MR Imaging of Entrapment Neuropathies of the Lower Extremity

Part 1. The Pelvis and Hip

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Entrapment neuropathies can manifest with confusing clinical features and therefore are often underrecognized and underdiagnosed at clinical examination. Historically, electrophysiologic evaluation has been considered the mainstay of diagnosis. Today, cross-sectional imaging, particularly magnetic resonance (MR) imaging and specifically MR neurography, plays an increasingly important role in the work-up of entrapment neuropathies. MR imaging is a noninvasive operator-independent technique that allows identification of the underlying cause of injury, differentiation between surgically treatable and untreatable causes, and guidance of selective diagnostic anesthetic nerve blocks. Pathologic conditions affecting the lumbosacral plexus and major motor and mixed nerves of the pelvis and hip include neuropathies of the lumbosacral plexus, femoral nerve, lateral femoral cutaneous nerve, obturator nerve, and sciatic nerve; piriformis muscle syndrome; and injury of the gluteal nerves. Diagnosis of entrapment neuropathies of the pelvis and hip with MR imaging requires familiarity with the normal MR imaging anatomy and awareness of the anatomic and pathologic factors that put peripheral nerves at risk for injury.

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LEARNING OBJECTIVES

After reading this article and taking the test, the reader will be able to:

- Describe the normal anatomy of the lumbar and sacral plexuses.
- List the pathologic conditions of the lumbar and sacral plexuses and the peripheral nerves of the pelvis and hip.
- Identify entrapment neuropathies in these regions on the basis of their MR imaging features.

TEACHING POINTS

See last page

Abbreviations: ASIS = anterior superior iliac spine, LFCN = lateral femoral cutaneous nerve
Introduction

Entrapment neuropathy is an underrecognized cause of pain and functional impairment caused by acute or chronic injury to peripheral nerves. Intuitively, neuropathy should manifest clinically with a predictable symptom profile specific to the affected nerve. More often, however, vague and poorly localized symptoms can produce a complex clinical picture that can be difficult to distinguish from more common, nonneurologic causes of pain. Moreover, other neurogenic causes such as disk disease can mimic peripheral entrapment neuropathies as well as coexist with them.

Although nerves may be injured anywhere along their course, they are more prone to compression, entrapment, or stretching as they traverse anatomically vulnerable regions, such as superficial or geographically constrained spaces.

Magnetic resonance (MR) imaging is a noninvasive, operator-independent modality with several advantages over electrophysiologic studies. Although both have the capacity to demonstrate the presence and location of neural injury, MR imaging can also demonstrate the underlying cause of injury and therefore allows discrimination between surgically treatable and untreatable causes. Imaging is also becoming increasingly crucial in guiding selective, often diagnostic, anesthetic nerve blocks. Early diagnosis is essential; the degree and duration of injury affect the extent of neural recovery.

This article focuses on entrapment neuropathies in the pelvis and hip and highlights the “vulnerable zones” where nerves are at greatest risk for injury. Normal MR imaging anatomy and MR imaging features of pathologic conditions affecting the lumbosacral plexus and major motor and mixed nerves of the pelvis and hip are presented. Specific topics discussed are direct and indirect MR imaging signs of nerve injury; neuropathies of the lumbosacral plexus, femoral nerve, lateral femoral cutaneous nerve (LFCN), obturator nerve, and sciatic nerve; piriformis muscle syndrome; and injury of the gluteal nerves.

MR Imaging: General Concepts

Peripheral nerves appear as singular or bundled longitudinally oriented structures, with intermediate signal intensity on T1-weighted images and images obtained with fluid-sensitive sequences. Large nerves are easiest to identify because of abundant perifascicular fat and because of greater fascicular composition (40–60 fascicles in the case of the sciatic nerve, one or two fascicles in the proper digital nerve) (Fig 1) (1). Perifascicular and perineural high signal intensity from fat makes nerves conspicuous on T1-weighted images and provides a model road map. The ideal imaging planes for nerves will depend on their course; however, axial images tend to be the most useful. Oblique imaging along or perpendicular to the course of a nerve can optimize nerve detection.

At MR imaging, neural disease is inferred from alterations in nerve signal intensity, size, morphology, and location. Nerve disease is presumed when there are secondary signs of neural injury (eg, denervation of the supplied muscles in the case of motor nerves). In general, sensory manifestations predominate in mixed motor-sensory nerves; in this case, no indirect imaging findings will be present.

Direct MR Imaging Signs of Nerve Injury

Signal Intensity

Normal nerves have intermediate signal intensity on T1-weighted images and are isointense or mildly hyperintense to muscle on T2-weighted and other fluid-sensitive images. Normal endoneurial fluid, when protected by an intact perineural blood-nerve barrier, is thought to be primarily responsible for the normal signal intensity pattern of nerves at T2-weighted neurography. Increased perifascicular and endoneurial signal intensity on T2-weighted images reflects a nonspecific response of the nerve to injury; in essence, T2-weighted neurography allows evaluation of changes in perifascicular and endoneurial signal intensity (2).

