRI showed no compression of the nerve structures.

The other three patients with unsatisfactory results reported no improvement after the operation. One of the three had a preoperative history of bilateral calf pain followed by the abrupt onset of bilateral anterior thigh pain. Physical examination revealed slight weakness of both tibialis anterior muscles, absence of tendon reflexes in the lower limbs and bilateral sensory deficit below the knees. CT scans showed stenosis at L3 to L5 levels, while electrophysiological studies demonstrated chronic axonal polyneuropathy. The patient underwent decompression from L3 to S1 but with no relief. The second patient had had a previous decompression for progressive weakness and pain in the lower limbs with partial pain relief but no improvement of the weakness. Five months later he experienced a rapid increase in weakness, pain and numbness in both legs and was found to have paralysis of the evertors and invertors of both feet, and weakness of both triceps surae muscles. The deep tendon reflexes were absent and there was some bilateral sensory deficit. CT and MRI showed severe stenosis at L2 to L3. He underwent a second decompression from L2 to L4 but no improvement occurred. The third patient complained of incapacitating pain in the left foot, made worse by standing and walking, with 'stocking' dysaesthesia, even at rest. She had had a coronary artery bypass and femoropopliteal bypass on the left side, but at the time of admission there was only slight insufficiency of the left tibial artery. Imaging studies showed degenerative spinal stenosis at the L3 to L4 level. Conduction was slowed in the left peroneal motor nerve and absent in the left tibial nerve. The patient underwent decompression and fusion from L2 to L4 but with little improvement.

Four of the five patients in group B who had unsatisfactory results at final follow-up had experienced immediate relief of symptoms after surgery. Three of these had late recurrence of claudication and/or radicular symptoms. The fourth patient developed severe back pain one year after the operation. The only patient in group B who did not improve at all had unrecognised stenosis at another level. He had a second operation three months later but still experienced little relief.

## DISCUSSION

Diabetic neuropathy is one of the conditions commonly considered in the differential diagnosis of lumbar stenosis. There are, however, few studies of the differences between the two conditions and the risk of a wrong diagnosis in patients with lumbar stenosis *and* diabetes is unknown.

Neurogenic claudication, which is present in between 68% (Postacchini 1989) and 94% (Hall et al 1985) of patients with lumbar stenosis, is considered to be the cardinal symptom of that condition. Other symptoms include radicular pain and numbness and weakness in the legs. These may appear only during walking but may also be present at rest, in which case they may be made worse by extension and improve with flexion of the spine. Low back pain is usually slight, the range of spinal motion is normal or only a little decreased and nerve-root tension tests are frequently negative. A motor deficit is present in less than half the patients (Hall et al 1985). Stenosis can usually be demonstrated by myelography, CT or MRI. Asymptomatic lumbar stenosis has, however, been detected by myelography in 12% of patients (Uden et al 1985), by CT in 9% and by MRI in 21% (Wiesel et al 1984; Boden et al 1990).

Diabetic neuropathy includes a wide variety of syndromes. Mononeuropathy affects individual nerves or nerve roots and usually causes pain and motor deficits (Bastron and Thomas 1981; Watkins 1990; Coppack and Watkins 1991) in one or both lower limbs. Its onset is unrelated to the duration of the diabetes and it usually recovers within one year (Watkins 1990; Coppack and Watkins 1991). Polyneuropathy causes a symmetrical, progressive, non-resolving sensory defect which is related to the duration of the diabetes (Watkins 1990). Both types may be present simultaneously (Bastron and Thomas 1981) and they may be associated with a painful syndrome characterised by the abrupt onset of excruciating pain which is not posture-related, night pain, contact discomfort and rapid weight loss (Bastron and Thomas 1981; Coppack and Watkins 1991).

The presence or absence of neurogenic claudication seems to be the best way of discriminating between the two conditions. In diabetic patients, however, there is a

high prevalence of peripheral angiopathy which may cause intermittent claudication (Kreines et al 1982). We found peripheral vascular deficiency in 20% of diabetics, compared with only 4% in the non-diabetic patients. When lumbar stenosis and vascular occlusion coexist, it can be difficult to determine which is the main cause of the patient's complaints (Stanton et al 1987; Dodge, Bohlman and Rhodes 1988) and several studies have reported patients with both conditions who underwent ineffective surgical treatment (Snyder, Mulfinger and Lambert 1975; Stanton, Rosenthal and Lamis 1980; Stanton et al 1987). This probably occurred in one patient in our series who had had multiple vascular occlusions and gained no relief from his preoperative claudication after spinal surgery. In patients with lumbar stenosis and diabetes, confusion may increase when peripheral angiopathy and neuropathy are both present. In these cases intermittent claudication cannot be considered to be a discriminating symptom and mistaken diagnoses can easily be made. In diabetic patients without peripheral vascular disease neurogenic claudication is by far the most diagnostic symptom of spinal stenosis.

