In their article, presented in this issue of *Pain Practice*, Bosscher and Heavner touch upon an important issue in pain therapy that is to say, ascertain the correct pain generator for the patient’s low back and or leg pain since to date no gold standard is available for this purpose. To identify the correct spinal level from where low back or radicular pain originates, the authors prospectively compared the findings from epiduroscopy with clinical examination and MRI results in 143 patients. In 40 patients (28%), the spinal level identified by epiduroscopy correlated with clinical examination; whereas compared to MRI, this was only the case in 28 patients (20%). Absence of pure dermatomal distribution and heterogeneity of pathology reported on MRI are heralded as possible causes of this discrepancy, while the functional nature of epiduroscopy and reproducibility of pain provocation during the procedure are put forward by the authors as arguments for using epiduroscopy as a guide for targeted treatment.

A prerequisite for successful treatment of any condition is to establish the correct diagnosis since even the best therapies will fail if used for the wrong indication. Historically, clinical examination, imaging techniques, and diagnostic blocks provided the backbone in clinical decision making for pain therapists. Now the question is whether epiduroscopy can be an added value to this inventory.

A clear answer to this question, however, is first of all troubled by the terminology used in the literature to determine the vertebral or spinal level of the pathology. Second, the lumbosacral radicular syndrome should be clearly distinguished from nonradiating low back pain.

Furthermore, clinical examination for specific causes of low back and/or leg pain is often equivocal. Although indicative, the distribution of pain along a dermatome in radicular pain shows a large variation of spinal levels involved. The added diagnostic value of provocative manoeuvres such as the straight leg raise or crossed straight leg raise comes at the expense of specificity and sensitivity, respectively. The same goes
for clinical examination in specific causes of low back pain. Revel’s criteria for diagnosing facetogenic low back pain could not be reproduced in a subsequent study, whereas for discogenic pain no clinical signs have been put forward. Sacroiliac joint pain is the only condition where 3 or more positive provocative tests bestow a good sensitivity (78%) and specificity (94%).

Over the past decades, new imaging techniques offered higher spatial and contrast resolution in the spine thus providing the ability to distinguish anatomically normal from abnormal or degenerated structures. Although the value of CT scan or magnetic resonance imaging is indisputable in the assessment of red flags related to back pain, imaging techniques failed to reliably confirm abnormal or degenerated facet or sacroiliac joints as a cause of low back pain in response to diagnostic blocks. Another good example is the presence of postoperative epidural fibrosis: roughly as many radiological studies confirm it to be the cause of failed back surgery syndrome as there are studies that refute it. Moreover, in another study, Bosscher and Heavner demonstrated that the diagnosis of epidural fibrosis on MRI was correct only 16.1% when compared to epiduroscopy.

In view of these data, there is a strong need for improved diagnostics in low back and leg pain. Epiduroscopy offers the advantage of visually identifying structures in the epidural space. Moreover, the patient can report if the pain elicited by probing epidural structures is concordant with his daily pain sensation. This functional nature of a test to diagnose painful nerve roots seems rational, looking at the experimental evidence for the distribution of radicular pain signals. Reproducing the patient’s paresthesia with a radiofrequency generator was the most reliable in localizing the affected dermatome.

However, a few words of caution are in order. Although identifying the spinal level responsible for reproducing the patient’s pain during epiduroscopy, Bosscher, and Heavner did not mention which epidural structures were responsible for pain generation (eg, facet joints, discs, nerve roots, etc.). It would have been helpful if the authors also stated the spinal structure they probed to reproduce the patient’s pain and compared this with the probable diagnosis according to clinical examination and MRI findings. Diagnostic blocks, although hampered by false positive and negative results, still represent the benchmark for diagnosing most causes of low back and leg pain and the findings during epiduroscopy need to be confirmed against it. Finally, there is a certain amount of overlap between true radicular pain and pseudoradicular pain caused by facetogenic, discogenic, or sacroiliac joint pain irradiating into the leg. Therefore, pain from probing these structures might be mistaken for radicular pain.

In conclusion, every technique used to establish a diagnosis in pain therapy has its disadvantages or shortcomings. Therefore, the pain physician should not rely on one particular feature to ascertain the cause of pain but rather look at the bigger picture and determine whether there is conformity between clinical presentation, imaging results, diagnostic blocks and eventually epiduroscopy to ascertain the cause of the patient’s pain.

REFERENCES


