

# The Agonist-Antagonist Myoneural Interface



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## KEYWORDS

• Agonist-antagonist myoneural interface • Amputation • Proprioception • Prostheses

## KEY POINTS

- The agonist-antagonist myoneural interface (AMI) comprises innervated muscles that are biomechanically linked to recapitulate normal muscle-tendon agonist-antagonist dynamics, and a neural control architecture that electronically interfaces the linked muscle tendons to a powered external prosthesis.
- When incorporated into a limb amputation procedure, AMIs have the potential to augment volitional control of prostheses, preserve musculotendinous proprioception, and prevent residual limb atrophy.
- AMIs may be constructed from natively innervated or regenerative muscle units.
- To date, AMI construction has been applied to elective lower and upper extremity amputations as well as to lower extremity residual limb revision procedures.

## INTRODUCTION

Profound changes are occurring in the realm of limb amputation. Once regarded as a form of surgical failure, amputation increasingly is being reframed as a reconstructive procedure—one with far more ambitious goals than those espoused in the past.<sup>1</sup> Whereas the prior aim of amputation largely was limited to providing a stable, padded residuum, current strategies have expanded expectations to include freedom from pain, high-fidelity control, neurosensory interfacing, and restoration of proprioceptive and cutaneous afferents. Fortunately, novel surgical approaches have been introduced over the past several years to meet these goals—among them, the agonist-antagonist myoneural interface (AMI). The AMI is a surgical construct and neuroprosthetic interfacing strategy designed to preserve or restore limb and joint proprioception, augment volitional control of adapted prostheses, and prevent or reverse limb atrophy. This article presents the authors' experience to date with the

development and implementation of the AMI in the setting of extremity amputation.

## CONTEXT

Proprioception is the perception or awareness of the relative spatial positioning of one's body parts and the amount of force exerted on the environment.<sup>2</sup> Sometimes referred to as “the sixth sense,” proprioception is critical to dexterity, joint stability, and mobile adaptation.<sup>3</sup> Although proprioceptive sensation has been ascribed to a symphony of mediators in human beings, the primary drivers appear to be muscle spindle fibers and Golgi tendon organs intrinsic to normal muscle-tendon architecture.<sup>4</sup> It is the stimulation of these structures, with the activation of dynamic agonist-antagonist muscle-tendon pairs in the intact body, that primarily establishes a neural loop between the peripheral and central nervous systems that, in turn, forms the basis for natural sensations of joint movement and load.<sup>5</sup>

The standard approach to amputation, however, disrupts dynamic agonist-antagonist muscle

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relationships, resulting in isometric contraction of effector muscles with attempted phantom joint movement without compensatory stretch of their antagonist pairs. The resulting discordant neural milieu often produces aberrant phantom limb sensation that may contribute to the development of phantom pain.

The potential value of preserving proprioception in the setting of limb amputation initially was recognized through the development of the technique of cineplasty. First credited to Vanghetti<sup>6</sup> in 1898, the notion of cineplasty in its original form involved the construction of muscle loops or tubes covered with skin grafts to control adapted extremity prostheses. This loop design was further adapted by Sauerbruch<sup>7</sup> in 1916 to incorporate the use of “both protagonist and antagonist muscles to give physiologically correct control.” Cineplasty achieved a moderate degree of popularity in the United States in the early 1950s, but its application largely was limited to the military population and never achieved widespread adoption due to issues with skin irritation, infection and muscle fatigue. As recently as 1998, proponents of cineplasty forecast the possibility of linking an adapted prosthetic device to a “cineplastized muscle’s own proprioceptive receptors [in order to] provide feedback information about the prosthesis’ state”<sup>8</sup>; however, an effective means of achieving a true, dynamic agonist-antagonist muscle-tendon unit coupled to a biomechanical device had yet to be described.

## CONCEPT AND FOUNDATIONAL SCIENCE

The AMI represents an alternative vision to the loop cineplastic approach of agonist-antagonist coupling that exploits modern surgical techniques and neuroprosthetic interfacing to achieve enhanced prosthetic controllability and proprioceptive percepts. In contrast to cineplasty, the AMI approach does not seek to physically attach muscle-tendon units to an external prosthesis in order to complete the agonist-antagonist neuro-mechanical loop; rather, the AMI approach connects agonist-antagonist muscle-tendon units within the biological residuum and then bidirectionally communicates and interprets these tissue dynamics to an external prosthesis using sensors, stimulators and a control system. Because there does not exist direct physical loading between an external prosthesis and an agonist-antagonist muscle pair, the AMI approach mitigates the cineplastic issues of skin irritation, infection, and muscle fatigue.