T2-weighted neurography is accomplished by using sequences with long echo times (at least 90 msec), radiofrequency saturation pulses to suppress signal from adjacent vessels, and frequency-selective fat suppression (3,4). To be effective, the latter requires a homogeneous magnetic field; this is optimized by shimming, use of a small field of view, and isocenter positioning of the region of interest. Because the chemical shift between water and fat increases with increasing field strength, T2-weighted neurography is best performed with higher field strength MR imaging systems (5). The resultant heavily T2-weighted fat-suppressed images increase the conspicuity of T2 signal changes in the nerve.
Figure 1. Normal anatomy of the sciatic nerve. (a) Axial T1-weighted MR image (repetition time msec/echo time msec = 553/12) shows the fascicular composition of the sciatic nerve (arrow). (b) Coronal T1-weighted MR image (713/25) shows the abundant perifascicular fat of the sciatic nerve (arrows).

Figure 2. Femoral neuropathy. Axial image from T2-weighted neurography (4420/145), obtained at the S1 level, shows increased size and increased signal intensity of the left femoral nerve (solid arrow). The right femoral nerve is normal (open arrow).

However, increased signal intensity in the nerve does not always indicate underlying disease. The magic angle effect is a well-recognized artifact in brachial plexus and lumbosacral plexus imaging. Chappell et al (6) were the first to describe this phenomenon in peripheral nerve imaging, to our knowledge. Unlike in tendons, however, where the magic angle artifact can be overcome with longer echo times (>40 msec), spurious high nerve signal intensity related to the magic angle can persist at higher echo times (66 msec) as well as on short inversion time inversion-recovery images. These angle-specific signal changes must be kept in mind when interpreting MR images, particularly when increased nerve signal intensity is the sole abnormality.

Because normal nerves are slightly hyperintense on fluid-sensitive images, high signal intensity that is focal or similar to that of adjacent vessels is more likely to be significant.

As in the central nervous system, the presence of a blood-nerve barrier allows neural enhancement with gadolinium contrast material only when this protective barrier is compromised. However, it is not yet agreed whether intravenous gadolinium contrast material is necessary for assessment of nerve disease. Both the sensitivity and time course of neural enhancement with gadolinium in injured nerves have yet to be defined.

The use of diffusion-weighted imaging in the evaluation of nerves has only recently been reported, largely for the evaluation of the optic nerve and other cranial nerves, brachial plexus, and lumbosacral plexus (7,8). The small size of many peripheral nerves limits its current use.

Size and Morphology
Neural enlargement, loss of the normal fascicular appearance, or blurring of the perifascicular fat are morphologic changes that suggest neural injury (Fig 2). These findings are more easily appreciated in the larger nerves of the lower extremity, such as the sciatic nerve or common peroneal nerve. Size differentials and fascicular appearance are more difficult to discern in smaller nerves such as the inferior and superior gluteal nerves.
Indirect MR Imaging Signs of Nerve Injury

Motor neuropathy may be inferred from a characteristic pattern of skeletal muscle denervation as manifested by changes in muscle signal intensity. Within days or weeks, reversible acute muscle denervation is seen as homogeneous and diffuse muscle signal hyperintensity on fluid-sensitive images; this appearance is due to fluid shift into the extracellular compartment and increased blood volume secondary to an enlarged capillary bed. In this early stage, the T1 signal of muscle remains normal. There are no abnormalities of the subjacent fascia or subcutaneous tissues; the normal architecture and size of the muscle are preserved, and, except in aberrant denervation patterns, muscle involvement follows a specific nerve distribution. These features help distinguish denervation from other causes of increased muscle signal intensity, such as muscle strain, tear, infection, or infarct.

As denervation progresses, muscle signal intensity is increased on both T1-weighted and fluid-sensitive images owing to ongoing, still reversible, denervation. The increased T1 signal indicates denervation-related muscle atrophy. With prolonged denervation, the bulk of the muscle will decrease and be completely replaced by fat. This process manifests as nonspecific increased muscle signal intensity on both T1-weighted and fluid-sensitive images and indicates irreversible end-stage disease and muscle atrophy.

Often, affected muscles are distal to the site of nerve injury and will not be included in routine field-of-view imaging; in these cases, imaging distal to the site of neural injury may be necessary. Extended field-of-view imaging with a whole-body MR imaging system can also be useful in these situations. Although denervation often results in stereotypic muscle involvement (Table), aberrant or cross-innervation can produce atypical denervation patterns.

**Innervation of the Pelvis and Thigh Muscles**

<table>
<thead>
<tr>
<th>Supplying Nerve</th>
<th>Muscles Innervated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sacral plexus</td>
<td>Piriformis, inferior and superior gemellus, obturator internus, quadratus femoris</td>
</tr>
<tr>
<td>Femoral nerve</td>
<td>Iliopsoas, pectineus, quadriceps, sartorius</td>
</tr>
<tr>
<td>Obturator nerve</td>
<td>Adductor brevis, adductor longus, anterior head of adductor magnus (plus sciatic supply), obturator externus, gracilis</td>
</tr>
<tr>
<td>Sciatic nerve</td>
<td></td>
</tr>
<tr>
<td>Tibial division</td>
<td>Biceps femoris (long head), semitendinosus, semimembranosus, adductor magnus</td>
</tr>
<tr>
<td>Peroneal division</td>
<td>Biceps femoris (short head)</td>
</tr>
<tr>
<td>Superior gluteal nerve</td>
<td>Gluteus medius, gluteus minimus, tensor fascia lata</td>
</tr>
<tr>
<td>Inferior gluteal nerve</td>
<td>Gluteus maximus (sole supply)</td>
</tr>
</tbody>
</table>

*Rectus femoris and vastus lateralis, medialis, and intermedius.

