The general term “spinal stenosis” can be applied to a variety of pathologic conditions that can constrict the cauda equina or individual nerve roots in the lumbar spine. The focus of this pathway is narrowing and possible nerve entrapment that occurs in the lateral aspects of the spinal canal. “Lateral recess” stenosis was first described by Verbiest in 1940. Others have termed this area variously as the “entrance zone,” the “foraminal zone” or the “subarticular recess.” Neurological symptoms can be caused by entrapment and vascular compromise of the nerve root or the dorsal root ganglion which resides in the foraminal zone. Since the lateral recess is bordered laterally by the pedicle, dorsally by the superior articular facet, and ventrally by the posterior surface of the vertebral body, narrowing of this space can occur with degeneration or hypertrophy of these structures.

Kirkaldy-Willis described lateral stenosis as “part and parcel of the degenerative process.”1

Symptoms of LSS can range from none to severely disabling. Johnson, et.al. followed 32 untreated lumbar stenosis patients over 4 years and found 75% did not change, 15% worsened and 15% improved.2 Treatments vary from “watchful waiting” to aggressive spinal surgery. Rates of surgery in the Medicare population are increasing and there is evidence of 12-fold geographic variation in rates of surgery across the US. While surgery appears to produce better results in the short term, long term results are similar among surgical and non-surgical patients. Conclusions from a recent 8-10 year follow up study “support a shared decision-making approach among physicians and patients when considering treatment options for lumbar spinal stenosis.”3 A Cochrane Collaboration review in 2005 concluded that “Surgical investigations and interventions account for large health care utilization and costs, but the scientific evidence for most procedures is still limited.”4

Subjective Findings and History

- Symptoms of pseudoclaudication (radiation of nerve pain down legs mimicking sensation of true claudication from peripheral vascular disease) are associated primarily with central lumbar stenosis.
Pain improved with forward bending or sitting
Pain worse with extended walking

- In contrast, patients with purely lateral recess stenosis:
  - Usually do not develop symptoms of neurogenic claudication
  - Typically have radicular symptoms in a specific dermatomal pattern
  - Often have pain at rest, at night, and with the Valsalva maneuver
  - Tend to be younger (mean age 41 years) than patients with central canal stenosis (mean age 65 years)
  - Variable degree of mechanical low back pain is very common

**Objective Findings**
There are no pathognomonic signs in lateral recess stenosis.
  - Limited lumbar range of motion, especially extension;
  - Focal motor weakness and diminished subjective sensation in a specific root distribution;
  - Variable straight-leg tension signs;
  - Diminished reflexes in specific root distributions

**Imaging**
- Plain radiographs including dynamic flexion and extension studies, allow the intervertebral foramen to be depicted, but findings must be correlated with the history and examination findings because false positives at x-ray are common.
- CT imaging allows detection of bony encroachment on the lateral recess.
- Electromyogram (EMG) or nerve conduction studies, can identify damage to or irritation of the nerves and help determine location of nerve involvement.
- The findings most suggestive of stenosis on MRI include a foramen of diminished size and a paucity of perineural fat surrounding the nerve root on T1-weighted images
- Very little evidence exists correlating degree of narrowing of the lateral recess with the presence or severity of the signs, symptoms, or conditions associated with stenosis.

**Assessment**
- The differential diagnosis must include radiculopathy from a herniated disc, significant lateral recess stenosis midzone stenosis associated with a hypertrophic pars interarticularis defect, or extraforaminal causes, mechanical and neurological causes of low back and lower extremity pain, peripheral neuropathies, osteoarthritis of the spine or hip and trochanteric bursitis.
- Ruling out the “red flags” of cauda equine syndrome is crucial.
- Assess for intersegmental and sacro-iliac (SIJ) dysfunction. Symptoms of LSS overlap with a number of other conditions and the differential for LSS includes vascular claudication.
- Assess relative and absolute contraindications to spinal manipulation (e.g. advanced DJD/DDD).

**Plan**
*Passive care:*
  - Distraction mobilization
  - Neural mobilization
Active care:
- Exercises “cat and camel,” “nerve flossing.”
- Spinal stabilization and/or mobilization exercises, as necessary
- Medications including gabapentin (Neurontin) and pregabalin (Lyrica), Cortisone (steroid) injections in the lumbar spine (epidural), to decrease inflammation and swelling.

Length of treatment
Duration and frequency of treatment depend on patient response. Murphy suggests ~13 treatments as an adequate trial.

Referral criteria
- Failure to respond
- Worsening neurological signs and symptoms, including loss of bowel and bladder control and loss of sexual function.
- Cauda equine syndrome
- Surgical consult

Resources for Patients
Medline Plus

ACA patient page. JACA Online April 2008.

References

The Evidence


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