Peroneal intraneural ganglia: the importance of the articular branch. A unifying theory

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Object. Based on a large multicenter experience and a review of the literature, the authors propose a unifying theory to explain an articular origin of peroneal intraneural ganglia. They believe that this unifying theory explains certain intriguing, but poorly understood findings in the literature, including the proximity of the cyst to the joint, the unusual preferential deep peroneal nerve (DPN) deficit, the absence of a pure superficial peroneal nerve (SPN) involvement, the finding of a pedicle in 40% of cases, and the high (10–20%) recurrence rate.

Methods. The authors believe that peroneal intraneural lesions are derived from the superior tibiofibular joint and communicate from it via a one-way valve. Given access to the articular branch, the cyst typically dissects proximally by the path of least resistance within the epineurium and up the DPN and the DPN component of the common peroneal nerve (CPN) before compressing nearby SPN fascicles. The authors present objective evidence based on anatomical, clinical, imaging, operative, and histological data that support this unifying theory.

Conclusions. The predictable clinical presentation, electrical studies, imaging characteristics, operative observations, and histological findings regarding peroneal intraneural ganglia can be understood in terms of their origin from the superior or tibiofibular joint, the anatomy of the articular branch, and the internal topography of the peroneal nerve that the cyst invades. Understanding the controversial pathogenesis of these cysts will enable surgeons to perform operations based on the pathoanatomy of the articular branch of the CPN and the superior tibiofibular joint, which will ultimately improve clinical results.

Key Words • peroneal nerve • intraneural ganglion • cyst • tumor

Although relatively rare, the peroneal intraneural ganglion cyst has historically been a controversial clinical entity because of its poorly understood pathogenesis, unusual clinical presentation, and tendency to recur postoperatively. Based on a large clinical experience and an extensive review of the literature on peroneal intraneural ganglia, we present a unifying theory that will clarify these issues by providing an anatomical explanation for intriguing observations made by others. Among these are the universal occurrence of intraneural ganglia near joints and their tendency to extend proximally, the high percentage of preceding trauma and of knee or superior tibiofibular joint abnormalities, the predominance of DPN dysfunction, an absence of pure SPN involvement, the frequent (40%) finding of a pedicle to the superior tibiofibular joint, and a high (10–20%) postoperative recurrence rate.

The Unified Theory

We believe that peroneal intraneural ganglia originate from the superior tibiofibular joint (Fig. 1) and that the articular branch of the peroneal nerve serves as a conduit for the cyst fluid to pass from a capsular defect in the joint. This possibly occurs by a one-way valve (such as a ball valve, Bunsen valve, or hydraulic mechanism). Patients such as those with abnormal joints or joint capsules (that is, those in whom these sites have been previously traumatized, degenerated, or congenitally weakened) may be predisposed to the development of intraneural ganglion cysts. Under pressure, the articular branch may become markedly enlarged. The fluid appears to take the path of least resistance, typically dissecting proximally through the epineurium up the articular branch to the DPN and the DPN component of the CPN, or even the sciatic nerve. Within the CPN, the cyst is formed eccentrically within the epineurium, the result being lateral displacement and even compression of the nerve fascicles. The SPN fascicle(s) may also be displaced over time.

Because the peroneal nerve is by far the most common site of intraneural ganglia, we have chosen it as the prototype in support of our theory, although our theory can also be extrapolated to explain the development of extraneural peroneal ganglia as well. Such extraneural ganglia are well known to arise from the superior tibiofibular joint via capsular defects and to extend intramuscularly, intracompartmentally, or even intraosseously. We suggest that the finding of combined intraneural and extraneural ganglia (Fig. 1) or intraneural and intraosseous ganglia...
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indicates a shared mechanism. The concurrent presence of these different types of ganglia (especially when they are separate and connected to different parts of the joint) (Fig. 2), and the development of an intraneural or extraneural cyst after otherwise successful treatment of the other type of cyst in the same patient illustrates the spectrum of joint pathology. Recurrences are likely to be related to a persistent pathological condition within the superior tibiofibular joint. The cyst fluid may reform from continued production within the joint and, depending on the dynamic increased intraarticular pressures associated with loading and joint mechanics, the fluid may be forced out of the joint through the point of maximal weakness in the capsule, resulting in intraneural, extraneural, or combined intra–extraneural recurrence.

