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Mary Claire Manske  
*Washington University School of Medicine in St. Louis*

Kathleen E. McKeon  
*Washington University School of Medicine in St. Louis*

Jeremy J. McCormick  
*Washington University School of Medicine in St. Louis*

Jeffrey E. Johnson  
*Washington University School of Medicine in St. Louis*

Sandra E. Klein  
*Washington University School of Medicine in St. Louis*

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# Arterial Anatomy of the Posterior Tibial Nerve in the Tarsal Tunnel

Mary Claire Manske, MD, Kathleen E. McKeon, MD, Jeremy J. McCormick, MD, Jeffrey E. Johnson, MD, and Sandra E. Klein, MD

*Investigation performed at Washington University School of Medicine, St. Louis, Missouri*

**Background:** Both vascular and compression etiologies have been proposed as the source of neurologic symptoms in tarsal tunnel syndrome. Advancing the understanding of the arterial anatomy supplying the posterior tibial nerve (PTN) and its branches may provide insight into the cause of tarsal tunnel symptoms. The purpose of this study was to describe the arterial anatomy of the PTN and its branches.

**Methods:** Sixty adult cadaveric lower extremities (thirty previously frozen and thirty fresh specimens) were amputated distal to the knee. The vascular supply to the PTN and its branches was identified, measured, and described macroscopically (the thirty previously frozen specimens, prepared using a formerly described debridement technique) and microscopically (the thirty fresh specimens, processed using the Spälteholz technique).

**Results:** On both macroscopic and microscopic evaluation, the PTN and the medial and lateral plantar nerves were observed to have multiple entering vessels within the tarsal tunnel. On microscopic evaluation, a vessel was observed to enter the nerve at the bifurcation of the PTN into the medial and lateral plantar nerves in twenty-two (73%) of the thirty specimens. There was a significant difference ( $p < 0.05$ ) in vascular density between the PTN and each of its branches.

**Conclusions:** The abundant blood supply to the PTN and its branches identified in this study is consistent with observations of other peripheral nerves. This rich vascular network may render the PTN and its branches susceptible to nerve compression related to vascular congestion. The combination of vascular and structural compression may also elicit neurologic symptoms.

**Clinical Relevance:** Advancing the understanding of the arterial anatomy supplying the PTN and its branches may provide insight into the cause and treatment of tarsal tunnel syndrome.

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The anatomy of the posterior tibial nerve (PTN) at the ankle is largely defined by its position within a fibro-osseous tunnel. Impingement of the nerve, tarsal tunnel syndrome, is a neurologic condition affecting the PTN and its associated branches at the medial aspect of the ankle. This syndrome often causes aching or burning pain, dysesthesias, and paresthesias on the medial border and plantar aspect of the foot. It is considered the most common nerve-compression disorder of the foot and ankle<sup>1</sup>. The borders of the proximal tarsal tunnel are classically described as the flexor retinaculum, which forms the roof of the tunnel, and the medial wall of the talus, the calcaneus, and the distal tibia, which form the floor. In addition to the PTN, the tunnel serves as a conduit for the

posterior tibial tendon, the flexor digitorum longus tendon, the posterior tibial artery and vein, and the flexor hallucis longus tendon<sup>2</sup>. The distal tarsal tunnel is defined by the course of the branches of the medial plantar nerve (MPN) and the lateral plantar nerve (LPN) as they travel adjacent to the abductor hallucis muscle<sup>3</sup>. The branching patterns of the nerve have been described, with the most common site of branching in proximity to the leading edge of the abductor hallucis fascia<sup>4-6</sup>. The fascia of the abductor hallucis has been considered as a site of compression in the clinical presentation of tarsal tunnel syndrome<sup>7</sup>. Frequently, venous comitantes and multiple branching veins are noted at the leading edge of the abductor hallucis fascia.

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**TABLE I Macroscopic Analysis**

No. of Specimens with a Vessel at Measured Distance (N = 30)	Distance of Vessel Entry Proximal to Medial Malleolus* (cm)	Distance of Vessel Entry Proximal to Abductor Hallucis Fascia* (cm)	Distance of Vessel Entry Distal to Abductor Hallucis Fascia* (cm)
<b>PTN</b>			
26 (87%)	4.0 ± 0.6		
17 (57%)	2.1 ± 0.6		
13 (43%)	0.7 ± 0.3		
<b>MPN</b>			
27 (90%)		0.8 ± 0.5	
25 (83%)			0.5 ± 0.4
22 (73%)			1.7 ± 0.7
5 (17%)			3.0 ± 0.4
<b>LPN</b>			
26 (87%)		0.8 ± 0.5	
19 (63%)			0.4 ± 0.3
8 (27%)			1.9 ± 0.7
5 (17%)			4.1 ± 0.6

\*The values are given as the mean and the standard deviation.