**Figure 3.** Major nerves of the lumbar and sacral plexuses. The femoral nerve (open arrow) is formed from the L2–L4 roots and exits the pelvis under the inguinal ligament. The obturator nerve (open arrowhead), which is also formed from the L2–L4 roots, exits the pelvis under the superior pubic ramus in the upper outer corner of the obturator foramen. The LFCN (solid arrowhead), which is often formed from the L2 and L3 roots, exits in the region of the anterior superior iliac spine (ASIS) under, over, or through the inguinal ligament. The sciatic nerve (solid arrow), which is formed from the L4–S3 roots, receives contributions from both the lumbar and sacral plexuses.
Introduction to Normal Anatomy
The ventral rami of L1–L4 and the ventral rami of L4–S3 coalesce to form the lumbar and sacral plexuses, respectively. Together, via the lumbosacral trunk, they form the lumbosacral plexus, which innervates the lower extremity.

The lumbar plexus is formed within or posterior to the psoas muscle. It gives rise to nerves that exit either lateral (iliohypogastric, ilioinguinal, genitofemoral, femoral, and LFCN) or medial (obturator nerve and lumbosacral trunk) to the psoas muscle border.

The sacral plexus is formed anterior to the piriformis muscle and gives rise to the sciatic nerve, the superior and inferior gluteal nerves, and the pudendal and posterior femoral cutaneous nerves. The lumbosacral trunk, which is made up of L5 and a small branch of L4, effectively connects the lumbar and sacral plexuses; it is for this reason that the term lumbosacral plexus is often used to refer to the combined neural complex (Fig 3).

Specific Neuropathies
Lumbosacral Plexus

Normal Anatomy and Imaging.—The lumbar plexus (Fig 3) is formed within (or less commonly posterior to) the psoas muscle from the ventral rami of L1–L4 at approximately the level of the L2–L5 transverse processes (9). Therefore, imaging for lumbosacral plexus disease should begin at least as high as L1.

Although the lumbar roots can be followed as they exit their neural foramina, the lumbar plexus is difficult to visualize at routine imaging. Once the ventral rami of L1–L4 enter the psoas muscle, the roots and resultant plexus are difficult to identify on MR images, although they are occasionally seen as fascicular structures surrounded by fat within or posterior to the muscle. The terminal branches of the lumbar plexus are first detected once they exit the psoas muscle.

The iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, and femoral nerves exit along the lateral border of the muscle, whereas the obturator nerve and lumbosacral trunk exit along the medial border.

The sacral plexus (Figs 3, 4) is formed along the anterior surface of the piriformis muscle from the lumbosacral trunk and the first through third sacral roots. Small field-of-view images aligned along and perpendicular to the plane of the sacrum significantly improve visualization of the sacral plexus.

The lumbosacral trunk can be followed in the axial (and sometimes the coronal) plane as it descends to join the first, second, and third sacral nerve roots along the anterior surface of the piriformis muscle. Smaller field-of-view coronal oblique images oriented along the plane of the sacrum, axial oblique images perpendicular to the sacral plane, and sagittal images centered around the sacrum allow the sacral nerve roots to be seen as they exit the sacral foramina and coalesce along the anterior surface of the piriformis muscle before separating into the sciatic nerve, superior and inferior gluteal nerves, and pudendal nerve.

Pathologic Conditions.—The lumbosacral plexus may be directly affected by extrinsic compression and diffuse infiltration or may be secondarily involved in the setting of systemic or inflammatory processes. Retroperitoneal processes are more likely to affect the lumbar plexus, whereas pelvic disease is more likely to affect the sacral plexus.

Psoas muscle disease related to surgery or trauma, anticoagulant therapy–related hematoma, abscess, and tumor infiltration (neoplastic plexopathy) can directly compress the lumbar plexus. Painful neoplastic plexopathy due to tumor infiltration of the psoas muscle, known as...
**Figure 5.** Radicular symptoms after a right-sided sacral fracture. Axial fat-suppressed image from T2-weighted neurography (4600/110) shows increased size and signal intensity of a right sacral root (white arrow). Linear increased sacral signal intensity (black arrow) is compatible with a fracture.

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**Figures 6, 7.** (6) Left L4 neuropathy. Axial fat-suppressed T2-weighted MR image (4000/75) (a) and coronal image from T2-weighted neurography (4000/110) (b) show increased size and signal intensity of the left L4 nerve root (open arrow). The right L4 nerve root is normal (solid arrow). (7) Right-sided pain and radicular symptoms due to sacroiliitis. Axial fat-suppressed T2-weighted MR image (4000/110) shows edema and enlargement of the right nerve roots (arrow). Note the increased signal intensity at both sacroiliac joints (*).

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**Figure 8.** Polyneuropathy. Axial fat-suppressed T2-weighted MR image (4600/110) of the leg shows denervation-related signal intensity changes in the medial gastrocnemius (MG), lateral gastrocnemius (LG), soleus (S), tibialis posterior (TP), and tibialis anterior (TA) muscles. The extensor digitorum longus (EDL) and peroneal (P) muscles are relatively spared.
malignant psoas syndrome, may clinically mimic cauda equina syndrome (10,11). Neoplastic plexopathy most commonly follows direct invasion of the psoas muscle by adjacent tumors of colorectal, ovarian, uterine, or cervical origin. It may also occur in the setting of metastatic spread of disease, most commonly from breast cancer, sarcoma, lymphoma, and multiple myeloma. Direct invasion of the lumbosacral plexus from perineural tumors is less common.

