

Intraneural ganglia: a clinical problem deserving a mechanistic explanation and model

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Intraneural ganglion cysts have been considered a curiosity for 2 centuries. Based on a unifying articular (synovial) theory, recent evidence has provided a logical explanation for their formation and propagation. The fundamental principle is that of a joint origin and a capsular defect through which synovial fluid escapes following the articular branch, typically into the parent nerve. A stereotypical, reproducible appearance has been characterized that suggests a shared pathogenesis. In the present report the authors will provide a mechanistic explanation that can then be mathematically tested using a preliminary model created by finite element analysis. (DOI: 10.3171/FOC.2009.26.2.E11)

KEY WORDS • cyst • finite element analysis • intraneural ganglia

INTRANEURAL ganglion cysts are mucinous lesions found within the epineurium of nerves. They occur most commonly in the peroneal nerve but have been described in many nerves in the vicinity of synovial joints. These intraneural ganglion cysts typically result in neurological deficit due to the displacement of nerve fascicles by the cyst contents.^{44,45,47}

Intraneural ganglion cysts have been considered curiosities for 2 centuries. Different theories, without a scientific basis, have been proposed.^{4,6–8,15,19,20,24,27} The articular (synovial) theory,^{42,45} based on robust clinical, imaging, and histological evidence,^{11,39–42,44,45,47,49,50} provides a logical, consistent explanation that clarifies and unifies the observations made by many over the years. Developed on the prototype of the peroneal nerve ganglion cysts, the theory can be extrapolated to intraneural ganglion cysts involving other nerves. The core principle for the formation of these cysts is a joint connection via an articular branch. This finding can be reliably demonstrated with imaging and at operation provided appropriate techniques and experience.^{44,45} The fact that these articular connections may be small,^{36,44} seemingly remote,^{1,31,32,43,54} and externally normal³⁴ explains why they may not be readily recognized and why other theories have been proposed.

The recent characterization of predictable, stereotypical patterns of growth of intraneural ganglia^{42,45,46} as demonstrated by the peroneal nerve model (Fig. 1),

suggests that they are the result of a shared mechanism. Thus, it appears that this clinical problem can be simulated and solved. The design of a model would help define the precise pathogenesis underlying the formation and propagation of intraneural ganglia, and this in turn could refine treatment modalities. In this report we will provide a mechanistic explanation that then can be mathematically tested with a preliminary model created by using FEA.

Mechanistic Explanation

The mechanistic explanation of intraneural ganglion cyst consists of the analysis of the mechanical interactions of the cyst fluid, the nerve tissue (for example, epineurium), and the tissue surrounding the nerve (for example, muscle, bone, soft tissue) in relation to the environment (for example, gravity). As the mechanical interactions are specific to each interface (1] joint-capsule, 2] capsule-articular branch, 3] articular branch–parent nerve, and 4] parent nerve–proximal major nerve interfaces), each will be described individually. For simplicity, the peroneal nerve, the most common site for an intraneural ganglion cyst, will be used as the prototype. However, this mechanistic explanation can be applied to intraneural ganglion cyst of any nerve.

Joint–Joint Capsule Interface

The formation of the intraneural ganglion cyst oc-

Abbreviation used in this paper: FEA = finite element analysis.

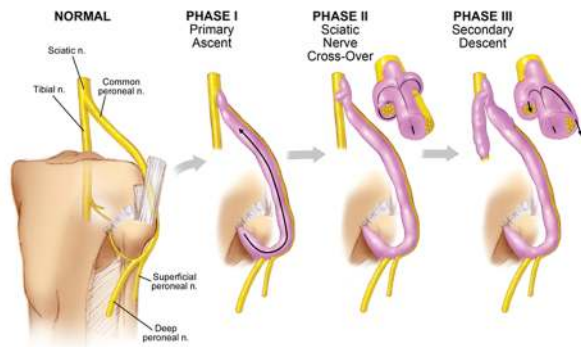


FIG. 1. A predictable phasic pattern for intraneural cyst formation and propagation in peroneal intraneural ganglia has been observed and described. Primary ascent (Phase I) allows cyst fluid derived from the anterior portion of the superior tibiofibular joint dissects along the articular branch into the common peroneal nerve. Cross-over (Phase II) occurs with expansion within the common epineurial sheath of the sciatic nerve. Secondary descent (Phase III) down the tibial and/or peroneal nerves can occur after cross-over. In this figure, the predominant primary ascending pathway is shown (primary descent, which is typically less prominent, may occur within the proximal portions of the deep and superficial peroneal nerve branches and is not depicted). n = nerve. Modified with permission from Spinner RJ, et al: Dynamic phases of peroneal and tibial intraneural ganglia formation: a new dimension added to the unifying articular theory. *J Neurosurg* 107:296–307, 2007.