In contrast to other entrapment neuropathies, the pathogenesis of tarsal tunnel syndrome is often unclear and frequently multifactorial. Both intrinsic etiologies (venous congestion, hypertrophic flexor retinaculum, osteophytes, space-occupying lesions, arterial insufficiency, and ischemia) and extrinsic etiologies (trauma, footwear, hindfoot deformity, and edema) have been described<sup>8</sup>. The specific etiology of tarsal tunnel syndrome is identified in 60% to 80% of cases, with the remaining 20% to 40% considered idiopathic<sup>9</sup>. Among the cases of tarsal tunnel syndrome without a clear source of compression, vascular etiology has been considered. Both impaired circulation, causing ischemia, and abundant vasculature, causing nerve compression,

can result in peripheral nerve dysfunction, as evidenced both clinically and electrodiagnostically<sup>10-18</sup>. In the upper extremity, the extraneural and intraneural anatomy has been described for the median and ulnar nerves<sup>19-24</sup>. Both the distribution and location of arterial branches have been considered as contributing factors to nerve compression in these cases<sup>10,14,19-21</sup>. Knowledge of the arterial anatomy supplying the peripheral nerves may provide clinical insight into the pathogenesis of tarsal tunnel syndrome.

The purpose of this study was to provide a description of the extraneural and intraneural arterial anatomy of the PTN and its branches in the region of the ankle and hindfoot. Improved understanding of the arterial anatomy may provide

**TABLE II Microscopic Evaluation**

No. of Specimens with a Vessel at Measured Distance (N = 30)	Distance of Vessel Entry Proximal to Nerve Bifurcation* (cm)	Distance of Vessel Entry Distal to Nerve Bifurcation* (cm)
<b>PTN</b>		
26 (87%)	2.0 ± 0.7	
22 (73%)	4.1 ± 0.6	
22 (73%)	At bifurcation	
<b>MPN</b>		
23 (77%)		1.8 ± 0.8
15 (50%)		4.0 ± 0.8
<b>LPN</b>		
26 (87%)		1.3 ± 0.7
22 (73%)		3.6 ± 0.6

\*The values are given as the mean and the standard deviation.



Fig. 1  
A posterior view of the vascular supply from the macroscopic evaluation. An asterisk indicates the location of an entering vessel. PTN = posterior tibial nerve, Ach = Achilles tendon, and M = medial malleolus.

insight into the potential role of the surrounding vasculature in the development of tarsal tunnel syndrome.

### Materials and Methods

This study was determined to be exempt by our university's institutional review board. Thirty matched pairs of adult cadaveric lower extremities were obtained, for a total of sixty cadaveric lower extremities used in this study. Our technique for evaluating the microscopic and macroscopic vascular anatomy was previously described<sup>25</sup>. Macroscopic evaluation was performed on thirty specimens that were previously frozen but not embalmed. Because freezing of the specimens disrupts the microvasculature, fresh specimens obtained within seventy-two hours of death were used in the microscopic evaluation. Limbs were excluded if they had evidence of foot or ankle deformity or had undergone surgery or trauma. The number of rejected cadaveric limbs was not recorded.

Both fresh and frozen specimens were amputated at the junction of the proximal and middle-third parts of the leg. Circumferential skin incisions were made in each toe at the proximal interphalangeal joint. An 8-French triple-lumen catheter was inserted into the anterior tibial, posterior tibial, and peroneal arteries, and saline solution was injected into each artery under constant manual pressure until clear effluent was observed from the skin incisions in the toes. The saline-solution injection was followed by an injection of India ink into each artery under manual pressure until the cutaneous surface of the foot was stained with ink and ink was observed flowing from the incisions in the toes. Finally, blue latex (Ward's Science) was injected in an identical manner.

