

Tailored Reconstruction for Hand Paralysis: From Principles to Diverse Applications

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Restoration of intrinsic hand function remains one of the most challenging goals in peripheral nerve reconstruction. In high or severe ulnar nerve lesions, the long distance required for axonal regeneration often results in irreversible motor endplate degeneration before regenerating axons can reach the intrinsic muscles. Supercharged end-to-side (SETS) transfer of the anterior interosseous nerve (AIN) to the motor branch of the ulnar nerve (MBUN) provides an additional source of motor axons close to the target muscles while preserving the native ulnar nerve pathway. However, as our clinical experience with this procedure has expanded, we have found that intrinsic hand paralysis cannot be adequately addressed by a uniform nerve transfer strategy.

Our approach has therefore evolved from conventional SETS AIN transfer toward a function-oriented and tailored motor reconstruction. The fundamental principle is to identify not only the injured nerve, but also the specific intrinsic functions that require reinforcement. Preoperative assessment includes conventional intrinsic muscle strength and grip and pinch measurements, together with more detailed evaluation of functional deficits involving thumb control and precision, coordinated function of the four digits, and small-finger/hypothenar function. These findings provide a functional map of the individual patient's paralysis and serve as a guide for subsequent reconstruction.

Intraoperatively, the MBUN is explored and functionally mapped using direct electrical stimulation. Different fascicular regions can produce preferential responses involving the thumb, four digits, or hypothenar muscles. Our sequential stimulation experience has demonstrated that the order and location of recipient fascicular exploration influence the ability to obtain clear and reproducible motor responses. This functional mapping provides information beyond visual fascicular anatomy and allows the recipient nerve to be approached according to the motor deficit of the individual patient. The donor AIN is harvested distally with multiple terminal branches to the pronator quadratus. Rather than treating these branches as a single donor unit, they can be distributed through separate epineurial windows along selected regions of the MBUN. The number and location of coaptations can therefore be modified according to the functional domain requiring greater reinforcement. This multi-branch, multi-target SETS strategy represents an evolution from anatomical nerve transfer toward selective motor augmentation while maintaining continuity of the native ulnar motor pathway. The same reconstructive principle has been extended to diverse clinical settings. In severe ulnar neuropathy associated with degenerative elbow disease, SETS AIN transfer can be combined with cubital tunnel decompression,

anterior transposition, and treatment of the underlying elbow pathology. More recently, we have applied the technique to adults with longstanding ulnar neuropathy secondary to neglected childhood lateral humeral condyle nonunion and chronic valgus deformity. In these patients, satisfactory elbow motion may coexist with severe intrinsic paralysis. A nerve-focused reconstruction combining proximal decompression and transposition with distal AIN motor augmentation may provide functional recovery without necessarily requiring complex osseous reconstruction. These experiences have progressively changed our view of AIN transfer. The procedure should not be regarded simply as an anatomical transfer from the AIN to the ulnar motor nerve. Rather, it can serve as a platform for individualized reconstruction of the paralyzed hand, integrating preoperative functional analysis, intraoperative fascicular mapping, selective donor allocation, and pathology-specific management of the proximal nerve. The future direction of hand paralysis reconstruction may therefore lie in moving from a standardized “one donor–one recipient” concept toward precision motor reconstruction, in which the pattern of paralysis determines how available donor axons are distributed. Such a tailored strategy may broaden the application of distal nerve transfer across diverse etiologies and patterns of intrinsic hand dysfunction while preserving opportunities for native nerve regeneration.