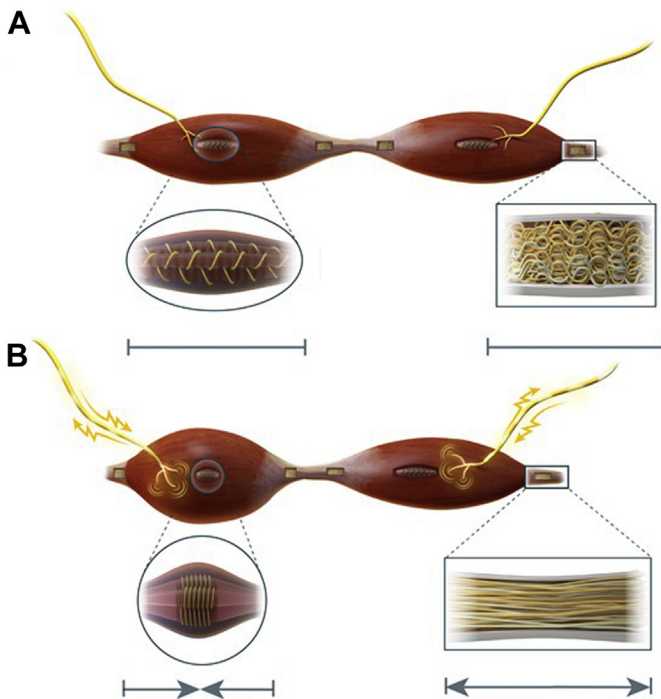
The core notion of the AMI tissue construct is that a primary effector muscle (the agonist) is

biomechanically linked in series to a counter-muscle (the antagonist) within the amputated residuum, such that contraction of the agonist results in simultaneous proportional stretch of the antagonist. Triggering of the agonist and/or antagonist may occur either through volitional, neural activation or via commanded means, including functional electrical stimulation (FES) from prosthetic processors. The stress and strain resulting from this dynamic relationship cause simultaneous stretch of the muscle spindle fibers and Golgi tendon organs within the agonist and antagonist muscle tendons, resulting in activation of both afferent and efferent neural pathways that recapitulate those of a natural biological system (Fig. 1). The specificity of agonist and antagonist muscle function imbues an individual AMI with the ability to serve as a proxy for a single lost or compromised joint; therefore, multiple AMIs have the potential to control multiple degrees of freedom within an external prosthesis.

When healthy, innervated, vascularized agonist-antagonist muscles with discrete, innate functionality are available, they may be redirected and biomechanically linked to enable the construction of native AMIs.<sup>9,10</sup> When such tissues are not available, however, they may be constructed de novo via techniques, including targeted muscle reinnervation (TMR) and/or regenerative peripheral nerve interface (RPNI) creation, and subsequently coapted via a passive material, such as tendon, thereby enabling the construction of regenerative AMIs (Fig. 2).

Preclinical studies of native AMI architecture provided the first proof of concept evidence regarding the validity of this reconstructive concept. Initial murine investigations demonstrated muscle stretch and integrated electroneurography (ENG) profiles for native AMIs to be similar to those of intact, uninjured agonist-antagonist hindlimb muscle dyads. Furthermore, the preservation of native neural loops was evidenced via the successful elicitation of antagonist muscle spindle fiber activation with agonist contraction.<sup>11</sup> Subsequent studies of native AMI construction in both below-knee amputation (BKA) and above-knee amputation (AKA) scenarios in larger caprine models demonstrated intact coupled motion of ankle and knee constructs, respectively, via both artificial muscle stimulation and direct mechanical manipulation.<sup>12</sup>

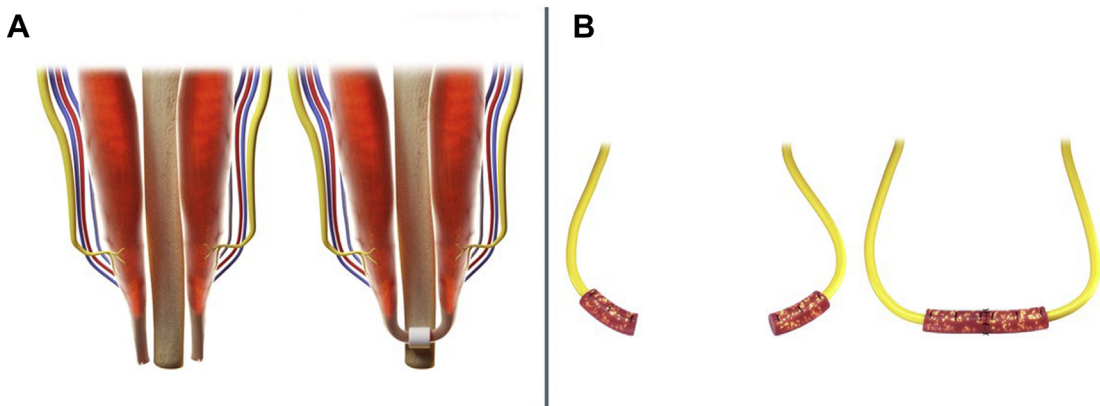
Validation of the regenerative approach to AMI construction was undertaken in parallel and demonstrated similarly promising results. Investigations in which single-stage AMI construction was performed via linked RPNI creation in a murine model evidenced coupled motion and