Infectious or arthritic diseases of the sacroiliac joints, pelvic and hip fractures, and pelvic surgery may directly injure or stretch the sacral plexus (Fig 5). Colorectal and cervical cancers can directly involve or compress the sacral plexus. Pelvic irradiation and aortic aneurysms are less common causes of sacral plexopathy.

Most patients present with significant and debilitating pain, proximal muscle weakness and wasting, sensory deficits, and loss of deep tendon reflexes. They have difficulty getting up from a chair and climbing stairs. Bowel and bladder incontinence can rarely occur and clinically mimic cauda equina syndrome.

The imaging features of lumbosacral plexopathy depend on the underlying process. Psoas muscle abnormalities, such as seen in malignant psoas syndrome, range from focal or diffuse enlargement to masslike deposits in the muscle. Neural enlargement and increased T2 signal may also be seen (Fig 6). MR imaging evidence of inflammatory sacroiliac joint disease (Fig 7) and of pelvic fractures dictates careful examination of the adjacent nerve roots.

As opposed to injuries to peripheral nerves where symptoms follow stereotypical innervation patterns, injuries that involve the lumbosacral plexus manifest with symptoms and skeletal muscle changes related to multiple spinal levels or multiple nerve distributions (Fig 8). When the symptoms are unilateral, the contralateral plexus can be used as an internal standard for comparison of signal intensity. However, bilateral involvement should raise the possibility of a systemic process such as diabetes mellitus, neurofibromatosis (Fig 9), or metastatic disease, most commonly from breast carcinoma.

**Femoral Nerve**

*Normal Anatomy and Imaging.*—The femoral nerve is a mixed motor and sensory nerve and the largest branch of the lumbar plexus. After it is formed from the L2–L4 nerve roots, the nerve (along with direct contributions from the first two or three lumbar roots) innervates the psoas muscle. While in the pelvis, the nerve travels through the iliacus compartment, a constrained space bound by the iliacus and psoas muscles and roofed by the iliacus fascia. Approximately 5 cm before exiting under the inguinal ligament, the nerve gives off a motor branch to the iliacus and psoas muscles.

In the proximal thigh, the femoral nerve travels in a rigid fibromuscular canal bound by the inguinal ligament, iliopsoas muscle, and iliopsoas fascia. Here, it immediately splits into an anterior division innervating the pectineus and sartorius muscles and a posterior division innervating the quadriceps muscles. The saphenous nerve, which arises from the posterior division, provides sensory innervation to the distal medial thigh, anteromedial knee joint, and medial leg and foot.
The femoral nerve in the groin is at risk for injury in the setting of hernia repair and after arterial punctures; in this setting, scar may compress or deviate the course of the nerve. Neural compression, retraction, or vascular insult can occur during gynecologic (hysterectomy) and pelvic and hip (joint replacement) surgeries. The nerve is also susceptible to compression against the inguinal ligament in the lithotomy position and after femoral catheterization.

Alterations in the course, signal intensity, and size of the femoral nerve are easier to detect in the intrapelvic portion of the nerve. In the thigh, these changes are less obvious owing to the smaller size of the nerve.

Pathologic Conditions.—The femoral nerve is most vulnerable to injury while traveling in the iliacus compartment and at the level of the inguinal ligament. Most reports of entrapment neuropathies of the femoral nerve are related to traumatic injuries to the iliopsoas compartment, with computed tomographic and MR imaging evidence of mass effect due to iliacus or iliopsoas muscle tear and hematoma (13–15). Femoral neuropathy secondary to iliopsoas compartment masses and a distended iliopsoas bursa has also been described (16).

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Alterations in the course, signal intensity, and size of the femoral nerve are easier to detect in the intrapelvic portion of the nerve. In the thigh, these changes are less obvious owing to the smaller size of the nerve.

Signal intensity alterations compatible with denervation of the iliopsoas muscle signify injury to the intrapelvic femoral nerve. (The proximal psoas muscle is also directly innervated by the L2 and L3 nerve roots.) Conversely, injuries at or distal to the inguinal ligament spare the branch to the iliopsoas muscle and instead may manifest as
signal intensity changes in the pectineus and sartorius muscles and the quadriceps muscle group (Fig 11) (17).

Clinically, femoral neuropathy is associated with decreased knee extension (quadriceps weakness) and decreased hip flexion (iliopsoas muscle weakness). An absent knee jerk and sensory deficits in the anterior and medial thigh and medial knee, leg, and foot are also noted. Femoral neuropathy may be clinically confused with lumbar plexopathy and L4 radiculopathy. Conservative treatment of femoral neuropathy should be attempted first, since the complications associated with surgical intervention can have serious consequences.