occurs at the joint–joint capsule interface. Joint fluid is produced by the synovium of the joint and escapes through a capsular defect (rent). The capsular defect may be preex-



FIG. 2. Oblique coronal MIP (maximum intensity projection) image made from a VIPR (vastly undersampled isotropic projection reconstruction) data set shows a peroneal intraneural cyst arising from the superior tibiofibular joint (arrow) and extending via the articular branch into the parent common peroneal nerve (plus signs). Note the extraneural cyst (asterisk), which extends from the joint into the anterior compartment.

isting and is likely the result of a traumatic, degenerative, or congenital process.^{11,44,45} In the peroneal intraneural ganglia model, evidence suggests that direct or indirect cumulative trauma^{13,14,20,44} to the superior tibiofibular joint itself or in relation to the neighboring (and often communicating) knee joint is important in the development of the cysts.⁴⁵ Increased intraarticular pressure (that is, static or dynamic) is the other mechanism involved at the joint–joint capsule interface. Prior to the escape of the fluid through the defect, increased intraarticular pressure may lead to bulging of the joint capsule. This is possibly due to the relative ease to expand the capsular tissue initially. As the intraarticular pressure increases, the potential energy of the system increases. In accordance with the principle of minimum potential energy, it is then easier for the fluid to escape through the rent than to expand the capsule further. The synovial fluid “chooses” the path of least resistance. In the absence of a rent, increased pressure could result in rupture of the capsule.

Joint Capsule–Articular Branch Interface

In intraneural ganglion cysts the capsular rent is closely associated with an articular branch; in contrast, in extraneural ganglia the rent is distinct from the articular branch. At times, these 2 types of cysts can coexist (Fig. 2). In intraneural ganglia, the synovial fluid enters the articular branch. While a joint “connection” of the articular branch to a neighboring synovial joint can be well seen on imaging and at surgery, the direct “communication” between the joint and the cyst has been demonstrated on arthrography.^{10,18,23,26,31,32,40,41} Once the fluid has entered the nerve, the increased intraarticular pressures can result from: 1) continued production of synovial fluid within the joint and 2) the dynamic increase in intraarticular pressure associated with loading and joint mechanics. These increased forces would logically lead to an increase in the pressure within the newly initiated cyst. Resulting forces from the joint promoting cyst formation are greater than the resisting forces (Fig. 3). Resisting forces can be either intrinsic or extrinsic. Intrinsic resisting forces relate to the resistance to the elastic deformation of the nerve. Extrinsic resisting forces come from the surrounding tissue such as bone, muscle, or soft tissue (such as compartments). Gravity can also play a role in facilitating or resisting cyst propagation. The role of cyst resorption and/or the potential for rupture is unknown.

It seems intuitive that in clinically apparent, persistent cysts, the intraarticular pressure remains larger than the resisting forces, resulting in further cyst formation: expansion (in diameter) and extension (in length). Expansion and extension are further favored for two reasons: 1) maintenance of joint forces (that is, synovium continues to produce fluid, in fact probably at an increased rate due to the association of joint-related disease), and 2) the relative ease of further cyst growth (that is, increased volume of the cyst results in a reduction of the intrinsic resisting forces). This observation would be consistent with the well-established principle of energy minimization. Less energy is required for cyst expansion and extension, in view of the relatively weak neural tissue (especially in