**Fig. 1.** AMI conceptual illustration. (A) Coapted agonist and antagonist muscle pair at rest, with quiescent muscle spindle fibers (*left*) and Golgi tendon organ (*right*). (B) Musculotendinous afferents along native neural pathways are produced when contraction of the agonist stretches the linked antagonist (or vice versa), causing activation of muscle spindle fibers and Golgi tendon organs.

physiologically relevant strains in all regenerative AMIs as well as the generation of graded electromyography (EMG) and ENG signals with construct stimulation.<sup>13</sup> Furthermore, follow-up murine studies in which 2-stage AMI construction was undertaken via RPNI creation during a first-stage surgery followed by AMI construct creation during a second-stage surgery (as

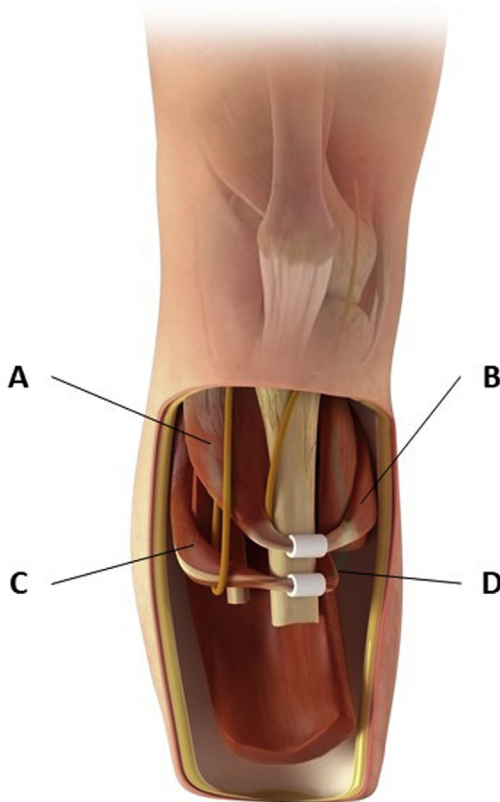
would be required in a residual limb revision scenario) demonstrated similarly intact construct motion, afferent and efferent signaling, and force production. An additional serendipitous finding of this investigation was significant reversal of single RPNI muscle atrophy following AMI agonist-antagonist coaptation during the second-stage surgery.<sup>14</sup>



**Fig. 2.** Variations on AMI construction. (A) Native AMI design in which innervated, vascularized muscles with intended function are present. In this scenario, the distal ends of constituent agonist and antagonist muscles (*left*) may be simply redirected and coapted (*right*). (B) Regenerative AMI design, in which agonist and/or antagonist components must be created de novo. In this scenario, initial construction of innervated, vascularized muscle units first must be performed via TMR and/or RPNI techniques (*left*). Subsequent coaptation of regenerative units (*right*) may be performed as a second stage once innervation has been confirmed.

### CLINICAL IMPLEMENTATION AND SURGICAL TECHNIQUES

The first human implementation of AMI construction was performed in 2016 in a modified BKA procedure that subsequently has been named the Ewing amputation. In this operation, 2 native AMIs were constructed; the first, composed of the tibialis anterior and lateral gastrocnemius, served as a tibiotalar joint emulator, whereas the second, composed of the peroneus longus and tibialis posterior, served as a subtalar joint emulator. Construction of each AMI was aided via the utilization of the patient's tarsal tunnels as spare parts, which were secured to anterior tibia using suture anchors and provided lubricious gliding mechanisms to which the constituent muscles were coapted (Fig. 3). Tensioning of each AMI construct was set to approximate normal



**Fig. 3.** Ewing amputation (AMI BKA) schematic. The upper AMI serves as the tibiotalar joint emulator and is composed of the (A) tibialis anterior and (B) lateral gastrocnemius, which is redirected around the posterior aspect of the limb to enter the AMI medially. The lower AMI serves as the subtalar joint emulator and is composed of the (C) peroneus longus and (D) tibialis posterior.

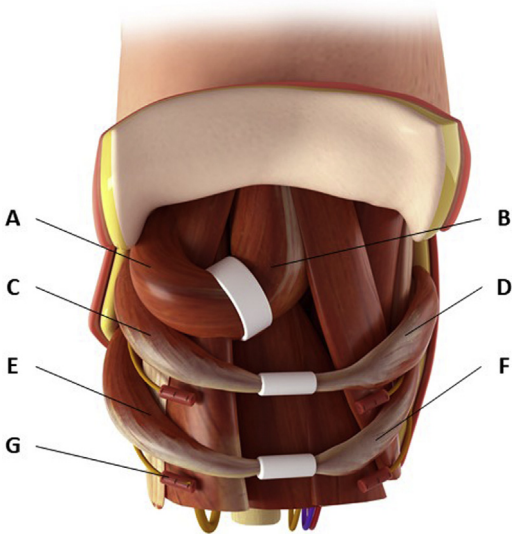
physiologic agonist-antagonist relationships, and radiopaque tantalum beads were inserted into each construct in order to facilitate noninvasive visualization of coapted motion in the postoperative period. Closure was performed via standard soleus myoplasty and approximation of soft tissue envelope edges.<sup>15</sup>

