

Superficial Peroneal Nerve Transfer for the Treatment of Intractable Lower Extremity Pain: A Novel Technique

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Statement of Purpose

Intractable nerve pain is a common and often debilitating complication following lower extremity trauma, which can result in physical, psychological, social and economic complications.¹⁻³ Due to the complexity of these types of injuries, there is no consensus in regards to appropriate management. Nerve injuries, first defined by Seddon include: neuropraxia (Type I), axonotmesis (Type II) and neurotmesis (Type III).⁴ Traditional surgical intervention for Type II injuries (neuromas-in-continuity) includes excision, end-capping and subsequent burial into muscle (neuromyodesis) or bone (neuro-osteodesis).⁵⁻⁸ Despite these techniques, painful stump neuroma formation due to aberrant axon sprouting or pressure irritation can result.⁷ Recently, nerve transfers combined with nerve allografts, have demonstrated encouraging outcomes. Due to this "closed-loop" technique, aberrant axon sprouting or pressure irritation and subsequent stump neuroma formation is eliminated.⁹⁻¹²

The purpose of this study is to evaluate the efficacy of a novel nerve transfer technique for treatment of neuromas-in-continuity, of two peripheral nerves, performed proximal to the zone of initial traumatic injury. We present 37 patients who underwent SPN to DPN or SPN to sural nerve transfer with allograft and conduit repair for treatment of intractable neurogenic pain.

Nerve Injury	Traits	NCV Findings
Type I	Myelin Disruption	Normal Amplitudes
Type II	Axonal Degloving (Neuroma-in-continuity)	Decreased Amplitudes
Type III	End Neuroma	Absent amplitudes

Table 1: Seddon Nerve Injury Classification with trait and NCV description

Methodology & Procedures

Patient Selection
All patients experienced localized pain with percussion and generalized pain to the distribution of the SPN, DPN and/or sural nerves. Preoperative blocks with corresponding pain relief were used to verify/confirm nerve injuries. All patients were seen by a neurologist and EMG and NCV studies were performed. In all cases, there was over a 50% decrease in axon amplitudes as well as reduction in conduction velocities of two nerves.

Operative Technique
Surgery was performed with the patient under general anesthesia without a central or peripheral nerve block. No tourniquet or muscle relaxing agents were utilized as nerve stimulation was required to distinguish the sensory and motor components of the recipient nerve. The procedure was partially performed under surgical loupe magnification (3.5x-5x) for nerve dissection, as well as operative microscopy (6x-10x) for nerve reconstruction.

An incision was placed overlying the proximal course of the SPN. The nerve was identified and an external neurolysis was performed. The SPN was then transected. Dissection was continued until the DPN or sural nerve was identified, depending on which transfer was to be completed. After identification of the secondary nerve, subsequent external neurolysis and neurectomy were performed at the target site. Transection of the DPN was performed as far distal to the nerve's motor checkpoint as possible (noted with a nerve stimulator).

The allograft-coupler construct was then sutured to the free ends of the SPN and DPN or sural nerve at the 6:00 and 12:00 positions. Within the conduits, the native nerve was positioned approximately 2-3 mm from contact with the allograft (Figure 3).

All nerve transfer sites were augmented with bone marrow aspirate (proximal tibia donor site) and platelet rich plasma for autologous stem cell implantation. Each patient underwent primary skin closure and a semi-compressive sterile dressing, with an elastic bandage as the top cover.

Postoperative Course
Postoperatively, patients were admitted for observation. They were encouraged to perform ankle dorsiflexion and plantarflexion and knee ROM to prevent fibrosis at the surgical site. Physical therapy assisted with non-weight bearing gait training.

Postoperative pain management consisted of a PCA pump, which was discontinued after 24 hours. Following this, all narcotics were eliminated and a trimodal therapy approach including: gabapentin 100-300 mg TID, tramadol (50 mg BID) and tylenol (625 TID) was utilized. Weight bearing as tolerated occurred upon suture or staple removal. Patients transitioned to normal shoe gear as tolerated at 4-6 weeks postoperatively.