The specimens were frozen for forty-eight hours after the injections and then were thawed completely at room temperature and amputated 10 cm proximal to the ankle joint. A pin was placed through the calcaneus, the talus, and the tibia to maintain the integrity of the ankle joint. Macroscopic analysis was performed to identify the major vessel entry points into the PTN and its branches. Microscopic analysis confirmed true vascular entry and was further used to identify branching patterns within the nerve. Any areas of hypovascularity were identified on microscopic analysis.

### Macroscopic Evaluation

In the thirty previously frozen macroscopic specimens, the skin and subcutaneous fat were sharply excised from the specimens. The specimens were submerged in 6% sodium hypochlorite. As the soft tissues, including the vascular walls, were chemically debrided by the sodium hypochlorite, casts of the vessels created by the latex became visible. The specimens were evaluated at twenty-

minute intervals, and photographs were obtained to document the progression to capture all aspects of the vascular supply of the PTN. Debridement was complete when the PTN and its vascular supply were visible. The location of the vessels entering the PTN relative to the tip of the medial malleolus and abductor hallucis fascia was measured and recorded.

### Microscopic Evaluation

The thirty fresh specimens for microscopic evaluation were processed using the Spalteholz technique<sup>23</sup>. The skin and subcutaneous tissues were removed from these specimens through sharp dissection. The PTN was identified and carefully excised while preserving all vascular branches to the PTN, the MPN, and the LPN. The location of the leading edge of the abductor hallucis fascia was marked with a 2-0 polypropylene suture. The PTN and its branches were removed from 5 cm proximal to the bifurcation of the nerve into the MPN and LPN to 5 cm distal to the bifurcation. The harvested nerves were sutured to a wooden tongue blade and stored in 10% neutral buffered formalin for forty-eight hours and then washed in running water for two hours before serial dehydration in increasing concentrations of ethanol (50%, 75%, 95%, and 100%) for two hours at each concentration and an additional two hours in 100% ethanol. The nerves were then placed in xylene for twelve hours followed by twelve hours in a 1:1 solution of methylsalicylate and xylene, and then stored in methylsalicylate. Each nerve was evaluated using a stereoscopic microscope. The locations of all entering vessels relative to the bifurcation of the PTN into the MPN and LPN were recorded. All nerves were photographed in a methylsalicylate bath. As part of the microscopic analysis, the number of vessels per centimeter was calculated as a representation of the density of vessels entering each nerve. A Student t test was performed to determine if the vascular density of the PTN, MPN, or LPN was significantly different.

### Results

Both macroscopically and microscopically, the PTN, the MPN, and the LPN were observed to have an abundant arterial supply.

### Macroscopic Evaluation

In the thirty specimens used for macroscopic evaluation, a mean (and standard deviation) of  $1.9 \pm 0.7$  vessels (range, one to three vessels) entered the PTN within 5 cm proximal to the medial malleolus (Figs. 1 and 2). This mean does not include vessels

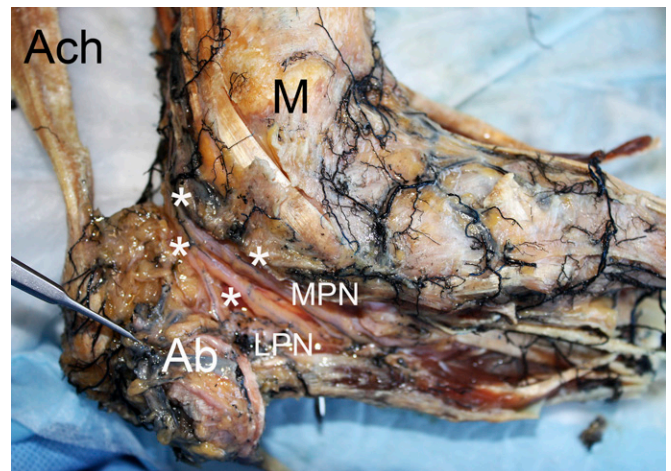


Fig. 2  
A medial view of the vascular supply from the macroscopic evaluation. An asterisk indicates the location of an entering vessel. MPN = medial plantar nerve, LPN = lateral plantar nerve, Ach = Achilles tendon, Ab = abductor fascia (pulled back), and M = medial malleolus.