Since 2016, the Ewing amputation subsequently has been performed on 25 lower extremities in 22 patients under Partners institutional review board (IRB) Protocol 2014p001379. The original surgical technique has been modified slightly to include creation of a tibial periosteal overlay to protect the tarsal tunnel coaptation points and subfascial dissection of the posterior soft tissue envelope in order to augment skin perfusion. The most notable alteration, however, has been the incorporation of TMR and/or RPNI construction for neuroma prophylaxis of sensory nerve termini at the time of amputation; currently, the authors' practice is to ablate 5 distal nerves with significant cutaneous territories (tibial, superficial peroneal, deep peroneal, medial sural, and saphenous) via these techniques at the same time as performing AMI construction.

The authors' experience with the Ewing amputation has also informed the development of a modified approach to AKA, first performed in 2018 under Partners IRB Protocol 2014p001379. In this procedure, native AMIs are constructed for the tibiotalar and subtalar joints using the same muscle pairs as in the Ewing amputation; these are complemented, however, by the additional construction of a knee joint AMI composed of the rectus femoris and lateral head of the biceps femoris.<sup>16</sup> The tibiotalar and subtalar AMI muscles are recruited via isolation and mobilization of their discrete neurovascular pedicles—in essence, requiring the establishment of 4 neurovascular muscle island flaps that subsequently are configured circumferentially around the distal thigh musculature (Fig. 4). Tarsal tunnel recruitment is utilized for the 2 lower AMIs, as in the Ewing amputation, but provision of a gliding canal for the knee AMI is accomplished via utilization of ankle retinaculum or discarded muscle fascia.

AMI construction also has been incorporated into the design of novel approaches to modified upper extremity amputation at both the below-elbow amputation and above-elbow amputation (AEA) level under Partners IRB Protocol 2018p001893. In the former, AMI emulators for thumb interphalangeal joint flexion/extension, composite digital flexion/extension, and wrist flexion/extension are constructed via pairing of the flexor pollicis longus/extensor pollicis longus, flexor digitorum superficialis/extensor digitorum



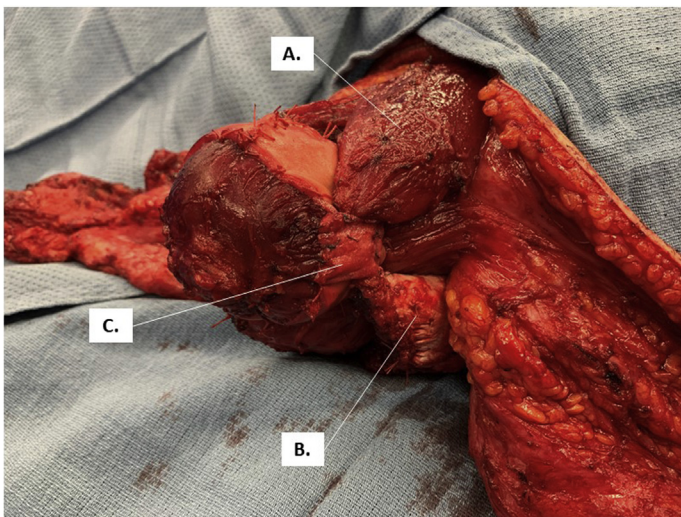


**Fig. 4.** AKA AMI schematic. The uppermost AMI serves as the knee joint emulator and is composed of the (A) lateral head of the biceps femoris and (B) rectus femoris. The middle AMI serves as the tibiotaral joint emulator and is composed of the (C) tibialis anterior and (D) lateral gastrocnemius. The lowermost AMI serves as the subtalar joint emulator and is composed of the (E) peroneus longus and (F) tibialis posterior. (G) Distal sensory nerve RPNI construct.

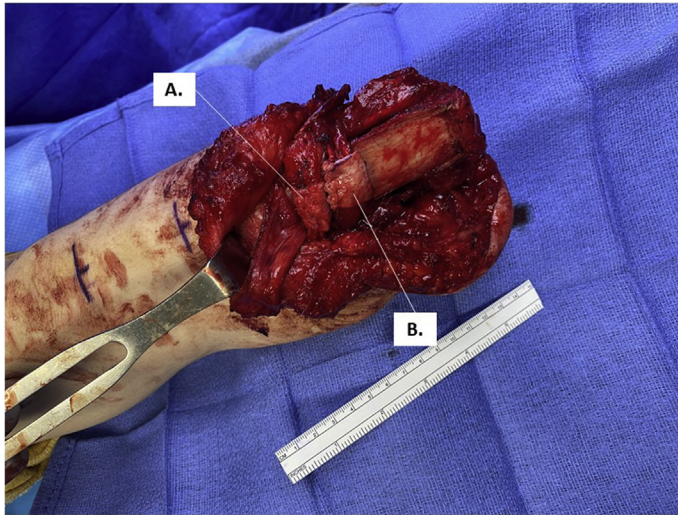
communis, and flexor carpi radialis/extensor carpi radialis brevis, respectively; in lieu of tarsal tunnels, the extensor retinaculum is utilized as a pulley stabilization platform over the distal end of the bony residuum.<sup>17</sup> In the single AEA procedure performed to date, a native AMI emulator for elbow flexion/extension was constructed utilizing pairing