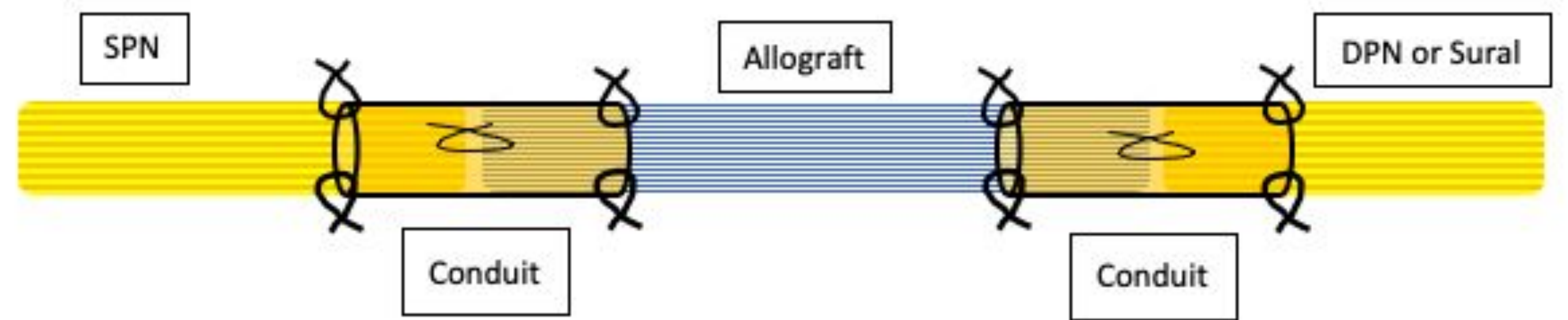


Figure 3: Allograft-coupled conduit construct for nerve transfer

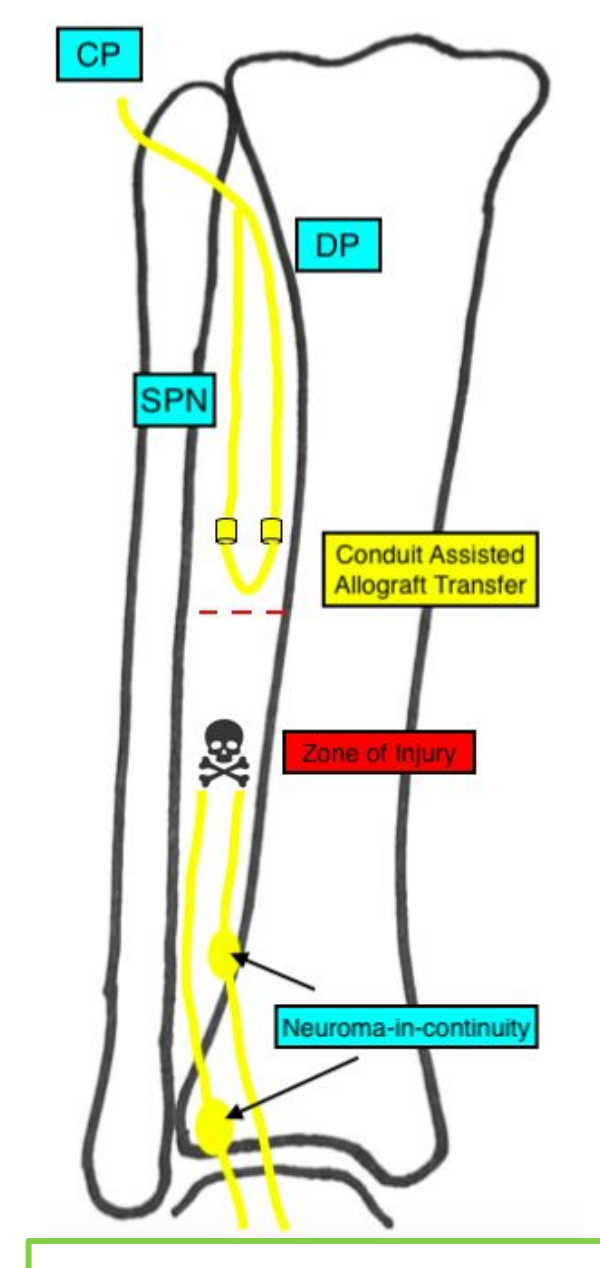


Figure 1: SPN to DPN transfer

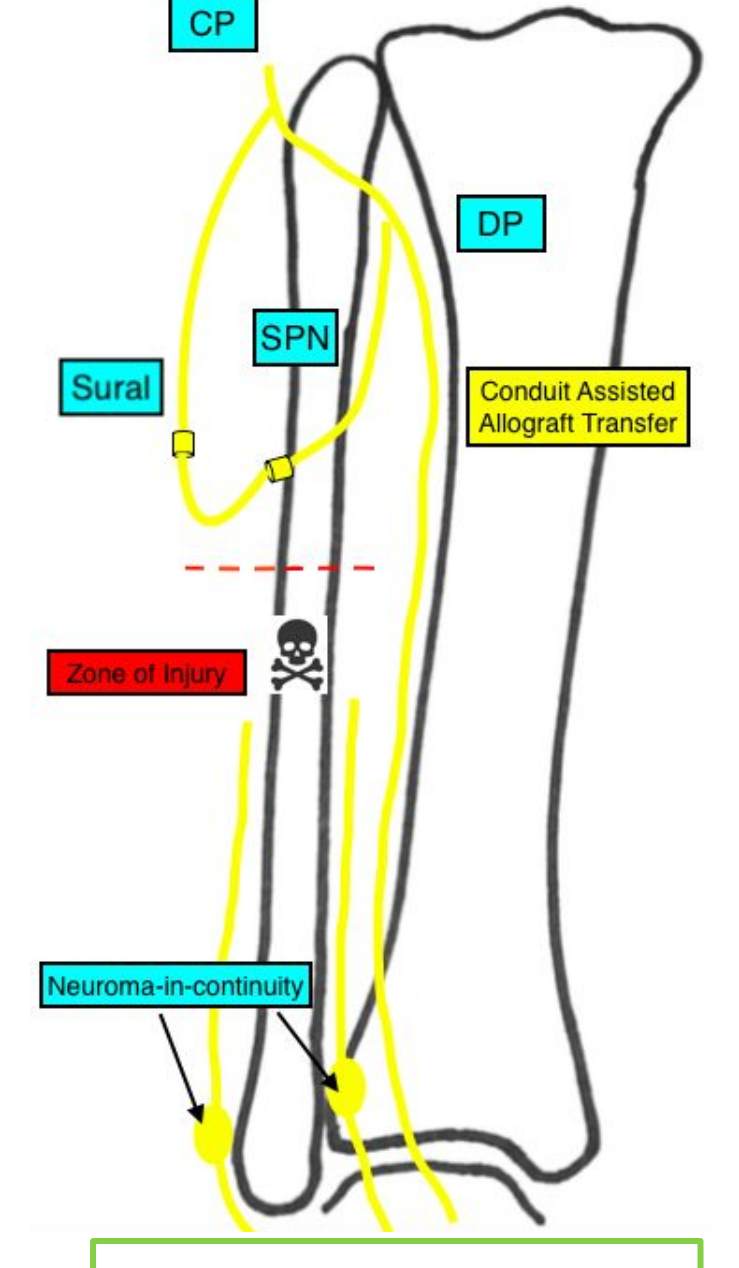


Figure 2: SPN to Sural nerve transfer

Results

Patient	Sex	Age (Years)	Neuroma Etiology	Preoperative VAS	Final Follow-up VAS	Follow-up time (Months)
1	M	62	Ankle fracture	9	2	32
2	F	41	Ankle sprain	9	3	27
3	M	48	Ankle sprain	9	1	15
4	F	73	5th metatarsal fracture	10	0	41
5	F	54	Ankle sprain	7	3	31
6	F	53	Calcaneal fracture	10	3	47
7	M	39	Ankle fracture	9	3	23