A model to explain intraneural ganglia

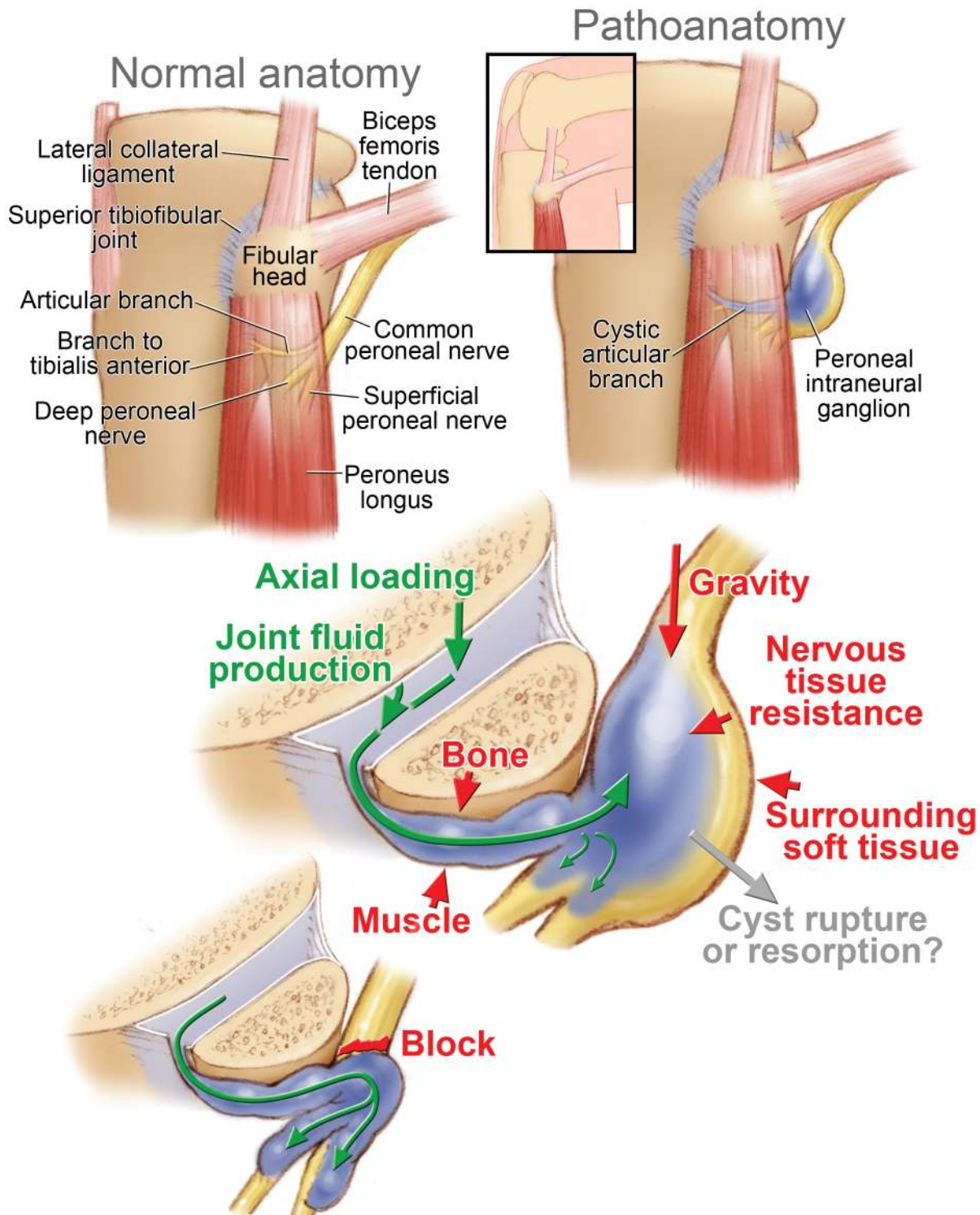


FIG. 3. The normal anatomy of the common peroneal nerve and its branches is shown in relation to the superior tibiofibular joint. A typical peroneal intraneural ganglion cyst (pathoanatomy) is illustrated. Its common features include: a cystic articular branch with balloon-like expansion at the level of the common peroneal nerve and preferential proximal ascent. These features suggest a common mechanism to explain their hallmark appearance. The different proposed forces playing a role in intraneural ganglia are shown. Increased intraarticular forces (joint fluid production and axial loading) facilitate the formation and propagation of the intraneural cyst (green labels). Resisting forces (nerve tissue, bone, muscle, soft tissue and gravity) are labeled in red. The unclear role of cyst resorption and/or rupture is shown in gray. Atypical patterns can be seen and explained by the presence of additional forces, such as a block (for example, caused by scar, surgery) that redirect the cyst into different pathways. Upper panel: Reproduced with permission from Spinner RJ, et al: Peroneal intraneural ganglia. Part I. Techniques for successful diagnosis and treatment. *Neurosurg Focus* 22(6):E16, 2007. Lower Panel: Printed with permission of Mayo Foundation, 2008.