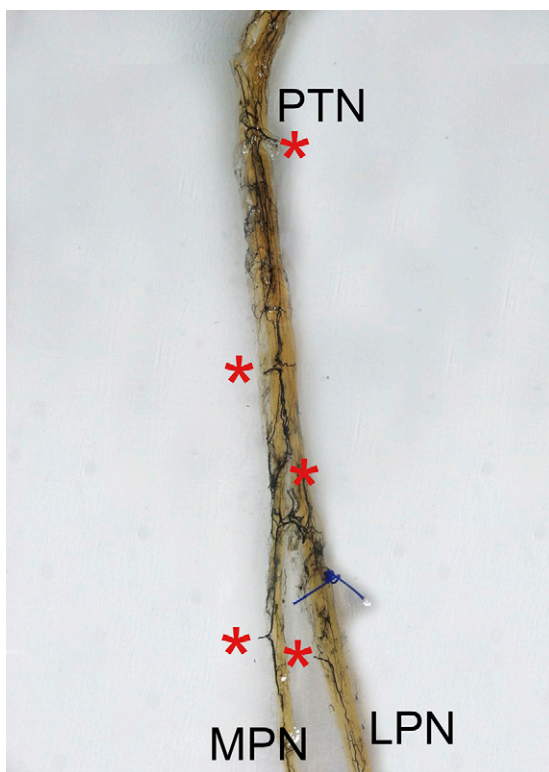


Fig. 3

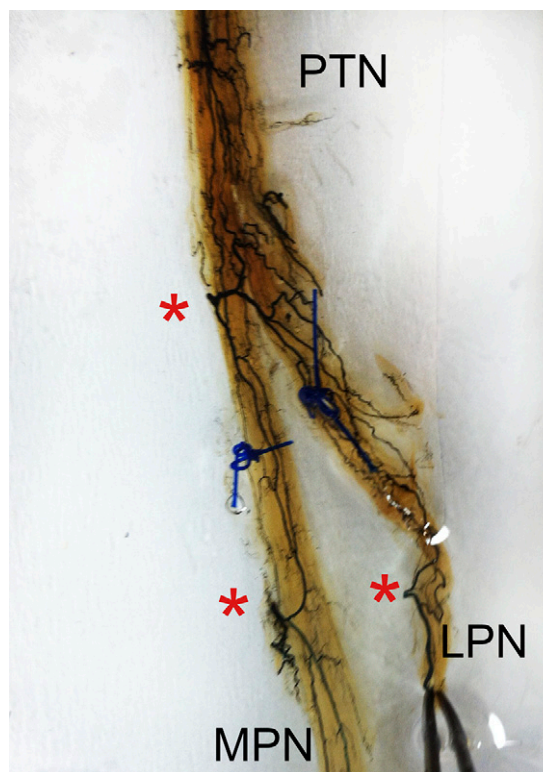


Fig. 4

**Fig. 3** A view of the nerves from the microscopic evaluation, demonstrating the location of vessels entering (\*) the posterior tibial nerve (PTN), the medial plantar nerve (MPN), and the lateral plantar nerve (LPN). The blue suture was placed at the level of the abductor hallucis fascia. **Fig. 4** A view of the nerves from the microscopic evaluation. An asterisk indicates the location of an entering vessel. Note the vessel entering at the bifurcation of the posterior tibial nerve (PTN), which supplies both the medial plantar nerve (MPN) and the lateral plantar nerve (LPN).

crossing over or lying adjacent to the PTN. There were three regions in which an entering vessel was identified: within 1 cm proximal to the medial malleolus, between 1 and 3 cm proximal to the medial malleolus, and between 3 and 5 cm proximal to the medial malleolus (Table I).

Macroscopically, vessels entering the MPN and the LPN were measured relative to the leading edge of the abductor hallucis fascia. The MPN was observed to be supplied by a mean of  $2.6 \pm 0.5$  vessels (range, two to three vessels). Very few specimens had a vessel entering the MPN >3 cm distal to the abductor hallucis fascia (Table I). Evaluation of the LPN identified a mean of  $1.9 \pm 0.6$  vessels (range, one to three vessels) supplying this nerve. The majority of vessels entered the nerve within 1 cm of the abductor hallucis fascia. Similar to the MPN, very few specimens had a vessel entering the LPN >3 cm distal to the abductor hallucis fascia (Table I).