The symptoms in the lower limbs were similar in the two groups although only the diabetic patients reported night pain, had no posture-related pain relief and described an abrupt onset of their symptoms. These clinical features are highly suspicious of peripheral neuropathy and should be carefully sought, since they may be the first symptoms of an undiagnosed diabetes (Bastron and Thomas 1981; Coppack and Watkins 1991).

Abnormal nerve conduction is one of the diagnostic criteria of diabetic neuropathy (Dyck et al 1985). Slight slowing in conduction velocity can be caused by the processes of segmental demyelination and remyelination (Chopra, Hurwitz and Montgomery 1969), but marked slowing is due to fibre loss (Dyck et al 1980). Nerve-conduction velocities in patients with simple lumbar stenosis have been poorly investigated. Johnsson, Rosen and Uden (1987) found that only patients with complete myelographic block had a slowing of nerve conduction, but the abnormalities were mild compared with those found in diabetics with severe neuropathy (Malik et al 1989). We found slowing in 16 (80%) of the patients tested in group A and in four (25%) of those tested in group B, two of whom had severe stenosis. We believe that nerve-conduction velocity should be measured in all diabetic patients with spinal stenosis and that diabetic neuropathy should be strongly suspected if there is marked slowing. Mild slowing indicates either diabetic neuropathy or nerve-root compression.

In lumbar stenosis, nerve-root compression has been shown to cause loss of the large myelinated fibres and adhesive changes in the pia-arachnoid (Watanabe and Parke 1986). Microcirculatory disturbances in nerve roots (Blau and Logue 1961; Evans 1964) and reduced CSF percolation (Watanabe and Parke 1986) have both been postulated as causes of neurogenic claudication. In diabetes, histological studies have shown a decrease in the large myelinated fibres in the posterior roots (Olsson et al 1968; Ohnishi et al 1983; Dyck et al 1986) and changes in the dorsal-root ganglia (Greenbaum et al 1964; Olsson et al 1968; Ohnishi et al 1983). Changes in the microcirculation of the peripheral nerves and the nerve roots are also thought to play a major role in the aetiology of nerve damage in diabetics (Dyck et al 1986; Johnson, Doll and Cromey 1986; Malik et al 1989; *Lancet* 1991). These observations suggest that in diabetic patients the nerve roots would be particularly susceptible to further injury, for instance from spinal stenosis, but the results of our study contradict this hypothesis. The presence of diabetes, even for 20 to 30 years before surgery, did not appear to affect the outcome in patients treated by lumbar decompression. A mistaken preoperative diagnosis was the cause of failure of surgical treatment in at least three of the diabetic patients. In two of them diabetic neuropathy was probably the main cause of their symptoms.

The results of our study suggest that in diabetic patients who have spinal stenosis, decompressive surgery is a worthwhile procedure, even in the presence of peripheral neuropathy, and that the clinical history, the results of nerve-conduction studies and CT are the most useful diagnostic tools. It should be noted, however, that it may be extremely difficult to identify the cause of leg symptoms when the two conditions occur together, particularly when different types of diabetic neuropathy and concomitant peripheral angiopathy are present.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

REFERENCES

**Bastron JA, Thomas JE.** Diabetic polyradiculopathy: clinical and electromyographic findings in 105 patients. *Mayo Clin Proc* 1981; 56:725-32.

**Blau JN, Logue V.** Intermittent claudication of the cauda equina. *Lancet* 1961; 2:1081-6.

**Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW.** Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. *J Bone Joint Surg [Am]* 1990; 72-A:403-8.

**Chopra JS, Hurwitz LJ, Montgomery DAD.** The pathogenesis of sural nerve changes in diabetes mellitus. *Brain* 1969; 92:391-418.

**Coppack SW, Watkins PJ.** The natural history of diabetic femoral neuropathy. *QJ Med* 1991; 79:307-13.

**Dodge LD, Bohlman HH, Rhodes RS.** Concurrent lumbar spinal stenosis and peripheral vascular disease: a report of nine patients. *Clin Orthop* 1988; 230:141-8.

