To the Editor:

In the article on piriformis syndrome by Foster, which appeared in the August 2002 issue of *Orthopedics* (25[8]:821-825), a distinction of piriformis syndrome in primary and secondary is presented and analyzed.

According to the author, intrinsic piriformis muscle problems constitute primary piriformis syndrome, such as the injury after a fall as described by Robinson,\(^1\) pyomyositis,\(^2\) or pyositis ossificans\(^3\) of the muscle. Furthermore, Foster defines secondary piriformis syndrome “as piriformis irritation secondary to the sacroiliac joint” as described by Yeoman\(^4\) or Pace and Nagle,\(^5\) or by a mass effect, as described in numerous case reports.\(^6\)

We believe, however, that the reference by Yeoman\(^4\) should cease or be reserved as a historic reference to sciatica, as he considered sciatica to be the result of a periarthritis involving the anterior sacroiliac ligament, piriformis muscle, and adjacent branches of the sciatic nerve.

In the article by Pace and Nagle,\(^5\) the pathophysiologic mechanism proposed is a focal irritability of the piriformis muscle, usually caused by trauma, resulting in a so-called trigger-point syndrome. Thus, these cases should be classified as primary piriformis syndrome, as the piriformis muscle is the structure of the symptoms’ origin.

In addition, we do not recognize any relation between sacroiliac joint syndrome and piriformis syndrome. Bernard and Kirkaldy-Willis\(^7\) reported an incidence of 20.55% and 0.33%, respectively. Stressing the need to recognize coexisting lesions, they found only 13 cases of muscle syndrome (not specific piriformis syndrome) associated with 336 cases of sacroiliac joint syndrome. Finally, piriformis syndrome treated effectively by injecting the sacroiliac joint has not been reported.

Aside from Foster’s classification, which is based on the presence (primary) or absence (secondary) of intrinsic piriformis muscle lesion, Chen\(^2\) classified piriformis syndrome based on the pain mechanism. He states two mechanisms explain buttock pain and sciatica: 1) myofascial pain syndrome as described by Pace and Nagle,\(^5\) and 2) sciatic nerve entrapment as described by Robinson.\(^1\) In buttock pain, thus piriformis muscle trauma (primary piriformis syndrome), two factors determine the energy absorbed: the magnitude of injury and the habitus of the patient. Low-energy absorption may cause a myofascial piriformis syndrome, in contrast to a high-energy absorption that causes hematoma formation and scarring and thus nerve entrapment. On the other hand, unresolved chronic irritation, such as a myofascial injury, may eventually lead to adhesions and sciatic nerve entrapment.

The sciatic nerve may be impinged or entrapped by an anatomical variant of the piriformis muscle as postulated by Pecina\(^8\) and in several case reports.\(^2,9\) These anatomic variations may increase the sciatic nerve susceptibility to injury, thus we believe these cases should be classified as secondary piriformis syndrome because intrinsic piriformis muscle pathology is not present. Some authors consider these variants an incidental finding\(^11,12\) in patients with primary piriformis syndrome.

McCrorry and Bell\(^13\) proposed the term “deep gluteal syndrome,” as they found it difficult to attribute all pain elements exclusively to the sciatic nerve and not to the posterior cutaneous nerve of the thigh or the gluteal nerves. They also considered that any hip rotator could compress the neural structures.\(^13\)

Extending their suggestion, we believe “pelvic outlet syndrome” includes all cases of extraspinal nerve compression and lower extremity pain, augmenting or replacing the term “secondary piriformis syndrome.” Compressive structures such as aneurysms or arterial malformations,\(^14\) benign or malignant tumors,\(^19,20\) endometriosis,\(^21,22\) inflamed structures,\(^23\) sciatric hernias,\(^26\) adhesions after total hip replacement,\(^27\) or malunited fractures\(^28,29\) may be sited anywhere before, at, or after the sciatic notch. The specific signs of piriformis muscle irritability, such as pain in flexion, adduction, and internal rotation or positive Pace test may be present. This classification would increase physician’s vigilance to obtain a detailed history and perform a complete physical examination, to use the available imaging and electrodiagnostic studies, and avoid unnecessary spinal operations in cases of obscured lower leg pain syndromes.

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REFERENCES

*Piriformis Syndrome* (continued on page 799)
(Letters to the Editor continued)


Reply:

The Yeoman reference is indeed historic, as it predated Mixter and Barr’s recognition of disk herniation; however, I consider sciatica as radiating pain below the knee, and I agree and re-emphasize the need to remain aware that sciatica is not solely a result of intraspinal pathology. Regardless of the role of the sacroiliac joint, overstated by Yeoman and in my opinion not much better understood today, most hip replacement surgeons encounter patients who not only have deteriorated disks but also have, as he describes, a “periarthritiss,” so leg pain is not an uncommon clinical diagnostic dilemma.

Regardless of the interpretation of Pace and Nagle’s article, they report a 6:1 female predisposition, with the most common complaint being dyspareunia. The focal irritability of the piriformis muscle in the patients of Pace and Nagle would be at the origin of the piriformis, which overlies the sacroiliac joint, intrapelvic, and is consistent with the female predisposition and dyspareunia, for which intravaginal injections have been done. Those injections also are in the vicinity of the sacroiliac joint. I do not consider an intrinsic problem of the piriformis muscle as a plausible cause of dyspareunia; in fact, I consider birthing children and subsequent sacroiliitis the only credible explanation for the female predisposition in Pace’s family practice population. In this I depart from Papadopoulos et al, as I consider sacroiliitis significantly correlated with piriformis syndrome.

Further, I would suggest that trigger points are not exclusively a problem intrinsic to muscle, and in my experience, many are referred or secondary; many lumbar “referred pain” syndromes or the muscles are normal.

Perhaps a pelvic outlet syndrome would be appropriate for a compression neuropathy, essentially “peripheral” in terms of being distal to the spinal canal, but the symptoms are from the sciatic nerve, which the piriformis overlies, and in my experience, has been hour-glassed in every case I have explored. The other external rotators (oburator internus and superior and inferior gemelli) are anterior to the sciatica nerve and the piriformis muscle, hence, when they are detached from the femur, retracted posteriorly for a customary total hip approach, they remove the sciatic nerve from the field and harms way. The piriformis has a specific role in this form of sciatic nerve irritation or compression by virtue of its anatomical position.

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