8	F	31	Contusion	8	1	14
9	F	36	Ankle sprain	9	1	19
10	M	61	3rd metatarsal fracture	8	3	20
11	F	44	Contusion	9	1	17
12	M	38	Ankle fracture	9	0	34
13	M	76	Contusion	9	0	4
14	F	41	Ankle sprain	7	1	44
15	F	30	Ankle fracture	7	0	48
16	F	69	Ankle fracture	10	2	15
17	F	38	Ankle fracture	9	0	17
18	M	73	Ankle sprain	10	1	43
19	M	45	Distal-tibial intra-articular fracture	10	1	50
20	M	52	Contusion	10	0	48

Patient	Sex	Age (Years)	Neuroma Etiology	Preoperative VAS	Final Follow-up VAS	Follow-up time (Months)
21	M	37	Calcaneal fracture	8	3	18
22	F	33	Ankle fracture	8	0	52
23	F	69	Ankle sprain	7	1	22
24	M	67	Ankle sprain	10	2	23
25	M	41	Calcaneal fracture	8	1	33
26	M	73	Contusion	10	2	25
27	M	50	Ankle fracture	10	2	37
28	M	65	Lis franc fracture	8	2	43
29	F	44	Ankle fracture	9	3	22
30	M	39	Contusion	10	2	15
31	M	55	Peroneal tendon rupture	9	2	23
32	F	38	Ankle sprain	9	2	18
33	M	52	Ankle sprain	10	2	18
34	M	43	5th metatarsal fracture	9	1	48
35	F	33	Calcaneal fracture	8	1	18
36	F	34	Calcaneal fracture	8	0	57
37	F	63	Ankle fracture	10	0	54

Average Age = 49.73 Years
Average Preoperative VAS = 8.89
Average Final Follow-up VAS = 1.41
Average Follow-Up Time = 30.14 Months