Lateral Femoral Cutaneous Nerve

Normal Anatomy and Imaging.—The LFCN is a sensory nerve that arises from the first three lumbar roots and has a relatively variable course. Most commonly, the nerve emerges from beneath the lateral aspect of the psoas and descends obliquely along the anterior surface of the iliacus muscle toward the ASIS. Here, the nerve may run under, over, or through the inguinal ligament approximately 1 cm medial to the ASIS. Distal to the inguinal ligament, the nerve branches into anterior and posterior divisions, which penetrate through the fascia lata, several centimeters distal to the ASIS, and provide sensory innervation to the anterior and lateral thigh, respectively (18).

The LFCN has a nearly horizontal intrapelvic course along the anterior surface of the iliacus, so it is rarely identified on axial images. Occasionally, it may be visualized as tiny dots of low intermediate signal intensity, accompanied by vessels, as it approaches and then pierces the fascia adjacent to the ASIS. The nerve abruptly dives vertically into the subcutaneous tissues at the level of the inguinal ligament, approximately 1 cm from the ASIS, and may serendipitously be seen arborizing in the subcutaneous fat of the upper thigh on the anterior-most coronal images. Therefore, it is important to include the anterior pelvic subcutaneous tissues on sagittal and coronal images.

Pathologic Conditions.—Neuropathy of the LFCN has been termed meralgia paresthetica and manifests as sensory deficits and paresthesias in the anterolateral thigh. The nerve is most commonly injured by mechanical compression at the level of the inguinal ligament. Constricting clothes or girdles, tool belts, and seat belts can extrinsically compress the nerve. A protuberant abdomen may compress the nerve against the inguinal ligament, accounting for symptoms in the obese and in women in late pregnancy. The sartorius muscle may compress the nerve in dancers when the leg is in the “turned out” position.

The LFCN is a sensory nerve, and imaging is performed to identify structural abnormalities that may physically compress it. In the appropriate clinical setting, neuropathy can be inferred when there is mass effect along the course of the nerve. Potential scenarios include avulsion fractures of the ASIS, sartorius tendon injury, pelvic osteotomy, and acetabular fracture. The proximity of the nerve to the iliac crest puts the nerve at risk for iatrogenic injury during anterior iliac bone harvest. Its branches lie close to the standard anterior portal for hip arthroscopy and can be injured during hip replacement surgery or laparoscopic hernia repair. The LFCN can also be compressed or tethered once a scar has formed. Signal intensity alterations of the LFCN are difficult to visualize at MR imaging owing to the small size of the nerve. Imaging must include the skin and subcutaneous tissues to include the nerve and its branches.
cular course, the nerve divides into anterior and posterior branches. The anterior branch descends anterior to the adductor brevis muscle and supplies the hip joint. It sends motor branches to the gracilis, adductor brevis, adductor longus, and, less frequently, pectineus muscles and provides sensation to the medial thigh. On axial images, the anterior branch is found in a thin stripe of fat posterior to the pectineus and adductor longus muscles and anterior to the obturator externus and adductor brevis.

The posterior branch descends posterior to the adductor brevis muscle to supply the knee joint. It supplies the obturator externus, the adductor component of the adductor magnus, and occasionally the adductor brevis and provides sensory innervation to the medial knee. On axial images, the posterior branch is found in a thin fat plane, first within and then distal to the obturator externus, in between the adductor brevis and adductor

Figure 12. Normal obturator nerve. (a) Axial T1-weighted MR image (553/12) shows the obturator neurovascular bundles entering the obturator canals (arrows). (b) Axial T1-weighted MR image (553/12) obtained below the obturator canal shows the anterior (arrowhead) and posterior (arrow) branches of the right obturator nerve. AB = adductor brevis, AL = adductor longus, OE = obturator externus, P = pectineus. (c) Coronal T1-weighted MR image (553/12) shows the obturator nerves (arrows) descending toward the obturator canals.

Obturator Nerve

Normal Anatomy and Imaging.—The obturator nerve arises from the ventral L2–L4 rami of the lumbar plexus. The nerve runs along the iliopectineal line and exits the pelvis via the obturator canal at the superior aspect of the obturator foramen (Fig 12).

In the pelvis, the obturator nerve descends vertically in the coronal plane and nearly vertically in the sagittal plane posteromedial to the psoas muscle. Abundant perineural fat makes it easy to identify the nerve in all imaging planes. It is slightly smaller and brighter than its accompanying vessels on T1-weighted images. The nerve is anteromedial to the obturator internus muscle at the level of the acetabular roof, just before it dives into the obturator canal.

The 3-cm-long obturator canal is a fat-filled constrained space in the superior aspect of the obturator foramen that transmits the obturator neurovascular bundle. It is bound by the bony obturator foramen superiorly and the obturator membrane inferiorly (19). During its intracanali-
magnus muscles. The divisions of the nerve may be difficult to visualize in young, muscular patients owing to paucity of intermuscular fat. Variability in the obturator nerve and its branches is common, as is communication with the femoral nerve and, when present, with the accessory obturator nerve.

Pathologic Conditions.—The obturator nerve is relatively protected by its deep intrapelvic location and surrounding muscles. Obturator neuropathy is relatively uncommon; it is most frequently seen in the setting of pelvic trauma or surgery and is related to mass effect and stretching, respectively. In the latter case, prolonged lithotomy position during genitourlogic surgeries and excessive retraction during total hip replacement surgery are reported (20). Mass effect on the nerve commonly occurs around the region of the obturator canal or as the nerve enters the thigh and may be related to pelvic trauma, periarticular cysts or bursae, or hernia (Fig 13).