of the long head of the biceps and long head of the triceps. A regenerative AMI emulator for wrist flexion/extension was constructed using the short head of the biceps and lateral head of the triceps, because the patient had previously undergone TMR of the median and radial nerves to these sites. Pulley sites were created de novo in this case utilizing acellular dermal matrix (ADM) affixed to the epimysium of the distal residual limb myoplasty<sup>18</sup> (Fig. 5).

Most recently, AMI construction has been applied to the revision of previously amputated lower extremities at both the BKA and AKA level under Partners IRB Protocols 2017p000685 and 2019p001681. In the setting of BKA revision, sufficient musculature typically remains in the residual limb to enable the creation of native AMIs via techniques similar to those employed in a Ewing amputation; the notable exception, however, is that tarsal tunnels are not available for pulley construction, requiring substitution of ADM canals<sup>19</sup> (Fig. 6). AKA level revision obligatorily requires a combination of native (knee) and regenerative (tibiotaral and subtalar) AMI construction techniques due to the loss of predictable sciatic nerve organization other than gross division of the common peroneal and tibial nerve branches. In this scenario, the authors have pursued a 2-stage approach consisting of initial fascicular splitting of the sciatic nerve and multiple RPNI construction, followed by EMG interrogation of RPNIs approximately 3 months to 6 months later and subsequent surgical coupling of the functionally specific regenerative units into AMI constructs as a follow-up procedure. This regenerative revision procedure has been coupled with simultaneous



**Fig. 5.** AEA AMI clinical photograph. Native elbow joint AMI composed of the (A) long head of biceps and (B) lateral head of triceps. (C) ADM canal and distal platform to stabilize AMI pivot point.



**Fig. 6.** AMI construction in BKA revision scenario. (A) Tibiotalar joint AMI. (B) Subtalar joint AMI.

placement of an experimental transfemoral osseointegrated implant (eOPRA device, Integrum, San Francisco, California) in order to maximize downstream functional potential and volitional control<sup>20</sup> (Fig. 7).

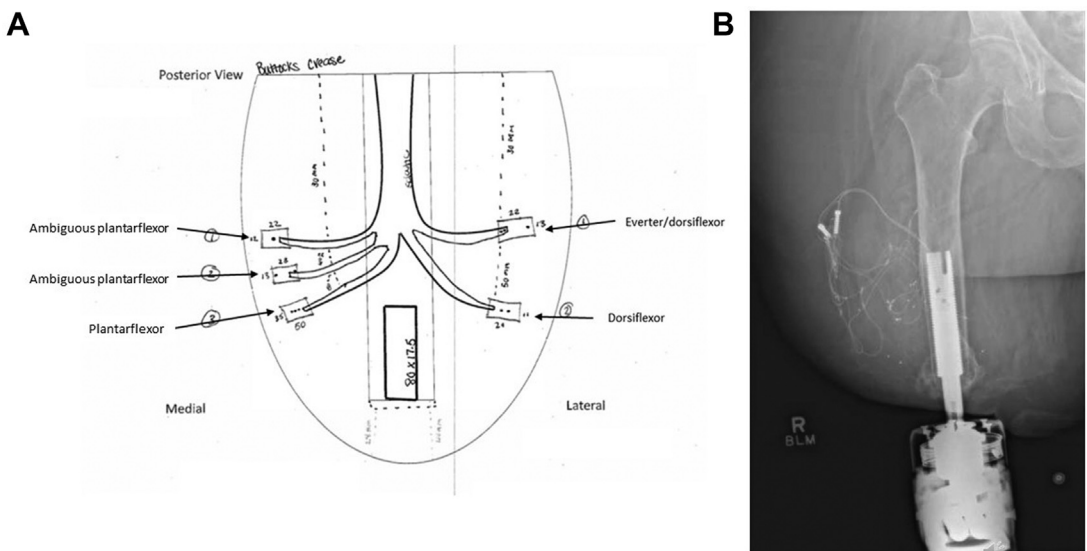
## OUTCOMES

To date, a majority of amputation procedures incorporating AMIs have been performed in elective BKA or AKA scenarios; as a result, the preponderance of outcomes data collected pertains to

these procedures. These outcomes are summarized.

