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ings?; 3) can selective white matter tract involvement be seen on MR images early in the patient's myelopathic state?; and 4) can magnetization transfer ratios (MTRs) be applied to normal-appearing cord tissue to show decreased MTRs in affected spinal cords? Although these and other issues are left unresolved by Chong's investigation, the article can serve as a springboard for important studies. Future studies could investigate the effect of newer treatment protocols for the imaging of patients with AIDS-associated myelopathy and the precise distribution of signal abnormalities in the cord in both the early and late stages of this complex disease.

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Three Pathways between the Sacroiliac Joint and Neural Structures Exist

At the beginning of this century, pain from the sacroiliac joints had been considered the main source of low back pain and radiculopathy. Since the discovery and acceptance of the lumbar-disk-complex model of radicular back pain, the theory that the sacroiliac joint contributes to a low back pain syndrome remains controversial and poorly understood as part of a broad category of nondiscogenic low back pain.

Fortin et al present in this issue of the *AJNR* (page 1429) an intriquing hypothesis asserting that pathways of communication exist between the sacroiliac joints and several neural structures. Tracing extravasation patterns on sacroiliac arthrograms and postarthrogram CT, the authors have delineated pathways in which contrast material from the sacroiliac joint communicates posteriorly with the first dorsal foramima, ventrally with the lumbosacral plexus, and dorsally along the sacral ala to the fifth lumbar epiradicular sheath. Drawing from the discogenic model of low back pain, the authors suggest that sacroiliac capsular irritation and cytokine release may cause adjacent neural insult by these communications. Furthermore, the variety of structures these pathways lead to may in turn explain the variety of symptoms and signs possible from sacroiliac disease.

Sacroiliac arthrography is an uncommon procedure in most radiology departments that often falls between specialty lines of neuroradiology, musculoskeletal radiology, and body imaging, because patients with sacroiliac pain come from a variety of orthopedic, neursurgical, neurologic, and rehabilitation specialty referrals. Most of these procedures include injection of anesthetic or corticosteroids, with any reduction of a patient's symptoms indicating the sacroiliac joint as the source of pain. Extravasation is very common in these procedures, and the patterns of extravasation described by the authors frequently are observed in clinical practice. Furthermore, although the validity of pain reproduction and reduction with anesthetic in the setting of extravasation may be challenged, it nonetheless occurs. The notion that these communications by arthrography provide the mechanism for pain arising from sacroiliac disease is unproved but still attractive.

Among the most frustrating conditions in medicine is atypical or nonradicular low back pain. Unlike the patient with persistent low back pain and a radiculopathy matching a structural lesion seen at imaging, in which one may be relatively confident of a relationship between that finding and symptoms, patients with atypical lumbosacral junction pain frustrate clinicians and radiologists. Without radicular symptoms, or with a radiculopathy that does not match a structural lesion, a scenario occurs in which management often is directed by the results of provocative injections of disks, facets, and sacroiliac joints. To make matters worse, asymptomatic imaging abnormalities are common at the lumbosacral junction, including disk herniations causing nerve compression that may misdirect treatment. Sifting through the significant and insignificant imaging findings of the spine and sacroiliac joints in light of a complicated or inconsistent clinical history of low back pain is a very difficult challenge. In this setting, Fortin's observations may provide a starting point to reexamine the nature of atypical radicular pain, particularly with close correlation of injection data and specific pain patterns. Previous work by the author correlating pain maps from sacroiliac injection in volunteers and patient-drawn pain maps in individuals with atypical lumbosacral pain warrant a close read by anyone imaging or treating patients in whom lumbosacral and sacroiliac pain must be differentiated.

Despite this, caution must be used before one should accept the authors' hypothesis. The precise mechanism of pain from any joint may involve not only capsular irritation, but also direct subchondral bone irritation through cartilage loss and marrow edema. In patients with seronegative spondyloarthropathies involving the sacroiliac joints, as well as in individuals with post-traumatic and degenerative sacroiliac pain, the subchondral plate frequently is compromised, and this mechanism cannot be disregarded. Indeed, one could argue that stimulation of subchondral, pressure-sensitive pain receptors in bone could account for much of the local pain observed in sacroiliac disease, with communication to adjacent neural structures accounting only for the radicular component in those individuals with mixed local and radicular pain. This would not account, however, for the authors' prior observation of pain in an identical distribution in

asymptomatic volunteers undergoing sacroiliac arthrography, assuming the volunteers had intact sacroiliac joint surfaces.