This unified theory for peroneal ganglia is intuitively attractive in that it follows the principle of Occam’s razor. It is also in keeping with the observations made by others. It has been difficult in the past to substantiate any particular theory, largely because of the rarity of these lesions, the retrospective nature of case reports, the diverse international literature, the limited use of MR imaging, and, often, a short follow-up period. For this reason, the controversy continues to this day.

The articular branch is important to the “synovial theory,” which has been supported by others including Brooks, Parkes, and Robert, et al. Still, an articular branch connection has reportedly been found in only approximately 40% of published cases of peroneal intraneural ganglia. We believe the pathological articular branch was not appreciated in the remaining cases. Because the presence and importance of this branch is not widely recognized, it is often not explored, even by experienced peripheral nerve surgeons. This has been demonstrated in our retrospective review of recurrences of previously treated cysts. In addition, the connection to the joint is not always obvious and may be missed. Seddon’s observation was that an intraneural ganglion can “lose its original connection” to the joint. Loss of a neural connection (articular branch) seems to be an unlikely occurrence to us. Articular connections of ganglia may be small, seemingly remote, externally normal, and not well known. It is not surprising that recent case reports have identified communications between other intraneural ganglia and their neighboring joints via articular branches, specifically those affecting ulnar, tibial, and median nerves in relation to elbow, superior tibiofibular, or radioscaphoid joints.

This theory also seems more logical than others put forth, such as the role of embryonal rests, cystic degeneration of a tumor, or organization of a hematoma. An additional theory, focal degeneration of epineurial or perineurial tissue, advocated by several authors, remains very popular. For it to support the predilection of intraneural ganglia to joints, however, it would have to combine degenerative change with a reproducible mechanical trauma within a confined anatomical space. Nevertheless we recognize that this degenerative theory may play a role in our theory, albeit at the joint or capsular level. Some authors have suggested that the cyst arises in the CPN and extends to the articular branch, thereby giving a false impression that it arises from the joint. This explanation of de novo cyst formation within the CPN with consistent anterograde filling of the articular branch also seems unlikely. This is not to say that the occasional joint-unassociated ganglion of nerve does not occur. We simply have not encountered one. Finally, we have also considered the possibility that an extraneural ganglion may infiltrate or invade the epineurium, producing an

Fig. 1. Composite drawing showing the spectrum of joint-related ganglia, their shared pathogenesis, and their relationship to the neural elements. Ganglia can be classified as either intraneural or extraneural, or they can be combined. An extraneural ganglion can follow a track along the course of the CPN or one of its branches, become adherent to this nerve, and cause extrinsic compression; this may be mistaken as an intraneural ganglion. br = branch. (Printed by permission of Mayo Foundation.)

Fig. 2. Two sequential T2-weighted fast–spin echo images with fat saturation obtained using a 3-tesla MR imaging system. (See the legends to Figs. 7 and 10 for further details on this patient.) A: The finding of an intraneural cyst (asterisk), a complex blow-out cyst (arrowhead), an intraosseous cyst of the fibular head (open arrow), and the “tail” (arrow) to the neighboring superior tibiofibular joint all in the same patient is indicative of a shared mechanism. B: One can clearly see the connection (curved arrow) of the tail of the cyst (arrow) to the superior tibiofibular joint, the complex lobular cyst (arrowhead), and the cyst (asterisk).
intraneural cyst; we do not believe that the path of least resistance for an extraneural cyst would lead to invasion of the nerve. Regardless of the exact mechanism of the cyst formation, which remains incompletely understood, the articular branch appears to be critical in the propagation of cyst fluid.

Part of the confusion related to intraneural ganglia also rests in the semantics. First, not all intraneural cysts are intraneural ganglia. Other cystic lesions do occur, such as cystic schwannoma, neurofibroma, sarcoma, and myxoma, all of which can be misdiagnosed as intraneural ganglia. Second, not all ganglia affecting the nerve are intraneural ganglia. Extraneural ganglia frequently adhere to surrounding structures and can extrinsically compress the nerve by their proximity or size; therefore, they may be easily confused with intraneural ganglia. Indeed, both of these lesions are represented in earlier reports of ganglia that affect the nerve. In some cases, it is difficult to specify from the literature the exact nature of the ganglion. Finally, intraneural ganglia are often referred to as “synovial ganglia,” although histological examination demonstrates no resemblance of these cysts to the synovium.