## Clinical Parameters

A total of 25 modified BKA procedures and 4 modified AKA procedures have been performed to date in a total of 26 patients. Average operative times for modified BKA and AKA procedures have been 340 minutes  $\pm$  63 minutes and 562 minutes  $\pm$  120 minutes, respectively. Average length of stay for BKA patients has been



**Fig. 7.** AMI construction in AKA revision scenario, including placement of eOPRA device. (A) Schematic diagram depicting construction of multiple RPNIs from sciatic nerve and ultimate reinnervated function determined via EMG. Appropriate agonist and antagonist pairs subsequently were coapted in a second-stage procedure. (B) Plain radiograph depicting eOPRA device and implanted electrode system connected to AMI tissue constructs.

5.4 days  $\pm$  1.6 days, whereas that of AKA patients has been 14.5 days  $\pm$  10.1 days. The clinical parameters pertaining to both categories of patients are summarized in [Table 1](#).<sup>21</sup>

### Complications

Among the modified BKA population, 3 (12%) operative limbs have demonstrated minor wound healing issues, whereas 4 (16%) have demonstrated major wound healing issues requiring operative intervention. Three (12%) BKA limbs have demonstrated minor infections requiring postoperative oral antibiotic therapy. Eight (32%) BKA limbs have required operative revision for soft tissue modification, osteophyte excision, neuroma ablation, and/or suture anchor removal. Among the modified AKA population, 2 patients (50%) have demonstrated significant necrosis of their soft tissue envelopes requiring operative débridement and revision closure with split-thickness skin grafts. No additional complications in either patient cohort have been reported to date.<sup>21</sup>

### Agonist-Antagonist Myoneural Interface Function

Subjective reports of the degree of AMI activation and excursion by both patients and providers have been ranked uniformly as good (46%) or very good (54%) in serial clinical examinations for up to

3 years postoperatively. Objective interrogation of AMI constructs via serial ultrasound studies have demonstrated persistent preservation of coupled motion in all modified BKA and AKA patients over time, with a mean excursion of 4.3 mm  $\pm$  1.2 mm across muscle coaptation sites that has remained stable for up to 33 months following amputation.<sup>21</sup> Furthermore, assessments of antagonist muscle fascicle strain with agonist muscle contraction have exhibited statistically significant differences between modified BKA patients and standard BKA controls<sup>22</sup> as well as between modified AKA patients and standard AKA controls.<sup>16</sup> Fascicle strain assessments between modified BKA and modified AKA patients were noted to be comparable, suggesting a relative equivalency of excursion dynamics across both experimental groups.

### Limb Morphology

As referenced in [Table 1](#), serial circumferential measurements obtained over time have demonstrated 94.3%  $\pm$  4.8% and 100.6%  $\pm$  5.4% preservation of residual limb volume in modified BKA and modified AKA patients, respectively, that has persisted for up to 36 months postoperatively. The etiology of this preserved limb bulk most likely is due to unconscious activation of constituent AMI musculature with regular ambulation, resulting in muscle volume maintenance and, in some cases, hypertrophy. This relative stability in limb

**Table 1**  
Agonist-antagonist myoneural interface lower extremity amputation study subject clinical parameters

Measure	Agonist-Antagonist Myoneural Interface Below-Knee Amputations		Agonist-Antagonist Myoneural Interface Above-Knee Amputations	
	Total/Mean	SD	Total/Mean	SD
Total patients	22	—	4	—
Total limbs	25	—	4	—
Age (y)	41.3	12.5	40.6	19
Gender (% M/F)	59/41	—	25/75	—
BMI (kg/m <sup>2</sup> )	27.7	5.3	23.1	8.2
Operative time (min)	340	63	562	120
Fluids (mL)	1536	603	2125	299
EBL (mL)	86	74	213	193
UOP (mL)	672	330	1078	454
LOS (d)	5.4	1.6	14.5	10.1
Drain removal (d)	12.3	3.4	11.5	6.4
Prosthesis fitting (d)	67.8	33.1	74.3	28.3
Volume preservation (%)	94.3	4.8	100.6	5.4

*Abbreviations:* BMI, body mass index; EBL, estimated blood loss; F, female; LOS, length of stay; M, male; UOP, urine output.



morphology has translated to subjective patient reports of infrequent requirements for socket refitting once postoperative swelling has resolved, with a majority of patients having unchanged interfaces for at least 12 months to 18 months.<sup>23</sup>

### **Pain**

All patients who have undergone modified BKA or AKA procedures have demonstrated a complete wean from narcotic medications in the postoperative period, with an average wean time of 58 days and a range of 4 days to 189 days. The upper end of this range is due at least in part to a subset of intervention patients who were on chronic opioid medications for many years prior to surgery and thus required extended wean protocols due to concerns about potential withdrawal; 38% of patients who have undergone these procedures report residual limb pain; of these, 78% report such pain to be rare or occasional and are managed via over-the-counter medications or nonpharmacologically. Two patients who did not undergo initial sensory nerve ablation at the time of their amputation subsequently required TMR procedures to successfully address neuropathic residual limb pain; 29% of intervention patients report some degree of phantom limb pain, but all such patients report this pain to be rare.<sup>21</sup>