#### Microscopic Evaluation

Microscopically, a mean of  $1.6 \pm 0.5$  vessels (range, one to two vessels) entered the PTN within 5 cm proximal to the bifurcation of the PTN into the MPN and the LPN, not counting the branch at the bifurcation (Fig. 3). A vessel was observed to enter the nerve at the bifurcation in twenty-two (73%) of thirty of the specimens (Table II). This vessel immediately divided into two branches, providing a vessel to both the MPN and the LPN (Fig. 4).

The MPN was supplied by a mean of  $2.0 \pm 0.3$  vessels (range, one to three vessels) within 5 cm distal to the bifurcation, including the vessel branch at the bifurcation (Table II). The LPN was supplied by a mean of  $2.3 \pm 0.6$  vessels (range, one to three vessels), including the branch at the bifurcation. Both the MPN and the LPN had variable distal vessel entry points, most commonly between 1 and 2 cm and between 3 and 4 cm distal to the bifurcation (Table II).

The mean vascular density (the number of vessels per centimeter) was  $0.17 \pm 0.07$  for the PTN,  $0.31 \pm 0.13$  for the MPN, and  $0.36 \pm 0.14$  for the LPN. There was a significant difference ( $p < 0.05$ ) in vascular density between the PTN and each of its branches, with the branches having a greater vascular density.

#### Discussion

Proper functioning of peripheral nerves requires adequate arterial supply while limiting nerve compression caused by abundant vasculature within a tight anatomic space. This is a relevant concern when considering the PTN in the tarsal tunnel. Both electrodiagnostic changes and clinical symptoms have been observed in response to nerve ischemia<sup>10,14,26</sup>. This relationship between abundant vascular supply and a confined space has been shown to cause nerve compression in the upper extremity. The Leash of Henry, the abundant network of vessels

of the recurrent radial artery near the radial nerve, is a well-recognized source of compression in radial tunnel syndrome, while a persistent or thrombosed median artery is a rare cause of carpal tunnel syndrome or pronator syndrome<sup>27,28</sup>. In the lower extremity, Sammarco and Chang reported that arterial vascular leases in the tarsal tunnel induced scarring and compression deformity of the PTN, and this was a common intraoperative finding at the time of tarsal tunnel release<sup>1</sup>. The deleterious effects of ischemia and direct compression are known to potentiate each other<sup>14,26</sup>.

Given the importance of vascular anatomy on peripheral nerve function, numerous investigators have evaluated the arterial anatomy of peripheral nerves, including the median, ulnar, and sciatic nerves, and have found an ample blood supply<sup>19-24,29</sup>. We evaluated the arterial supply to the PTN and its medial and lateral plantar branches, and report an abundant vasculature. This is consistent with what has been described in previous investigations of the PTN. As early as the late 19th century, both Tonkoff and Bartholdy independently described a profuse blood supply, especially in the distal aspect of the nerve<sup>30,31</sup>. Additionally, when evaluating the intraneural arterial supply to the sciatic nerve and its branches, including the PTN, with examination of histologic sections, Sunderland qualitatively described the PTN as "constantly receiving a large number (two to eleven) of small nutrient twigs at frequent though irregular intervals along its course."<sup>29</sup> More recently, Flanigan et al. performed a microscopic evaluation of the vascular supply of the PTN and its branches by injecting latex into thirteen cadaveric specimens (ten embalmed and three unfixated)<sup>32</sup>. They observed that supplying vessels entered the PTN approximately every 3.0 to 3.8 cm, which is similar to our findings. Additionally, they found a vessel consistently entering the nerve at the trifurcation of the PTN. Finally, they reported shorter distances (1.0 to 2.4 cm) between vessels entering the MPN and the LPN than between vessels entering the PTN, which is consistent with the greater concentration of vessels entering the MPN and the LPN compared with the PTN that was seen in our study.

In this study, we evaluated a large number of specimens, none of which were embalmed. We believe that the use of fresh specimens for microscopic analysis reduced the risk of damage to the microvasculature that could have impaired the ability to appreciate the arterial anatomy. We performed both microscopic and macroscopic analyses of the supplying vasculature. This allowed us to appreciate the arterial anatomy of the PTN and its branches relative to both macroscopic and microscopic anatomic landmarks. Injecting the arteries with latex allowed a macroscopic evaluation of larger vessels, while the injection of India ink facilitated the microscopic analysis by perfusing the small vessels to the nerve.