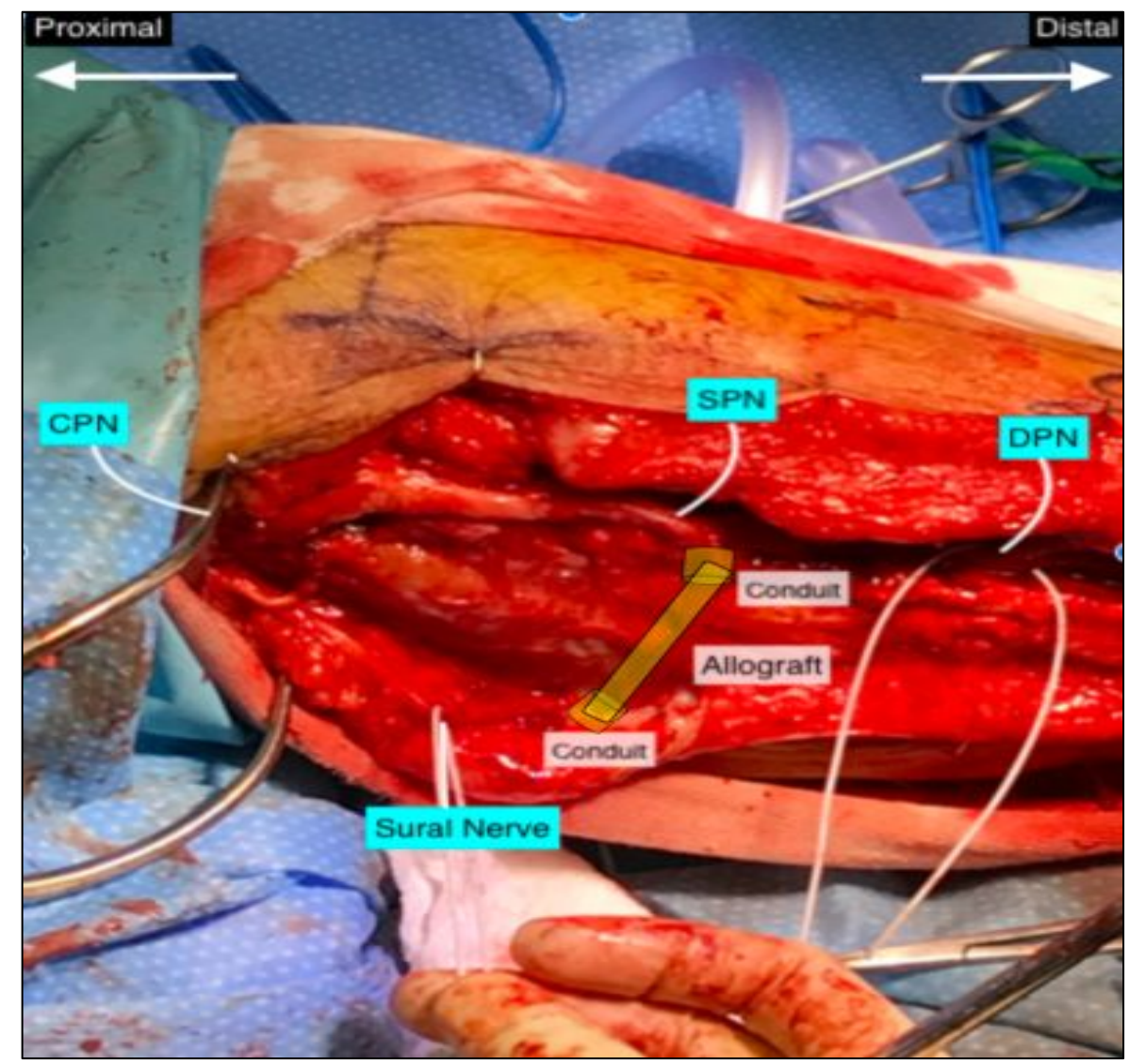


Figure 4: Intraoperative SPN to Sural nerve transfer (proximal tibia)