Even with the exact mechanism of pain remaining unproved, the article reinforces the role of provocative tests in the diagnosis of atypical low back pain, and provides an avenue for further investigation toward rational diagnosis and treatment of this vexing clinical problem.

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Balloon Dilatation of Middle Carotid Artery Occlusion Combined with Balloon Angioplasty and Stent Therapy of Internal Carotid Artery Stenosis

Acute ischemic stroke therapy continues to present unique therapeutic challenges. The treatment of acute intracranial thromboembolism and internal carotid artery (ICA) stenosis have been undergoing parallel advances and have been combined as a series in only a handful of individual reported cases. Mori et al, in this issue of the *AJNR* (page 1462), focus on two current stroke treatment techniques performed in a single patient: the combined therapy of balloon dilatation of middle carotid artery (MCA) occlusion and balloon angioplasty and stent therapy of ICA stenosis.

The recently reported Prolyse in Acute Thromboembolism Trial (PROACT) II study has confirmed the effectiveness and safety of medical recanalization of M1 and M2 occlusions achieved by using intra-arterial recombinant prourokinase within 6 hours of onset (1). A 15% absolute benefit and 60% relative benefit was achieved, with the end point being a modified Rankin outcome score of 0–2 (minimal nondisabling deficit). That study seems to confirm other anecdotal opinions that intra-arterial thrombolytic therapy offers some benefit for stroke patients beyond the 3-hour window accepted for intravenous tissue plasminogen activation (tPA) therapy.

Mechanical revascularization achieved by angioplasty of acute thrombotic occlusive lesions is standard in the coronary system, but its use in the MCA has been delayed by the concern that squeezing thrombus into lenticulostriate perforators (if not already there) is probably bad. Moreover, fragmentation of thrombus into distal vessels, perhaps decreasing collateral flow, may be worse than a stable proximal thrombus that already has done its major damage. In addition, it is not surprising that enthusiasm for angioplasty originated in Japan, where underlying atherosclerotic stenoses are known to be most prevalent. Nakano and colleagues' series of 10 patients with MCA occlusions, thought to be poor candidates for thrombolysis and at risk for hemorrhagic complications or reocclusion, supports the belief that balloon-assisted recanalization is a viable option (2). Fifty percent excellent to good, 50% fair, and no poor outcomes or deaths validates this concept.

Ueda et al reported that, with MCA occlusion resistant to thrombolysis, outcomes for angioplasty-treated patients were better than for patients treated by thrombolysis alone (3). Although this is a selected group of patients, some treated immediately and others treated some days later, this investigation gives additional insight into safety and patient selection.

Mori et al's patient had an initial National Institutes of Health Stroke Scale score of 18, possibly suggesting, according to PROACT II control-group data, a 25% chance of good outcome if nothing were done. On the other hand, the patient did not suffer the fatal outcome of 25% of MCA occlusions either. Therefore, we may assume a benefit was accrued by the balloon-assisted angioplasty, although the anecdotal nature of the report must not be overlooked. Time from ictus and arteriographic collateral flow characteristics are not described, so these additional concerns cannot be factored into the discussion of outcome. ICA angioplasty and stenting subsequently led to an excellent appearance of the ICA, but the exact degree of ''high-grade stenosis'' initially present is still not clear from the report.

The excellent outcome in an individual case must be viewed with the reservation that the same treatment paradigm might not be applicable to patients with similar angioarchitectural appearances. Heterogeneous patient-specific factors primarily will influence the applicability of any therapy. Intracerebral hemorrhage is certainly the most feared sequelae of revascularization, typically expected in 5% to 10% of thrombolysis patients. It is possible that MCA angioplasty at 3 hours post ictus may be safer than thrombolysis conducted from 3 to 5 hours, because time to recanalization, and hence ischemia time, is reduced. On the other hand, high-pressure recanalization of an ischemic (and maximally dilated) vascular bed 6 hours post ictus may be more harmful than an unsuccessful thrombolysis.

If an intracranial angioplasty result is not as excellent as those obtained by Mori et al, and thrombolysis of distal clot is required, it is not clear that additional thrombolytic therapy will be as beneficial. Perhaps if the patient is within a reasonable window for intravenous tPA therapy, we may find that IV tPA, given as rapidly as possible to delay the time to thrombolysis and followed by angiography and angioplasty or intra-arterial thrombolysis for residual occlusions, may also be safe and perhaps more efficacious.