From our macroscopic evaluation, we report the location of vessels entering the PTN relative to the medial malleolus and the location of vessels entering the MPN and the LPN relative to the abductor hallucis fascia. These reference points are easily identifiable surgical landmarks. The use of these landmarks also ensured that we included the entirety of the tarsal tunnel, as described by Dellon and Mackinnon<sup>6</sup>. Macroscopically, we

found a mean of two vessels entering the PTN within 5 cm proximal to the medial malleolus, with a majority of specimens having a vessel entering at a mean of 4.0 cm proximal to the medial malleolus and a second vessel entering at a mean of 2 cm proximal to the medial malleolus. The MPN and the LPN most commonly had one vessel entering within 1 cm proximal to the abductor hallucis fascia and one or two vessels entering at a mean of approximately 0.5 cm distal and nearly 2 cm distal to the fascia.

From our microscopic analysis, we describe the location of the entering vessels relative to the bifurcation of the PTN into the MPN and the LPN. The most common arterial pattern identified was two vessels entering the PTN, one at a mean of 4 cm and one at a mean of 2 cm proximal to the bifurcation. Similarly, the MPN and the LPN were each supplied by a mean of two vessels. The majority (73%) of the specimens were supplied by a vessel that entered the nerve at the bifurcation and provided a branch to both the MPN and the LPN. In addition, the MPN and the LPN often had additional vessel branches that entered each nerve at approximately 2 cm and 4 cm distal to the bifurcation. We found that the vascular density of the MPN and the LPN was significantly greater than the density of vessels supplying the PTN.

This was an observational anatomic study that had several limitations. Descriptions of the arterial anatomy do not establish causality between the vascular anatomy and the development of clinical symptoms. Accurate characterization of the arterial anatomy may be compromised by preexisting macrovascular and microvascular conditions, such as atherosclerosis and diabetes, which were not controlled for in this study. This study quantified the number and location of vessels entering the nerves, but it did not assess the volume or flow of these vessels. As a result, this is not a description of the perfusion of the nerves. In addition, we elected not to evaluate the medial calcaneal branch of the PTN or the first branch of the LPN, both smaller branches with variable branching patterns.

The abundant blood supply to the PTN and its branches identified in this study is consistent with observations of other peripheral nerves. Given the importance of adequate vascular perfusion of peripheral nerves for nerve function, we speculate that this robust blood supply may have evolved as a protective role against nerve dysfunction. Although it is not known whether the abundant vasculature contributes to nerve compression causing tarsal tunnel syndrome by mechanical rather than ischemic means, this phenomenon may be a factor in a symptomatic patient. It is possible that conditions that increase the contents of the canal (edema, hemorrhage, or venous congestion of surrounding venous comitantes) or decrease its volume (hypertrophic retinaculum) may tip the balance such that the abundant vasculature, particularly venous branches, becomes a source of symptoms rather than a means to prevent them.

In general, avoidance of disruption of the blood supply to the nerve may be an important component of successful tarsal tunnel release. Injury to the vessels supplying the nerve may induce ischemia to the nerve postoperatively. While this study does not

definitively implicate changes in the blood supply to the nerve as a potential factor in surgical outcome, it is an important consideration in the treatment of tarsal tunnel syndrome given the current knowledge of nerve response to ischemia. ■

Mary Claire Manske, MD<sup>1,2</sup>  
Kathleen E. McKeon, MD<sup>2,3</sup>  
Jeremy J. McCormick, MD<sup>2</sup>  
Jeffrey E. Johnson, MD<sup>2</sup>  
Sandra E. Klein, MD<sup>2</sup>

<sup>1</sup>Department of Orthopaedic Sports Medicine,  
University of Washington, Seattle, Washington

<sup>2</sup>Department of Orthopaedic Surgery, Washington University  
School of Medicine, St. Louis, Missouri

<sup>3</sup>Andrews Sports Medicine and Orthopaedic Center,  
Birmingham, Alabama

E-mail address for M.C. Manske: mclairemanske@gmail.com  
E-mail address for K.E. McKeon: mckeon.kathleen@gmail.com  
E-mail address for J.J. McCormick: mccormickj@wustl.edu  
E-mail address for J.E. Johnson: johnsonje@wustl.edu  
E-mail address for S.E. Klein: kleins@wustl.edu

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