Anatomical Evidence Supporting the Theory: the Articular Branch of the Peroneal Nerve

If the articular branch is accepted as a main component in the pathogenesis of these cysts, this explains the location of the lesions near joints and the propensity of the cyst for proximal dissection. Anatomical dissection of 20 cadaveric limbs was performed to study the branching pattern of the distal CPN, especially the articular branch, and the innervation of proximal muscles of the anterior and lateral compartments at this level. In all cases, the articular branch arose from the DPN (within 3 mm), just distal to the division of the CPN, into the DPN and SPN (Fig. 3). A relatively large J- or U-shaped recurrent articular branch was routinely demonstrated and, at times, approached the size of the DPN and SPN. We therefore consider the bifurcation of the CPN to be a trifurcation. This recurrent branch

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**TABLE 1**

Muscle innervation determined during dissection of 20 cadaveric limbs*

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Mean No. of Nerve Branches to Muscle (range)</th>
<th>Pattern of Muscle Innervation (No. of Limbs)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>DPN</td>
<td>Art Br Alone</td>
</tr>
<tr>
<td>TA</td>
<td>2.0 (1–3)</td>
<td>2</td>
</tr>
<tr>
<td>EDC</td>
<td>2.1 (1–3)</td>
<td>13</td>
</tr>
<tr>
<td>EHL</td>
<td>1.3 (1–2)</td>
<td>20</td>
</tr>
<tr>
<td>PL</td>
<td>2.6 (2–5)</td>
<td>0</td>
</tr>
<tr>
<td>PB</td>
<td>1.2 (1–2)</td>
<td>0</td>
</tr>
</tbody>
</table>

* Art Br = articular branch; EDC = extensor digitorum communis; EHL = extensor hallucis longus; PB = peroneus brevis; PL = peroneus longus; TA = tibialis anterior.
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Fig. 5. Schematic drawings of the internal topography of the CPN and its branches. A: Twenty-four millimeters below the neck of the fibula, fascicles of the articular branch (G) are separate, but close to fibers leading to the tibialis anterior muscle (triangle), the terminal section of the DPN (white squares), and the extensor digitorum longus muscle (open box). Additional combined fibers leading to the extensor digitorum longus muscle (box with diagonal) and the extensor hallucis longus muscle (circle with plus sign) are farthest from the articular fibers. B: At the neck of the fibula, fibers from the articular branch have merged with a fascicle to the tibialis anterior muscle. This fascicular group is closest to others of the DPN and farthest from the SPN. The articular fibers join the DPN trunk as a single fascicle. B = peroneus brevis muscle; C = cutaneous innervation; L = peroneus longus muscle; T = terminal section of the SPN. Reprinted with permission from Sunderland S: Nerves and Nerve Injuries, ed 2. Edinburgh: Churchill Livingstone, 1978.

Patients with peroneal intraneural ganglia share a common clinical presentation and similar electrodiagnostic findings of a predominant DPN involvement. The clinical course evolves in a predictable, sequential fashion that can be interpreted in relation to the intraneural anatomy (Fig. 6 and Table 2).

Typically, patients initially complain of poorly localized knee or proximal leg pain, which is presumably due to involvement of a pathological condition of the superior tibiofibular joint (Stage 0) and/or the articular branch (Stage I). This pain could be characterized as mechanical or proprioceptive. Resection of an articular branch (such as that performed in ulnar nerve transpositions or as part of joint denervation procedures) does not typically produce neuropathic pain. Although patients may present with joint pain or a new leg mass, it is the development of a neurological motor deficit or neuropathic pain that usually brings patients to medical attention.

With further cyst enlargement, the fibers of the DPN, which are in direct proximity to the articular branch, will start to fail first (Stage II). This may result in neuropathic pain and a neurological deficit in the DPN. Muscle weakness typically begins with a foot drop (weakness of the tibialis anterior muscle) before involving the toe extensors. Weakness in dorsiflexion may occur in Stage I as a result of the involvement of a tibialis anterior branch directly from the articular branch, or in Stage II due to the internal topography of the peroneal nerve (Fig. 5) and the partial selective involvement of DPN fascicles. This initial weakness of dorsiflexion is often misdiagnosed as a CPN palsy. These patients may also have a sensory disturbance in the DPN distribution (first dorsal web space). Last, the presence of neuropathic pain and motor weakness may be more apparent than sensory symptoms. This may be due to the selective involvement of motor fibers resulting from neural compression.