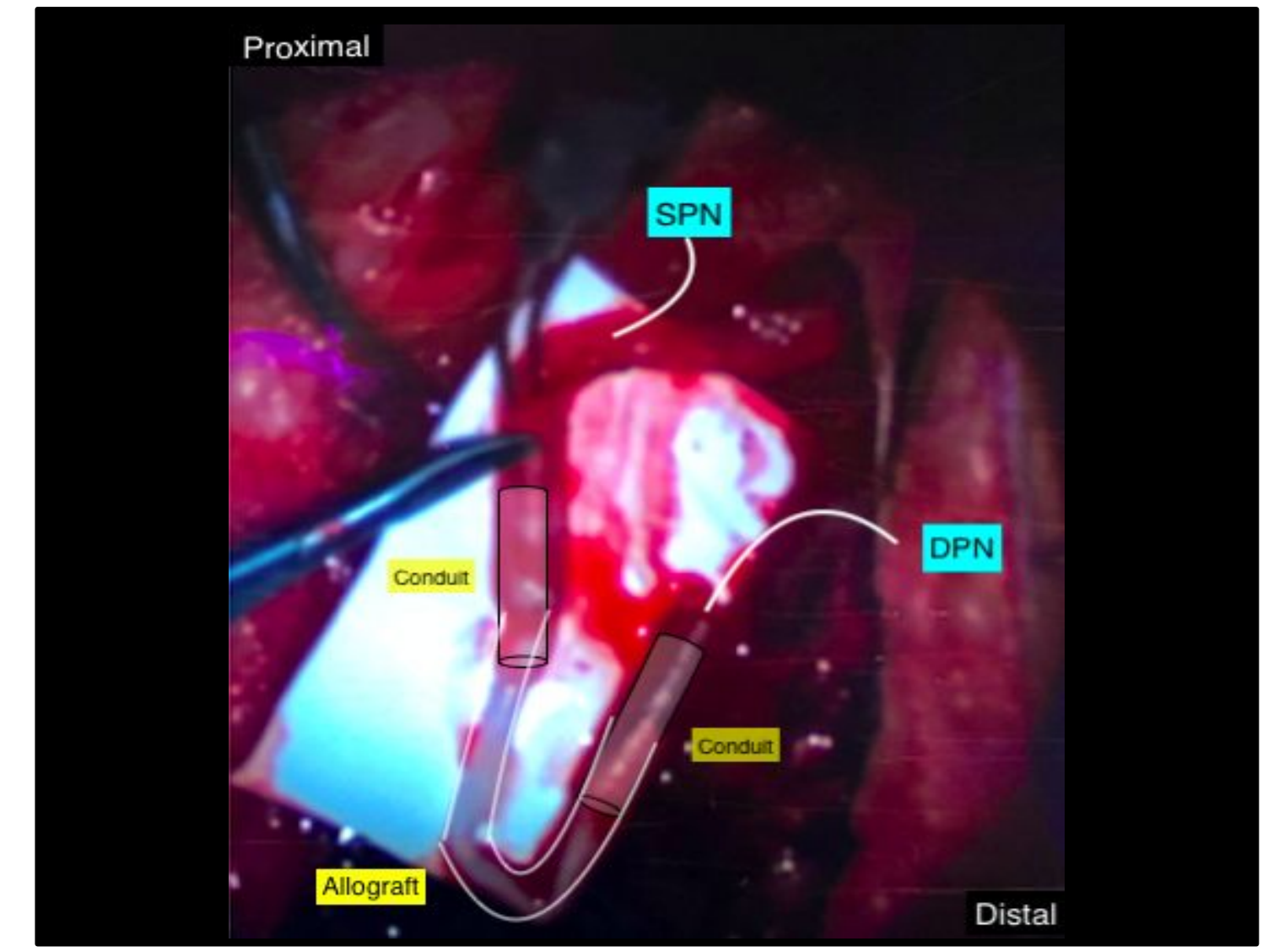


Figure 5: Intraoperative SPN to DPN nerve transfer under microscopy