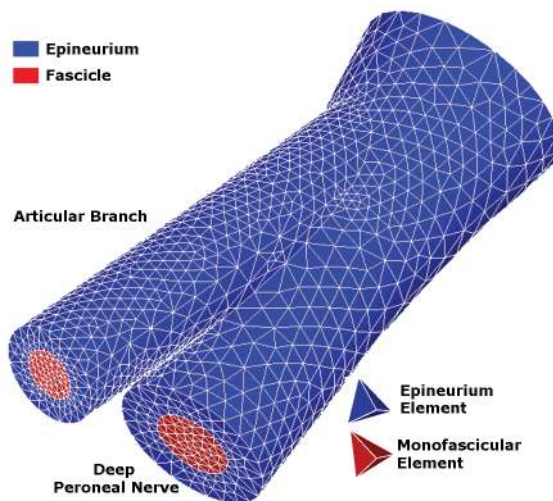


FIG. 4. An FEA model of the junction between the articular branch and the deep peroneal nerve. Two different nerve components are represented: the epineurial region (*blue*) and the fascicular region (*red*). Each region is assigned different material properties (that is, stiffness, strength, and so on). The triangular lines on the exterior imply building blocks called elements, which can be arranged to represent the mechanical behavior of the representative anatomical regions.

the epineurium), than for fluid reentry into the joint. The presence (or absence of) and type of valve is unknown.

Articular Branch–Parent Nerve Interface

At the articular branch–parent nerve interface, the same factors as those discussed above apply. With continued cyst growth, extension occurs within the confines of the epineurium into a parent nerve. At the articular branch–parent nerve junction, the cyst again follows the path of least resistance. It can either extend proximally and/or distally to varying levels. Contributing factors could include: 1) the degree of angulation of the articular branch to the parent nerve; 2) the relative location of the cyst within the articular branch and its specific position

as it reaches the articular-parent nerve junction (favoring growth along rather than around strong and stiff fascicles); and 3) increased additional resistances from intrinsic or extrinsic factors such as scarring or ligation. Any combination of these factors could dictate directionality. As clinical observation suggests, cyst expansion tends to be eccentric, displacing nerve fascicles (“signet ring” sign).⁴⁷ Assuming the absence of a morphological defect, the intraepineurial cleavage plane (specifically, within the outer epineurium) seems to be favored clinically. This could be explained by dissection according to the path of least resistance: 1) the outer epineurium appears to have less resistance than the combined inner epineurium and fascicles and 2) the continued forces promote cyst propagation within the same neural compartment. The cross-sectional anatomy of the articular branch and parent nerve interface is not well known; the location at which a well-defined inner and outer epineurium exists has not been characterized.

The articular branch is a small nerve branch and the diameter of its cystic enlargement in intraneural ganglia is relatively small compared with that of the parent nerve. This gives rise to the characteristic imaging features of intraneural ganglion cysts: a tubular cyst constrained by the epineurium with a small neck (“tail” sign) and balloon-like cystic involvement of the parent nerve (Fig. 2). The size and shape of the cyst in different regions are dictated by the architecture and diameter of respective neural branches and extrinsic forces overlying the neural tissues. The multilobulated but elongated appearance often seen clinically can potentially be due to the dynamic nature of the intraarticular pressures and variable effects from other forces. In contrast, the extrinsic and intrinsic forces for the formation of extraneural ganglia differ; their appearance tends to be more globular when they occur in soft tissue.

Parent Nerve–Proximal Major Nerve Interface

When a cyst propagates proximally and joins with another nerve, the cyst can either expand or elongate