Patients with peroneal intraneural ganglia usually have DPN deficit alone. Involvement of the SPN component can occur with long-standing compression (Stage III) or as a result of more proximal, diffuse cystic involvement of the nerve. Nevertheless, mild weakness in the peroneus longus muscle could also be explained by the occasional instance in which the DPN partially innervates this muscle. These

Clinical Evidence Supporting the Theory: Predominance of DPN Dysfunction

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patients would demonstrate either weakness of the peroneus brevis and peroneus longus muscles or sensory abnormality of the dorsum of the foot. No patient with a peroneal intraneural ganglion has been reported to have isolated SPN involvement, without a DPN abnormality.

Dissection may even extend proximal to the sciatic nerve bifurcation within the CPN component (Stage IV). This has been documented to produce peroneal nerve symptoms. We recently treated a patient (not included in our clinical series because there was an insufficient follow-up review) in whom there were tibial nerve symptoms as well as findings of a DPN lesion from proximal extension of the cyst (Fig. 7). We are unaware of another similar case in the literature.

Little is known of the temporal evolution of intraneural ganglia. Whether their formation is abrupt (uniphasic), episodic, or only slowly progressive, the result is the same. The occasional episodic nature of neurological symptoms and findings and the waxing and waning of the mass may be related to fluctuations in intraarticular pressure. Increased intraarticular pressures may be the result of axial loading, certain knee positions, repetitive motion, or physical activity.

**Imaging Evidence in Support of the Theory: Connection With the Superior Tibiofibular Joint**

In our experience, MR imaging has demonstrated a connection between the cyst and the superior tibiofibular joint capsule, but this connection may not be obvious and may be easily missed if thin enough sections are not obtained. The finding of a connection to this capsule, in conjunction with the MR imaging signal characteristics of the cyst, can firmly establish the diagnosis. The evidence of a persistent cyst on imaging ("tail sign") demonstrates that the connection to the joint remains intact (potentially patent). Magnetic resonance imaging can reveal connections not identified at the operation. This mode of imaging is the best to demonstrate the longitudinal extent and origin of the intraneural cyst and its selective muscle denervation. Magnetic resonance imaging can also be used to explain ap-
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TABLE 2
Stages of peroneal intraneural ganglia: correlation with clinical symptoms and signs*

<table>
<thead>
<tr>
<th>Clinical Symptoms &amp; Signs</th>
<th>Stage I</th>
<th>Stage II</th>
<th>Stage III</th>
<th>Stage IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>pain</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>mechanical</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>neuropathic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>potential neurological deficit (motor w/ or w/o sensory)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DPN</td>
<td>–</td>
<td></td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>SPN</td>
<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>sciatic†</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
</tbody>
</table>

* + = potentially present; – = absent.
† The peroneal division would be preferentially compressed, but the tibial division could also be affected.

parent “skip” lesions or normal external appearances noted during the operation. Other imaging modalities, including ultrasonography, CT scanning, and arthrography, may be indicated in the evaluation of cystic masses or ganglia about the knee, some of which can also compress the peroneal nerve. Although CT scanning and ultrasound have been used in the diagnosis of nerve-related ganglia, particularly peroneal intraneural ganglia, MR imaging is preferable in their evaluation. Whereas satisfactory MR images of peroneal intraneural ganglia have been obtained using 0.5- to 1.5-tesla magnets and standard imaging protocols, MR neurography, an imaging method that enhances the conspicuity and resolution of peripheral nerves, is the procedure of choice. Magnetic resonance neurography performed using a 3-tesla clinical magnet has not only demonstrated the intraneural location of the peroneal cyst, but has clearly delineated the pathological articular branch. Magnetic resonance neurography will play an increasingly important role in the diagnosis, treatment, and follow up of patients with peripheral nerve lesions.

Other imaging modalities have confirmed a true communication that was indicated by an imaging connection between the cyst and the joint. During preoperative dye injection of the superior tibiofibular joint (Fig. 8) and arthrography of the knee joint, especially studies performed using delayed imaging (via communication through the superior tibiofibular joint) (Fig. 9), the intraneural cyst (articular branch) has filled with contrast material. These observations support our use of intraoperative injections and our one-way valve theory.