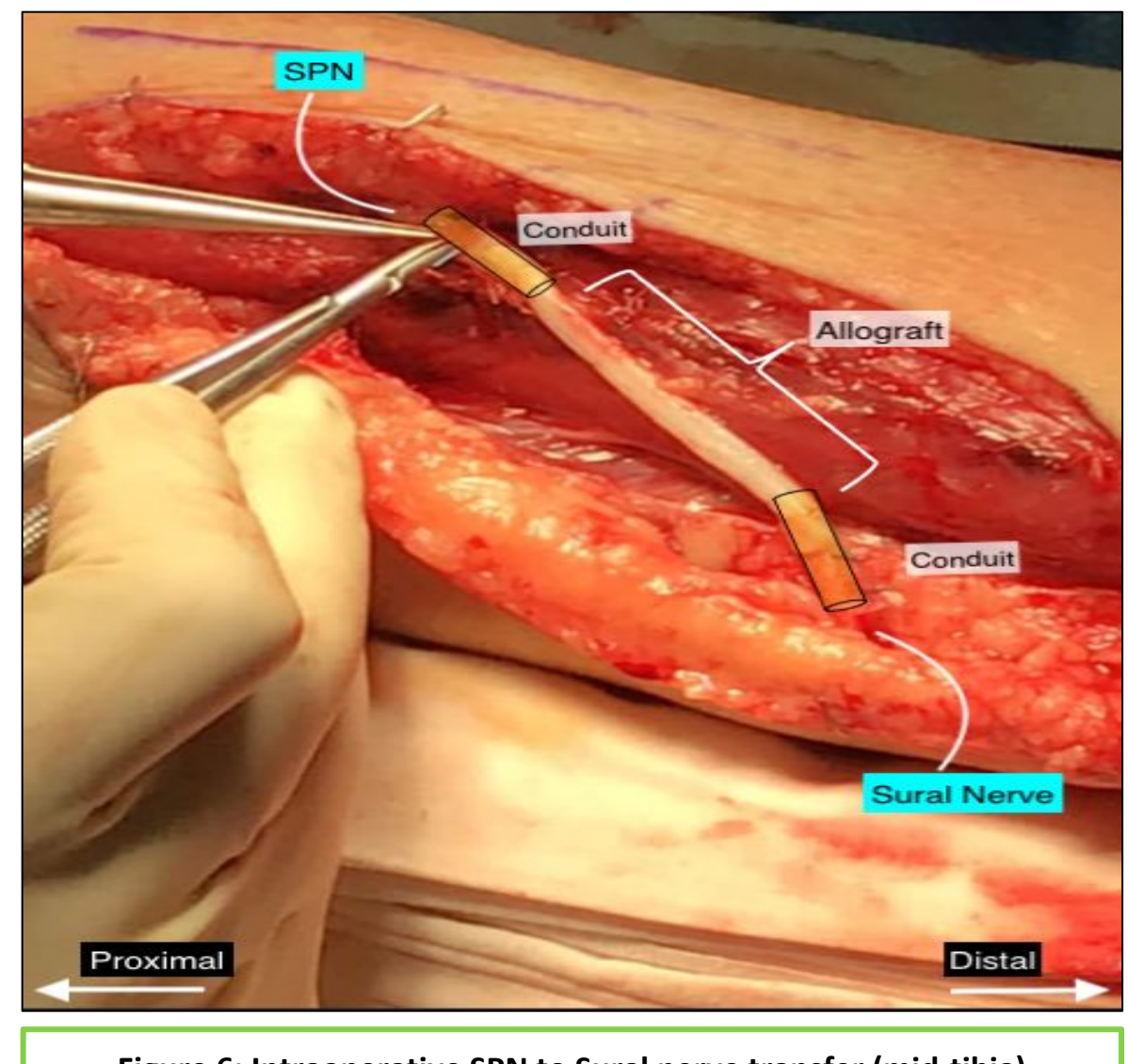


Figure 6: Intraoperative SPN to Sural nerve transfer (mid-tibia)