### **Rehabilitation**

All modified BKA and AKA patients have progressed to standard prosthesis fitting following complete healing of their residual limbs at a mean interval of 67.8 days  $\pm$  33.1 days and 74.3 days  $\pm$  28.3 days, respectively (see [Table 1](#)), and universally report utilization of their prostheses for 10 hours to 12 hours per day. Furthermore, they have been assessed via a battery of validated rehabilitation metrics at standard time points throughout their clinical courses. Comparisons of functional status performed in the preoperative versus post-prosthesis periods have demonstrated improvements in the 10-m walking test (20%  $\pm$  40%), timed up and go test (12%  $\pm$  44%) figure-of-8 test (23%  $\pm$  41%), side step (58%  $\pm$  57%), functional reach test (16%  $\pm$  70%), 4-stair climb test (14%  $\pm$  49%), and 6-minute walking test (42%  $\pm$  93%).<sup>24</sup>

### **Patient-reported Outcomes Measurements**

All modified BKA and AKA patients have been administered 3 validated patient-reported outcomes measurement instruments in the preoperative setting and at set intervals postoperatively (every 3 months for the first year and annually thereafter)—the EuroQol (EQ)-5D, the Lower

Extremity Functional Scale (LEFS), and the Patient Reported Outcomes Measurement Information System (PROMIS)-57. Reporting at a mean postoperative interval of 15.9 months  $\pm$  5.3 months, respondents have demonstrated uniform improvements in all measured categories relative to baseline. Specifically, intervention patients have demonstrated improvements in the EQ-5D health scale (129%  $\pm$  229%) and LEFS overall score (170%  $\pm$  185%) as well as PROMIS-57 physical function (144%  $\pm$  141%), anxiety (–11%  $\pm$  44%), depression (–4%  $\pm$  62%), fatigue (–41%  $\pm$  27%), sleep disturbance (–13%  $\pm$  47%), social roles and activities (150%  $\pm$  136%), pain interference (–55%  $\pm$  33%), and pain intensity (–57%  $\pm$  50%).<sup>21</sup>

### **Sensory Feedback**

Among modified BKA and AKA patients, 85% have reported significant phantom limb sensation following their procedures. A majority of patients report this phantom sensation to be anatomically correct in terms of position and proportion of their phantom limb, with specific territories of the phantom particularly present ([Fig. 8](#)). These patients typically state that their phantom limb sensation is augmented by activation of their AMI constructs, with many claiming an ability to “summon” or “telescope” their phantom limb with AMI motion. Furthermore, comparative studies have demonstrated significantly higher degrees of phantom limb sensation in AMI BKA patients versus standard BKA patients.<sup>22</sup>

### **Central Neuroplasticity**

Functional neuroimaging of AMI BKA patients, standard BKA patients and patients without amputations has demonstrated that AMI BKA patients manifest functional activation of Brodmann area (BA) 3a, which traditionally is considered to be the nexus for proprioception in the central nervous system, in a manner similar to those with intact limbs; this is in contrast to patients with standard BKAs, who demonstrate diminished activation of this cortical region. Furthermore, AMI BKA patients have demonstrated enhanced functional sensorimotor connectivity and phantom sensation compared with standard BKA patients, evidenced via increased activation of areas in the medial frontal cortex, including BA3a, BA3b, BA4a, and BA4p. These results suggest that AMI construction has a measurable neuroplastic effect in reengaging sensory feedback and motor imagery functionality.<sup>25</sup>





**Fig. 8.** Phantom limb sensation mapping in AMI BKA subject. Hatched areas depict regions of maximal phantom limb perception, as indicated by patient, in phantom dorsal foot, posterior leg, plantar foot and medial foot (left to right).