Operative Evidence in Support of the Theory: a Pathological Articular Branch and the Characteristic Appearance and Location of the Intraneural Ganglion Cyst

In all our cases, the so-called pedicle of the cyst to the joint was the articular branch. A clearly pathological, hollow articular branch (when transected) was demonstrated in all surgically treated cases in which the cyst was connected to the superior tibiofibular joint. At times, it was grossly enlarged and cystic, but rarely, it had a normal caliber and/or an external appearance (Fig. 10). This was confirmed histologically.

As mass lesions, the peroneal intraneural ganglia have a stereotypical appearance. Whether in our series or in the literature (including those thought not to connect to the superior tibiofibular joint), these lesions appeared in the same location of the nerve (the medial portion of the distal CPN, the DPN, and the articular branch). They expanded their dimensions proximal to the point at which the CPN passes under the fibrous arch of the peroneus longus muscle, that is, where external resistance is lower.

Outcome Evidence in Support of the Theory: Analysis of Success and Recurrence

In our series, in addition to cyst decompression, ligation of the articular branch eliminated intraneural recurrence without cyst resection. We are also aware of several cases that were surgically treated more than a decade ago and had a lengthy follow up in which the articular branch was not identified or treated during the operation, but the superior tibiofibular joint was obliterated. These patients fared well, presumably due to the fact that treatment of the joint included the inadvertent disconnection or detachment of the articular branch from the joint and eliminated the source of synovial fluid.

In our series, the cyst walls of the peroneal intraneural ganglia were not resected. The lack of reformation of the ganglia also argues strongly against a local, cyst-based source of mucin. In the two patients in our report with symptomatic recurrences of extraneural ganglia, removal of the synovium and resection of the superior tibiofibular joint alone (without excision of the cysts) eliminated the cysts on follow-up imaging.

Operative failures and recurrences are often predictable. 1) Relatively “simple” procedures such as blind percutaneous biopsy, limited open biopsy, aspiration, or incomplete cyst decompression may result in neuropathic pain or recurrence. 2) If the articular branch is not identified and eradicated, the resection is incomplete and recurrence is likely. 3) Intricate dissection of the cyst with internal neurolysis or resection of the cyst wall frequently produces neurological loss and pain. 4) Nerve resection with interpositional grafting should be avoided, wherever possible, be-
cause lengthy split repairs have not permitted useful recovery.5 6) Revision operations risk neurological compromise. Despite successful treatment of intraneural ganglia by ligation of the articular branch and cyst decompression, extraneural ganglia may still result.185

**Pathological Evidence in Support of the Theory**

The gross, histological, immunohistochemical, and ultrastructural features of the intraneural ganglia that we studied185 were those of a degenerative, nonneoplastic lesion. Their features very closely resembled those of ordinary ganglion cysts of soft tissue at varying stages of formation. The epineurial tissue appeared normal, aside from an increase in collagen content in the surroundings of the cysts. The general appearance of the cyst was in keeping with joint fluid having been mechanically introduced under pressure into the interfascicular epineurium. The process of cyst formation appears to go through phases, beginning with extrusion of joint fluid into the epineurium and its longitudinal dissection within this compartment. The mucus tract may be either solitary or may, on cross-section of the nerve, give the impression of being multiple.154 Once the fluid has been introduced, the soft tissue surrounding it undergoes a slow organization. The result is a fluid-filled cyst with a compact collagenous wall composed of fibroblastic/myofibroblastic cells, which are also found in conventional ganglia of soft tissues. Such cells bear no resemblance to the synovium. A regular finding, one also noted by other authors, is the presence of small cysts adjacent to the main one. These appeared to be more recent in development in that they were thin walled and surrounded by mucus. We believe these are, in every way, analogous to the “pseudopodia” or “blow-outs” previously described to be located in the periphery of soft-tissue ganglia.136 Presumably these smaller cysts form by extrusion of fluid from the main cyst through defects in its wall that have been created by increased pressure.