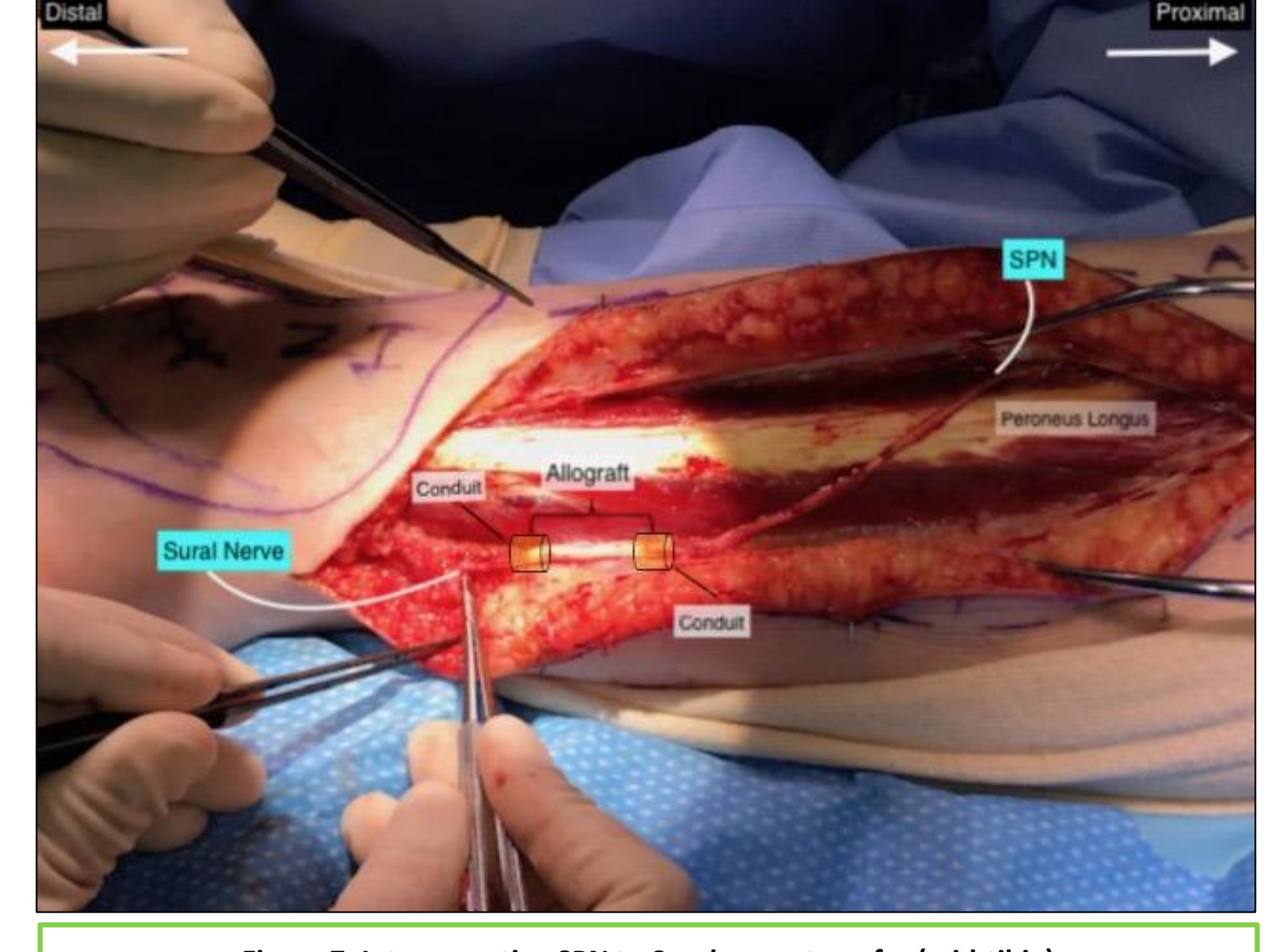


Figure 7: Intraoperative SPN to Sural nerve transfer (mid-tibia)

Analysis & Discussion

In this study, all injuries were Seddon Type II (neuromas-in-continuity) affecting two peripheral nerves of the lower extremity (SPN and DPN or SPN and sural). With this approach, the nerve transfer procedure was performed proximal to the zone of injury to avoid extensive fibrosis and scar tissue that was common from previous trauma. Additionally, this allowed neuronal tissue to glide without tension in a deeper plane of cushioning muscles. In doing so, both neuromas-in-continuity of the affected peripheral nerves were bypassed and never actually identified, which is different than previously described techniques.⁶⁻¹² Creation of this nerve loop construct proximal to the neuroma locations allowed for neuronal death proximally within the allograft and distally via wallerian degeneration, thus eliminating noxious stimulation.

In conclusion, nerve allograft conduit assisted transfer of SPN to DPN or SPN to sural nerve, proximal the zone of initial injury, has been shown to effectively treat intractable nerve pain following lower extremity trauma. This technique shows promise for management of neuromas-in-continuity resulting in predictable, significant and long-lasting pain relief.

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Declaration of Conflicting Interests:
Dr. Rodriguez-Collazo is a consultant for Orthofix, Integra, Ito Biologics and Axogen. The authors declare no potential conflicts of interest with respect to the research and/or authorship of this article.