Sports-related obturator neuropathy is believed to be due to tethering of the anterior branch of the obturator nerve in the thigh by fascial adhesions associated with adductor brevis tendinopathy. In male athletes (whose bony pelvic geometry forces the nerve to curve around bone structures at a more acute angle), the obturator nerve can also be tethered against the normal fascia, anterior to the adductor brevis. In cases where no anatomic basis for compression can be identified, anesthetic nerve block is confirmatory. The obturator nerve may be secondarily affected in the setting of osteitis pubis (21).

At MR imaging, mass effect on the nerve (eg, by a hematoma, fracture, bursa, tumor, metastasis, or periacetabular screws and cement) as well as infiltration of the fat around the nerve just proximal to and within the obturator canal can be appreciated (Fig 14). Infectious or inflammatory processes involving the symphysis pubis, particularly osteitis pubis, may secondarily involve the nerve in the obturator canal and manifest as infiltration of the perineural fat. The anterior and posterior branches of the obturator nerve may be more difficult to evaluate because of their small size and the paucity of fat between the muscle planes.
Figure 15. Motor innervation of the thigh. Axial image shows the motor innervation territories of the femoral nerve (quadriceps and sartorius muscles), the obturator nerve (adductor muscles), and the sciatic nerve (hamstring muscles).

The obturator nerve provides motor innervation to the muscles of the adductor compartment (Fig 15), and muscle changes related to denervation may be seen. It is particularly important to distinguish muscle strain from acute muscle denervation in the athlete with groin pain, as adductor tendinopathy may coexist with or lead to obturator neuropathy (22). Typically, alterations in muscle signal intensity related to adductor strain are focal, involve the fascial planes, and are located in close proximity to the muscle origin at the symphysis pubis. Conversely, denervation-related changes tend to be diffuse, may include the obturator as well as the adductor muscle group, and spare the fascial planes (12).

Variation in muscle innervation may result in unexpected patterns. For example, since the adductor brevis may be innervated by both the anterior and posterior branches of the obturator nerve, it may not exhibit denervation when only one of the nerve branches is entrapped. Similarly, the presence of the accessory obturator nerve may cause atypical signal intensity alterations in denervation. Clinically, the signs and symptoms of obturator neuropathy can also be caused by lumbar radiculopathy or plexopathy. In the latter instances, however, denervation-related muscle abnormalities will include muscles outside the distribution of the obturator nerve, such as the hamstring and quadriceps muscles.

Treatment of obturator neuropathy is initially conservative. In recalcitrant cases, nerve block or neurolysis and fascial resection may be considered.

Figure 16. Greater sciatic foramen. The piriformis muscle divides the greater sciatic foramen into the suprapiriformis foramen, where the superior gluteal nerve (curved arrow) exits, and the infrapiriformis foramen, where the inferior gluteal nerve (straight arrow) and sciatic nerve (arrowhead) exit.

Sciatic Nerve

Normal Anatomy and Imaging.—Formed by the L4–S3 nerve roots, the sciatic nerve (Figs 1, 16) is the largest nerve in the body. The nerve exits the greater sciatic foramen as distinct tibial and peroneal divisions, enclosed in a common nerve sheath. In the pelvis, the sciatic nerve may descend anterior to, above, or through the piriformis muscle. The nerve continues down the thigh, posterior to the adductor magnus and anterior to the gluteus maximus muscle. In the distal third of the thigh, the two divisions physically separate into tibial and common peroneal nerves.

The sciatic nerve is the lifeline of the lower extremity. It provides knee flexion by innervation of the posterior thigh muscles (Fig 15) and almost all sensory and motor functions below the knee. The tibial nerve provides all motor function to the posterior compartment of the leg and to the plantar muscles of the foot, while the common peroneal nerve provides motor function to the anterior and lateral compartments of the leg.

Because of its large size and abundant perineural fat, the sciatic nerve is easy to evaluate in all imaging planes (Fig 1). The nerve has intermediate signal intensity on T1-weighted images and mildly high signal intensity on fluid-sensitive
Figure 17. Sciatic neuropathy. Axial (a) and coronal (b) images from T2-weighted neurography (4340/145) show diffuse enlargement and increased signal intensity of the left sciatic nerve (arrow).

Figure 18. Bilateral sciatic neuropathy after plastic surgery. The surgery was performed with the patient in the lithotomy position for a long period. Axial fat-suppressed T2-weighted MR image (3500/50) shows increased signal intensity and size of both sciatic nerves (arrows).

images. High-resolution images demonstrate the distinct nerve fascicles arranged into two separate bundles, made up of the larger tibial division of the nerve and the more lateral and smaller peroneal division. The abundance of perineural fat should not be mistaken for fibrolipomatous hamartoma (nerve lipomatosis).

The sciatic nerve can be followed as it descends in the greater sciatic foramen and exits the pelvis under the inferior margin of the piriformis muscle. It curves around the ischial spine and descends lateral to the common hamstring tendons and posterior to the short external rotators of the hip. In the proximal thigh, the nerve is posterior to the adductor magnus and anterior to the hamstring muscles. At this level, the nerve flattens and may be more difficult to appreciate. It is again well seen in the distal thigh, where it splits into the common peroneal and tibial nerves.