Our study (described in our companion article185) provides convincing evidence of a joint space source for the fluid. Its consistency may vary, becoming more viscid with time, but it remains essentially joint fluid originally produced by the synovium.136 Whether the fluid is modified by a contribution of interstitial fluid from the cyst wall remains conjecture. Our gross findings lend little support to the notion that the mucus in joint-related intraneural ganglia originates from myofibroblasts that reside in the cyst wall.176 In rare cases of intraneural ganglia that are unrelated to a joint, elaboration of stromal mucins by myofibroblasts may well contribute to the cyst content. Thus, the mechanism of these myofibroblasts may explain the occasional occurrence of both conventional196 and intraneural ganglia that lie remote from or unrelated to joints, where in such instances, fibroblasts and myofibroblasts may indeed play a role in ganglion formation. The results of our study185 confirmed these authors’ finding of myofibroblasts176 within the cyst wall of intraneural ganglia; however, such cells are ubiquitous in reactive soft tissues. We consider it unlikely that joint-related intraneural ganglia owe their “secretory activity” solely to the contribution of reactive myofibroblasts.

In addition to the histological resemblance of intraneural and extraneural ganglia,9 the histological characterization of intraneural ganglia with and without pedicles has been identical. Differences between intraneural ganglia of the sort described herein and ordinary ganglion cysts of soft tissue are few. As a rule, ordinary soft-tissue ganglia are globular and many, but not all, are associated with a joint space. Instead, intraneural ganglia are elongated structures limited to a defined tissue compartment and, in our opinion, are almost invariably associated with a joint. In summary, our findings fully support the fact that ganglion cysts of the nerve are epineurial lesions and, although paraarticular, bear no histological resemblance to synovial cysts, such as Baker cysts.

**Analogy to Common Simple Ganglia, Synovial Cysts, and Rare Adventitial Cysts**

Intraneural ganglia are, in many ways, analogous to common simple ganglia, including those occurring on the dorsal aspect of the wrist. These ganglia develop at sites of ligamentous or capsular stress (like the scapholunate interval).5 Trauma14 (whether acute or repetitive) has been implicated in the pathogenesis of these lesions, and intraarticular derangements have often been found in the wrist.5,21 Interestingly, arthrograms7 show that dye injected into the wrist communicates with the ganglia, but dye injected into a ganglion will not enter the wrist joint.2 Such ganglia have been shown operatively and histologically to connect to the wrist joint by means of a pedicle via a continuous tortu-
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Fig. 10. Additional intraoperative and histological findings in the patient presented in Figs. 2 and 7. The articular branch connection to the joint seen during the operation corresponded well to the preoperative MR findings (demonstrated in Fig. 7). The intraneural cyst was traced from the distal CPN to the articular branch (red vessel loop) and to its most distal connection to the joint capsule (green vessel loop). The DPN, SPN, and other muscular branches are mobilized in blue vessel loops. A: A “skip lesion,” a relatively normal external segment of the nerve (red vessel loop) between two clearly abnormal cystic elements. This skip lesion was identified as the articular branch was traced distally beneath the peroneus longus muscle. The finding of an unaffected portion of nerve may be an indication to some surgeons that the distal end of the nerve lesion has been identified; however, a large blow-out cyst (articular branch) is apparent more distally. B: Distal to this large cyst, the articular branch connection to the joint becomes evident. The osteotome is in the superior tibiofibular joint. C: The superior tibiofibular joint has been resected (white arrow). The connection of the articular branch is well visualized. D: Photomicrograph of the most distal portion of the peroneal intraneural ganglion (segment in the green vessel loop in other panels) showing its lumen (asterisks) and accompanying fascicles of the articular branch (black arrows). Trichrome stain.

ous duct to the scapholunate ligament. The wrist joint (for example, the scapholunate joint) pumps out a modified joint fluid through a complex interconnected set of cysts that forms a one-way valve into the primary ganglion. These soft-tissue ganglia may encroach on neighboring nerves (that is, extraneural ganglia) causing compression. To avoid recurrence of the cyst, a successful operation must address the pedicle. This is typically done openly when the cyst is excised along with the pedicle and its “footplate,” but in fact, the main cyst may be left in situ and only the valvular system excised. An arthroscopic approach addresses the stalk of the cyst and a rim around the dorsal capsule (the site of capsular origin), ensuring that the one-way valve mechanism is resected; the procedure also decompresses the cyst and preserves the scapholunate ligament. Similar findings, pathogeneses, and treatment approaches also apply to other joint-related ganglia.