### ***Linkage with Adapted Protheses***

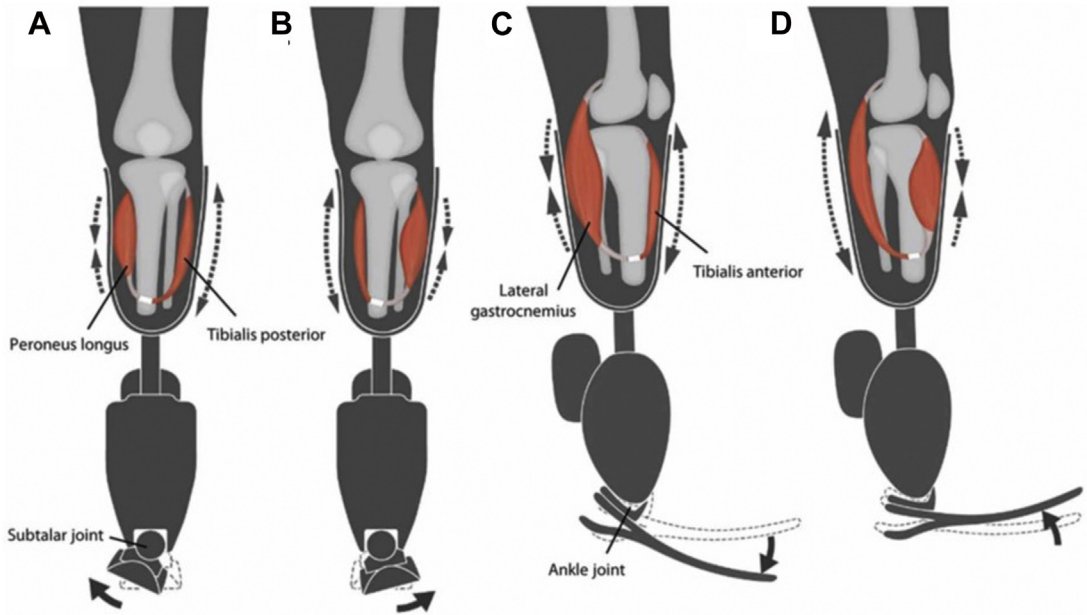
Patients who undergo modified amputations with AMI construction likely will achieve their maximal functional potential when their residual limb is coapted to an adapted prosthesis capable of interfacing with their unique surgical architecture. Toward this end, pilot investigations have been completed in which an experimental prosthetic leg with powered artificial tibiotalar and subtalar joints has been connected to the residual limb of a BKA AMI patient via both surface and percutaneous needle electrodes (Fig. 9). Compared with patients who underwent standard BKAs, the BKA AMI patient demonstrated markedly improved independent volitional control of joint position and impedance when linked to the experimental device as well as intuitive restoration of reflexive behaviors during various stages of the stair ascent and descent gait cycle. Furthermore, experiments in which FES was utilized in order to provide afferent proprioceptive feedback of joint torque resulted in markedly improved performance on torque control tasks in the BKA AMI patient that were not witnessed in standard BKA controls. Lastly, the BKA AMI patient demonstrated significant evidence of augmented embodiment with the adapted prosthesis, manifest through a variety of small behaviors including unconscious fidgeting of his mechanical foot while engaged in conversation as well as during interviews in which he stated, “I felt like I had a foot. I think that in just the short time I had it wired in and mounted to me it was quickly becoming a part of me.”<sup>26,27</sup>

### **LIMITATIONS**

Although the AMI appears to offer many advantages, it is not a true emulator of natural joint dynamics. In a normal, uninjured scenario, the agonist and antagonist muscles operating around an intact joint do not undergo loads exclusively via antagonistic muscle actions; in particular, they frequently undergo stretch due to forces arising from gravitation and inertia. The AMI agonist is capable only of experiencing these load conditions via volitional or external FES activation of its antagonist muscle partner; because both the agonist and antagonist muscles are innervated, this may result in an unnatural perception of antagonist activation by the person with amputation. Furthermore, the AMI necessarily reduces the mechanics of a given joint to an oversimplified architecture consisting of a single agonist and a single antagonist. The muscle dynamics that are witnessed in an intact joint, however, are characterized more frequently by the activation of several muscles with differential lines of action and, in turn, differential contraction/stretch profiles. Whether the AMI remains a close enough emulator of native joint dynamics remains unclear and will be the focus of ongoing research efforts.

### **SUMMARY AND FUTURE DIRECTIONS**

The AMI is a novel surgical construct that appears to augment volitional motor control of adapted prostheses, preserve proprioception, and maintain residual limb volume when applied to an acute lower extremity amputation model. Investigations



**Fig. 9.** Schematic depicting linkage of AMI BKA residual limb to experimental prosthesis. Surface and/or fine-needle electrodes transmit volitional activation of AMIs to powered limb, resulting in (A) eversion, (B) inversion, (C) plantarflexion, and (D) dorsiflexion.

to further validate the value of AMI construction in the setting of upper limb amputation and residual limb revision currently are under way, with pilot procedures of both of these scenarios already having been completed. Furthermore, AMI implementation also is being combined with the development of emerging technologies, including next-generation prostheses and osseointegrated implants specifically designed to interface with AMI surgical architecture. The authors look forward to sharing the results of these ongoing investigations in the future.

More broadly, AMI implementation figures into an emerging paradigm of functional limb restoration in which fundamental redesign of the peripheral nervous system and its effectors is possible. Currently, the use case for the AMI has been to optimize function and minimize pain in the scenario of limb loss; however, there is little reason to believe that similar value would not be achieved in the setting of partial limb injury, degenerative disease, and/or paralysis. Furthermore, AMI construction (either in isolation or in conjunction with other novel reconstructive procedures) potentially may be applied to scenarios in which the goal is to augment, rather than restore, human functionality—and thereby extend the potential capabilities beyond presently accepted norms. The

authors are excited about exploring such possibilities in the coming years.

### CLINICS CARE POINTS

- When performing limb amputation, consideration should be given by the care team to the potential benefits of incorporating AMI construction into the surgical plan
- AMI construction also should be regarded as an option when pursuing revision of residual limbs due to soft tissue breakdown or neuropathic pain

### DISCLOSURE

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