Pathologic Conditions.—Symptoms of sciatic neuropathy depend on the level of injury and the relative involvement of the tibial and peroneal divisions. Injury more commonly affects the peroneal division of the nerve because its fibers are more superficial, have less supporting connective tissue, and are fixed at two points (the sciatic foramen and the fibular head); the tibial division is fixed only at the sciatic foramen (23). Not surprisingly, sciatic neuropathy may clinically mimic the more distal common peroneal neuropathy, since both entities can manifest as foot drop due to loss of innervation of the anterior tibial muscle (24).

Sciatic nerve entrapment can occur in the hip region and less commonly in the thigh. The most common cause of sciatic neuropathy at the hip is iatrogenic and is associated with total hip replacement, related to stretching or direct trauma of the nerve. Piriformis muscle syndrome, a specific cause of sciatic neuropathy, is discussed in greater detail in the next section.

Direct MR imaging evidence of sciatic neuropathy includes course deviation, increased size, and increased signal intensity of the nerve (Figs 17–20). Abnormal increased signal intensity is more easily detected than in other lower extremity nerves owing to the large size of the sciatic nerve.
Obliteration of the fat planes around the nerve owing to tumor, scar, edema, or hematoma may also be noted.

In the thigh, signal intensity alterations consistent with denervation can involve the hamstring muscles as well as the hamstring component of the adductor magnus (Figs 20, 21). However, this is uncommon due to the high takeoff of the nerve branches to these muscles. Conversely, muscle denervation more distally can be missed in routine MR imaging studies of the pelvis and hip, since most muscles supplied by the sciatic nerve are located in the knee, leg, and foot (Fig 22).

**Piriformis Muscle Syndrome**

Piriformis syndrome is a controversial entity that requires separate mention.

The piriformis muscle fills much of the greater sciatic foramen as it extends from the sacrum to the greater trochanter. It may have variable origins from the sacrum, potentially overlying sacral foramina. The sciatic nerve typically runs in the infrapiriformis portion of the greater sciatic foramen; however, the nerve or one of its divisions, typically the peroneal, may lie above or course through the piriformis muscle (25).

Both known and unknown inciting factors lead to a common end pathway of hypertrophy, spasm, contracture, or inflammation and scarring of the piriformis muscle, which, in the setting of piriformis syndrome, can compress the sciatic nerve. The process may be initiated by trauma with muscle injury, hematoma, and scarring, as well as by acute or chronic muscle stretching associated with gait disturbances. In most cases, no inciting event is recalled.

The diagnosis of piriformis syndrome is challenging, as the symptoms are often nonspecific and electrodiagnostic tests are difficult to perform due to the deep location of the nerve.

Imaging diagnosis of piriformis syndrome is similarly problematic. Although the diagnosis can be inferred from alterations in the appearance of the affected nerve, evaluation of the size of the piriformis muscle and comparison with the contralateral side are less than satisfying, as muscle anomalies and significant variations in size are noted in symptomatic and asymptomatic individuals. In a review of 100 patients with no history or clinical suspicion of piriformis syndrome, Russell et al (26) found that muscle size asymmetry of 2 mm was present in 81% of patients. None of the patients with asymmetry of 4 mm or more had symptoms suggestive of piriformis syndrome.

Using MR neurography, Filler et al (27) found that, of the patients who responded well to piriformis surgery, 38.5% had ipsilateral muscle hypertrophy and 15% had muscle atrophy. Muscle asymmetry alone had a specificity of 66% and sensitivity of 46% in identification of patients with muscle-based piriformis syndrome. Conversely, ipsilateral nerve edema was associated with reproducible symptoms of piriformis syndrome (during adduction or abduction of the flexed internally rotated thigh) in 88% of patients. Use of both asymmetry of the piriformis muscle and increased nerve signal intensity improved the diagnostic
ability of MR neurography, with 93% specificity and 64% sensitivity in predicting the outcome of piriformis surgery.

Although there continues to be great interest in imaging diagnosis of piriformis syndrome, most authorities agree that delineation of neuromuscular relationships is the most important role of imaging. Until reproducible and reliable criteria for diagnosis have been established, piriformis syndrome should remain a diagnosis of exclusion (28).

The treatment of piriformis syndrome is initially conservative and includes anti-inflammatory medication, physical therapy, and image-guided corticosteroid muscle injection (29). Because the proposed underlying process relates to muscular compression, the use of botulinic toxin A has been explored with promising results (30). Surgical release of the sciatic nerve and sectioning of the piriformis muscle may be considered in refractory cases.
Figure 23. Normal superior gluteal nerve in the sciatic foramen. Coronal T1-weighted MR image (553/12) shows the superior gluteal neurovascular bundle (arrows) as it exits the pelvis above the piriformis muscle (*).

Superior Gluteal Nerve

Normal Anatomy and Imaging.—The superior gluteal nerve is formed by the posterior roots of L4, L5, and S1. It exits the pelvis and curves under the roof of the greater sciatic foramen, just above the piriformis muscle (Fig 23), to occupy the fat-filled space between the gluteus medius and minimus muscles. The superior branch ascends, then terminates in the gluteus minimus muscle. The inferior branch gives rise to branches to the gluteus medius and minimus and ends in the tensor fasciae latae.