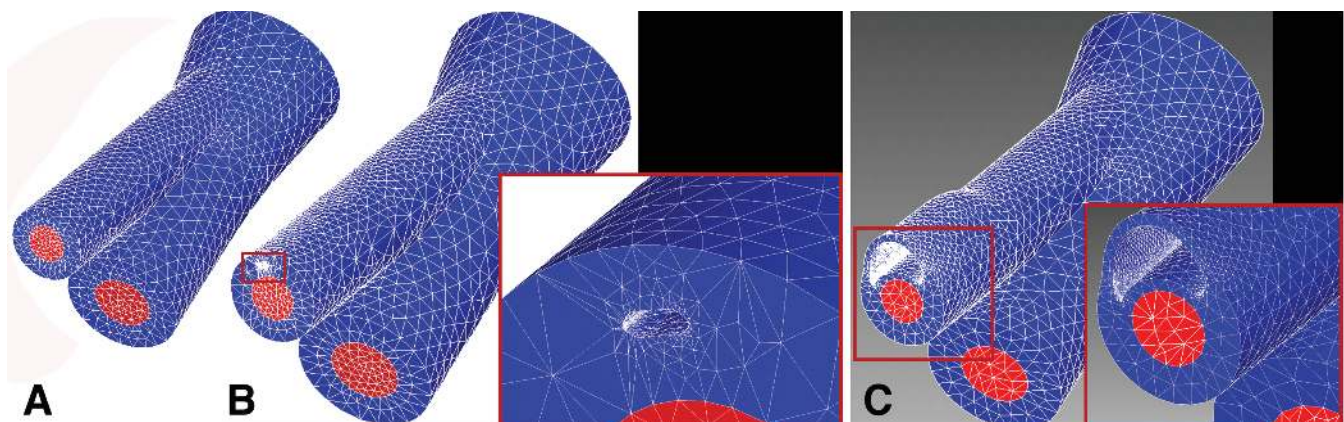


FIG. 5. Modeling of the development of an intraneural cyst. A: The FEA model without a cyst in the articular branch. B: The FEA model showing a small cleavage plane and the initiation of an intraneural cyst formation within the articular branch. A microscopic crack (*inset*) allows the influx of synovial fluid from the joint into the articular branch, initiating the process. C: At a more advanced stage (than in B), further influx of synovial fluid leads to growth and propagation of the intraneural cyst. This modeling appearance resembles the clinical appearance in intraneural ganglia.

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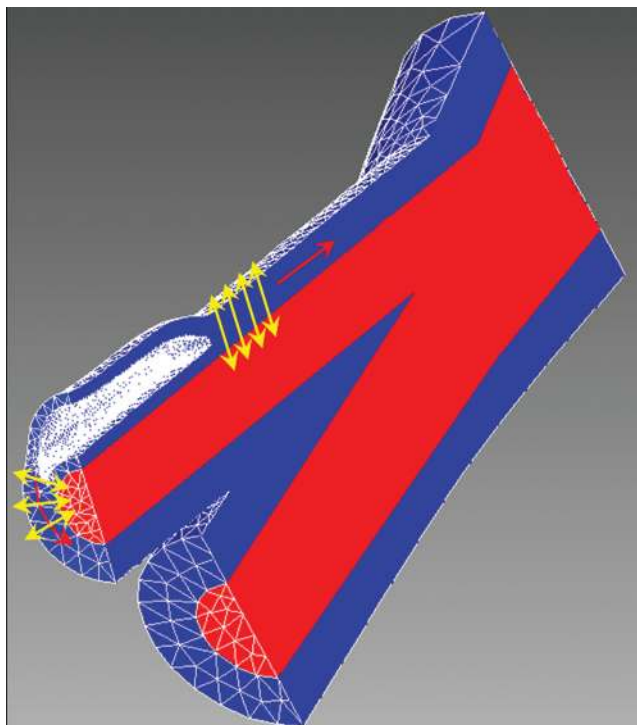


Fig. 6. The FEA model shows further influx of synovial fluid within the nerve. The *yellow arrows* indicate the direction of the forces causing the tissue to tear. The *red arrows* indicate the directions in which the cyst will tend to grow.

based on the aforementioned factors. With cyst expansion, cross-over can occur (that is, filling a common epineurial sheath whereby the cyst expands circumferentially around the entire nerve): a cyst originating in a single neural pathway can then affect or involve a second pathway. Pressure fluxes can dictate relative ascent or descent within the primary or secondary pathways. These processes explain the formation of several interconnected intraneural cysts—all dependent on the path of least resistance. In the case of a peroneal intraneural ganglion, different patterns may be seen: 1) continued ascent within the peroneal division of the sciatic nerve; or 2) cross-over within the sciatic nerve allowing cyst dissection within peroneal, tibial, and sciatic nerves.

Admittedly, the observations described of intraneural ganglion cysts are static representations of a dynamic process.⁴² Factors such as intraarticular pressure and/or pressure gradients may lead to dynamic fluxes over time.⁴² The dynamic component of pressure loading and associated dampening of pressure waves in fluid medium could explain differences in cyst propagation near and distant to the joint. These dynamic processes could explain the fluctuating clinical symptoms and signs,^{2,12,33,44} as well as the broad spectrum of operative and imaging findings encountered.⁴²

Finite Element Analysis Model

The aforementioned mechanistic explanation for the formation and propagation of intraneural ganglion cysts

can be described and studied by FEA, a computational tool that relates forces and deformations based on material properties. This technique has been widely used by engineers to study common clinical problems, and it has been applied to the study of peripheral vascular^{3,16,17,28,29,35,52,53} and cerebrovascular aneurysms.^{5,9,21,25,30,37,38} Because of some similarities in the development of aneurysms and intraneural ganglia, it seems logical that a similar approach can be used to model intraneural ganglia. In FEA, elements—or little volumes of material with ascribed properties—are arranged (Fig. 4) and subjected to prescribed forces and/or displacements. To analyze a given situation, the following components are necessary: material properties (stiffness and strength), geometry (dimensions), and boundary conditions (loads and deformation).