Implications of This Unified Theory

Pathogenesis. Based on a review of the literature and of our patients, we believe that trauma to the superior tibiofibular joint is important in the development of peroneal intraneural ganglia. The superior tibiofibular joint is a small synovial joint that dissipates torsional stresses at the ankle and lateral tibial bending movements, and provides tensile strength. It is a joint prone to both cumulative microtrauma and acute macrotrauma. Injury to the superior tibiofibular joint is apparent after the joint has received direct trauma or following ankle injuries, and diseases of this joint have been associated with diseases of the knee, especially with regard to the medial compartment. In more than half of the cases of intraneural peroneal ganglia that we reported, MR imaging revealed abnormal signal and arthritic changes within the superior tibiofibular joint and the knee joint, although it is possible that these changes in the knee are primarily or secondarily related to the cyst or are incidental findings. The superior tibiofibular joint is well known for its association with intraneural and extraneural ganglia of the peroneal and tibial nerves. Approximately 85% of the cases of peroneal intraneural ganglia have been identified in male patients, who are more predisposed to such trauma than female patients. The mean age of patients is 34 years (range 4–74 years).

Five mechanisms potentially contribute to the formation of peroneal intraneural ganglia: 1) direct trauma to the area
of the fibular neck; torsional trauma transmitted proximally from the ankle or the inferior tibiofibular joint; communication of the superior tibiofibular joint with the knee joint, as demonstrated on knee arthrography (Fig. 9), (a communication previously reported to occur in 10% of adults, although recently demonstrated to occur in 64% of specimens when MR arthrography was used); indirect trauma affecting the superior tibiofibular joint such as that produced by altered knee biomechanics (for comparison, review Cases 1 and 2 in our companion article); the joint capsule may be congenitally weakened. The finding of bilateral intraneural ganglia suggests a congenital predisposition.

In addition, we wonder whether the preponderance of peroneal intraneural ganglia over tibial ganglia may be a reflection of the underlying mechanism or the anatomy, for example, torsion stressing the lateral joint, intrinsic weakness related to the lateral capsular anatomy, or the relative proportion in size of the articular branch to the capsule. We also wonder whether the frequency of peroneal and ulnar intraneural ganglia is due to two factors: 1) their having relatively large articular branches; and 2) their proximity to joints predisposed to trauma.

Patient Outcomes

Treatment must address the pathoanatomy (Fig. 11). It is clear that the articular branch is an important factor in the development of intraneural ganglia. Still, the underlying pathological condition affecting the superior tibiofibular joint needs to be understood as the fundamental problem and, therefore, a joint procedure needs to be considered as well.

The development, type, extent, and timing of recurrence are not always predictable. Cyst fluid may preferentially dissect along different paths, depending on the integrity of the joint capsule and the neural pathway. For example, pathological or postoperative changes such as the presence of scarring or cyst loculations may result in cyst persistence due to incomplete decompression of the ganglia at surgery. If the pathway followed is out the same capsular defect into the articular branch, then intraneural recurrence will ensue (Fig. 12) (perhaps the point at which the articular branch enters the capsule is a vulnerable site for extravasation). Extraneural recurrence most commonly occurs when there is another defect in the superior tibiofibular joint capsule, but may also be due to an inadvertent transection of the articular branch or a rupture of the intraneural cyst’s contents. The occurrence of an extraneural ganglia can follow an operation for extraneural ganglia, and it seems plausible that the occurrence of an intraneural ganglia can also follow an operation for extraneural ganglia. Some re-
currences after a cyst operation may appear immediately postoperatively, or in a delayed manner. Massive or rapid recurrences may be misinterpreted as a malignancy and episodic recurrences as a peripheral neuropathy.

Conclusions

Based on our large clinical experience and a review of the literature, we believe that peroneal intraneural ganglia are in direct continuity with the superior tibiofibular joint capsule via their connection to the articular branch of the nerve and their communication via a one-way valve. With elevated intraarticular pressures, cyst propagation may occur, with the dissection along the path of least resistance. The characteristic pattern is for the fluid to dissect proximally up the articular branch within the epineurium to involve the DPN preferentially within the CPN before compressing the neighboring SPN fascicle(s). Knowledge of the pathogenesis of cyst formation is necessary to improve treatment and eliminate recurrences. In addition to cyst drainage, exploration and ligation of the articular branch is of paramount importance in the successful operation of intraneural ganglia. Although the articular branch is the important common denominator in the pathogenesis, we believe that the superior tibiofibular joint is the primary site from which the pathological condition arises. To this end, resection of the pathological superior tibiofibular joint (with its synovium) is likely to be a necessary part of the procedure in selected cases. Intraneural or extraneural recurrence frequently occurs when the pathoanatomy is not adequately addressed.

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