The superior gluteal nerve is often seen on coronal and sagittal images as it exits the pelvis, closely applied to the bony brim, in the supra-piriformis foramen. The superior and inferior branches of the nerve are also occasionally detected. However, distinguishing the nerve from the adjacent superior gluteal vascular pedicle may be difficult. The nerve can also be seen on axial images as it travels laterally in the fat plane between the gluteus minimus and medius muscles.

Pathologic Conditions.—Surgery, particularly excessive retraction or inadvertent resection during total hip replacement, is the most well-recognized cause of superior gluteal nerve injury. The most inferior branch of the nerve is vulnerable to iatrogenic injury during the direct lateral approach to the hip; the branches to the tensor fascia latae are at risk with an anterolateral or anterior approach (31). The high variability in the branching pattern and course of the nerve makes it even more susceptible to injury at surgery. Subclinical electromyographic abnormalities of both the superior and inferior gluteal nerves have been described in up to 77% of patients after total hip replacement, regardless of the surgical approach (32).

Injury to the superior gluteal nerve is also a well-recognized complication of percutaneous placement of iliosacral screws (33). As the nerve

Figure 24. Neuropathy secondary to bilateral sacro-iliitis. Coronal fat-suppressed T2-weighted MR image (6100/80) shows increased signal intensity in both sacroiliac joints (*). The sciatic (open arrows) and superior gluteal (arrowheads) nerves and possibly the inferior gluteal nerves (solid arrows) have increased size and signal intensity.

Figure 25. Superior gluteal neuropathy. Coronal fatsuppressed T2-weighted MR image (8340/35) of the pelvis shows denervation edema of the tensor fasciae latae (TFL) (arrow).
 exits the pelvis under the pelvic brim, it is commonly compressed by prominent osteophytes, bony excrescences related to fracture, and infectious and inflammatory processes (Fig 24).

MR imaging of the superior gluteal nerve is challenging. The nerve exits along the horizontal plane above the piriformis muscle and may be difficult to see in this location. Its branches can be seen between the gluteus medius and minimus. The most commonly encountered manifestation of superior gluteal nerve injury is related to denervation changes of the gluteus minimus, gluteus medius, and tensor fascia lata (Fig 25). It is useful to compare the size and signal intensity of the tensor fascia lata with those of the contralateral one in elderly patients, as this muscle is often atrophied in this population.

Superior gluteal nerve injury manifests as weakness in abduction of the hip. A gait limp, a positive Trendelenburg sign, and abnormal results of electrophysiologic studies are also noted. Resolution of symptoms occurs in a high percentage of patients within 1 year after total hip arthroplasty.

Inferior Gluteal Nerve

Normal Anatomy and Imaging.—The inferior gluteal nerve, the main motor nerve of the gluteus maximus, originates from the dorsal L5, S1, and S2 rami. It lies medial to the sciatic nerve and exits the pelvis through the greater sciatic foramen, inferior to the piriformis muscle. At the lower border of the piriformis muscle, the nerve turns backward and divides into upward and downward diverging branches, which enter the gluteus maximus. The nerve may also send a branch to the posterior femoral cutaneous nerve.

Pathologic Conditions.—Inferior gluteal entrapment neuropathy is rarely reported but is recognized as a complication of the posterior approach to hip arthroplasty. Like the superior gluteal nerve, the normal inferior gluteal nerve is often noted on coronal images, exiting the pelvis adjacent to the sciatic nerve. The nerve can be followed on axial images in the fat plane deep to the gluteus maximus. Direct abnormalities of the nerve may be difficult to detect due to the small size of the nerve, although signal intensity alterations in the gluteus maximus may be encountered (Figs 26, 27).
Conclusions

Familiarity with the regional topographic anatomy of the nerves and the patterns of nerve distribution is crucial in MR imaging assessment of neuropa-thy. As we advance our understanding of nerve injury and recovery, on both the electrophysiological and histologic level, and improve our ability to image smaller and smaller nerves, MR imaging will play a greater role and may even, in certain settings, replace electrophysiologic studies in the work-up and management of entrapment neuropathies of the lower extremity.

References

MR Imaging of Entrapment Neuropathies of the Lower Extremity Part 1. The Pelvis and Hip

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At MR imaging, neural disease is inferred from alterations in nerve signal intensity, size, morphology, and location. Nerve disease is presumed when there are secondary signs of neural injury (e.g., denervation of the supplied muscles in the case of motor nerves).

Page 984
Normal nerves have intermediate signal intensity on T1-weighted images and are isointense or mildly hyperintense to muscle on T2-weighted and other fluid-sensitive images.

Page 984
T2-weighted neurography is accomplished by using sequences with long echo times (at least 90 msec), radiofrequency saturation pulses to suppress signal from adjacent vessels, and frequency-selective fat suppression (3,4).

Page 985
Unlike in tendons, however, where the magic angle artifact can be overcome with longer echo times (>40 msec), spurious high nerve signal intensity related to the magic angle can persist at higher echo times (66 msec) as well as on short inversion time inversion-recovery images. These angle-specific signal changes must be kept in mind when interpreting MR images, particularly when increased nerve signal intensity is the sole abnormality.

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Injury more commonly affects the peroneal division of the nerve because its fibers are more superficial, have less supporting connective tissue, and are fixed at two points (the sciatic foramen and the fibular head); the tibial division is fixed only at the sciatic foramen (23).