For example, for intraneural ganglia, prediction of cyst propagation patterns requires knowledge of nerve architecture and configuration; the material properties (such as material stiffness and/or material strength) of nerve components; and quantified intrinsic and extrinsic factors (for example, pressure of cyst fluid and surrounding tissues, respectively). It is important to note that a material's stiffness refers to the relative ease in stretching the material for a given applied force; a higher stiffness requiring a larger force for a fixed deformation. A material's strength refers to the greatest force that the material can withstand without undergoing failure.

Nerve Architecture and Configuration

To show feasibility, a simplified FEA model was constructed to represent a typical neural junction (for example, the intersection of the articular branch and the deep peroneal nerve) using the commercial code ANSYS (Fig. 5). The model consisted of about 60,000 tetrahedral-shaped elements. As is shown in Figs. 5 and 6, this simplified nerve was modeled as having a monofascicular region within a surrounding epineurium. The dimensions and the angulation of the nerves that were assigned to the FEA model were determined from values obtained in 2 limbs of 1 cadaveric specimen using a digital caliper with a 0.01-mm accuracy (Industrial Direct Co., Inc.): the mean diameters of the articular branch and the deep peroneal nerve were 1.94 and 2.89 mm, respectively; the deep peroneal nerve portion proximal to the junction with the articular branch and distal to its junction with the superficial peroneal nerve to form the common peroneal nerve was 3.97 mm; the mean angle at which the articular branch meets with the deep peroneal nerve was 16.5°.

Nerve Material Properties

Assignment of material properties to neural tissue was difficult due to the lack of experimental data available on the mechanical properties of nerve tissue components. Close examination of the nerve fascicular structure⁵¹ reveals the presence of aligned collagen proteins in a manner similar to ligaments. It was assumed, therefore, that the fascicular element would have stiffness properties (the relative ease in stretching the material for a given applied force) comparable to ligamentous tissue.²² The epineurium, in general, has comparatively less col-

lagen than the peri- and endoneurium;⁵¹ this region was assumed to have a stiffness of an order of magnitude less than the fascicular region. For computational simplicity, the strength properties (the greatest force that the material can withstand without undergoing failure) were not modeled.

Intrinsic and Extrinsic Factors

The effect of the pressure and tissue deformation caused by cyst fluid was simulated in the FEA model through the application of force vectors applied onto the inner walls of the cyst (Fig. 5). Because there are no experimental estimates of cyst fluid pressures available from the literature, an approximate value of about 0.5 psi was surmised based on the expected mechanical response of the nerve.

Results

The results of the FEA modeling indicate the direction in which the cyst will tend to grow (Fig. 6). Examination of the internal forces predicted by the FEA analysis reveals that tensile forces (*yellow arrows* in Fig. 6) will cause the cyst tissue to tear in a manner that will cause the cyst cavity volume to both increase in directions along the nerve and also radially around the fascicle (*red arrows* in Fig. 6). It is expected that this process will continue as long as cyst fluid accumulates. Eventually, the cyst will extend, reaching a nerve junction where it will continue to propagate in one or more new directions; the course the cyst takes will be dependent on the architecture of the neural junction and any intrinsic/extrinsic loads at that location.

Future directions of this research will consist of the incorporation of experimental material property data into the FEA simulations and the inclusion of a more intricate representation of the nerve microarchitecture as determined from anatomical and operative dissections. Cyst growth will be simulated as it approaches specific nerve junctions to elucidate the propagation patterns of cysts. The influence of surrounding muscle and bone tissue on the growth behavior of cysts will also be simulated.

Conclusions

The 3 fundamental principles of the unifying theory are: 1) an articular branch connection from a degenerative joint; 2) cyst fluid dissection along an intraepineurial path of least resistance; and 3) pressure fluxes.^{42,45,46} These principles proposed by clinical and imaging features can be analyzed more scientifically. In this paper, we have provided a mechanistic explanation for the formation and propagation of intraneural ganglia. In addition, this can be simulated and tested using FEA. Further development and manipulation of this model will lead to improved understanding of the clinical problem and, as such, improved clinical outcomes.

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