**Dyck PJ, Sherman WR, Hallcher LM, et al.** Human diabetic endoneurial sorbitol, fructose and myoinositol related to sural nerve morphometry. *Ann Neurol* 1980; 8:590-6.

**Dyck PJ, Karnes JL, Daube J, O'Brien P, Service FJ.** Clinical and neuropathological criteria for the diagnosis and staging of diabetic polyneuropathy. *Brain* 1985; 108:861-80.

**Dyck PJ, Karnes JL, O'Brien P, et al.** The spatial distribution of fibre loss in diabetic polyneuropathy suggests ischemia. *Ann Neurol* 1986; 19:440-9.

**Evans JG.** Neurogenic intermittent claudication. *Br Med J* 1964; 2:985-7.

**Greenbaum D, Richardson PC, Salmon MV, Urich H.** Pathological observations on six cases of diabetic neuropathy. *Brain* 1964; 87: 201-13.

**Hall S, Bartleson JD, Onofrio BM, et al.** Lumbar spinal stenosis: clinical features, diagnostic procedures and results of surgical treatment in 68 patients. *Ann Intern Med* 1985; 103:271-5.

**Johnson PC, Doll SC, Cromey DW.** Pathogenesis of diabetic neuropathy. *Ann Neurol* 1986; 19:450-7.

**Johnsson KE, Rosen I, Uden A.** Neurophysiologic investigation of patients with spinal stenosis. *Spine* 1987; 12:483-7.

**Katz JN, Lipson SJ, Larson MG, et al.** The outcome of decompressive laminectomy for degenerative lumbar stenosis. *J Bone Joint Surg [Am]* 1991; 73-A:809-16.

**Kreines K, Johnson E, Albrink M, et al.** The course of peripheral vascular disease in non-insulin-dependent diabetes. *Diabetes Care* 1985; 8:235-43.

*Lancet.* Editorial. 'Understanding diabetic neuropathy'. *Lancet* 1991; 338:1496-7.

**Lee JH, McCarty R.** Pain threshold in diabetic rats: effects of good versus poor diabetic control. *Pain* 1992; 50:231-6.

**Linn BS, Linn MW, Gurel L.** Cumulative illness rating scale. *J Am Geriatr Soc* 1968; 16:622-6.

**Malik RA, Newrick PG, Sharma AK, et al.** Microangiopathy in human diabetic neuropathy: relationship between capillary abnormalities and the severity of neuropathy. *Diabetologia* 1989; 32:92-102.

**Ohnishi A, Harada M, Tateishi J, Ogata J, Kawanami S.** Segmental demyelination and remyelination in lumbar spinal roots of patients dying with diabetes mellitus. *Ann Neurol* 1983; 13:541-8.

**Olsson Y, Save-Soderbergh J, Sourander P, Angervall L.** A patho-anatomical study of the central and peripheral nervous system in diabetes of early onset and long duration. *Pathol Eur* 1968; 3:62-79.

**Postacchini F.** Results of treatment. In: *Lumbar spinal stenosis.* Wien, etc: Springer-Verlag, 1989.

**Snyder EN Jr, Mulfinger GL, Lambert RW.** Claudication caused by compression of the cauda equina. *Am J Surg* 1975; 130:172-7.

**Stanton PE Jr, Rosenthal D, Lamis PA.** Differentiation of vascular and neurogenic claudication. *Am Surg* 1980; 46:44-9.

**Stanton PE Jr, Rosenthal D, Clark M, Vo N, Lamis P.** Differentiation of vascular and neurogenic claudication. *Am Surg* 1987; 53:71-6.

**Uden A, Johnsson K-E, Jonsson K, Pettersson H.** Myelography in the elderly and the diagnosis of spinal stenosis. *Spine* 1985; 10:171-4.

**Watanabe R, Parke WW.** Vascular and neural pathology of lumbosacral spinal stenosis. *J Neurosurg* 1986; 64:64-70.

**Watkins PJ.** Natural history of diabetic neuropathies. *Q J Med* 1990; 77:1209-18.

**Wiesel SW, Tsourmas N, Feffer HL, Citrin CM, Patronas N.** Study of computer-assisted tomography. I. The incidence of positive CAT scans in an asymptomatic group of patients. *Spine* 1984; 9:549-51.