SCIENTIFIC ARTICLE

Nerve Transfers for Restoration of Elbow Flexion in Patients With Acute Flaccid Myelitis

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Purpose The purpose of this study was to evaluate short- to midterm outcomes of patients with acute flaccid myelitis who underwent nerve transfers for restoration of elbow flexion.

Methods Patients with a minimum of 10 months of follow up after undergoing nerve transfers to restore elbow flexion were clinically assessed using the Active Movement Scale (AMS). They were evaluated for any postoperative complications, particularly weakness in the distribution of the donor nerve(s). Fifteen of 25 consecutive patients who were treated using this surgical technique were included in the final analysis.

Results All patients exhibited poor elbow flexion preoperatively (AMS 0 to 3). At a mean follow up of 17.3 months, 80% (15/25) of patients achieved excellent elbow flexion (AMS 6 or 7); 9 of these 15 had full active range of motion. Two patients achieved good elbow flexion (AMS 5) with antigravity movement to less than 50% of the passive range of motion. No cases of superficial or deep infection were reported, and all patients maintained identical motor function, relative to preoperative status, of the muscles innervated by the donor nerves.

Conclusions Nerve transfer surgery has shown promising short- to midterm results for recovery of nerve and muscle function, particularly for the restoration of elbow flexion. We recommend this treatment option for patients not demonstrating clinical improvement after 6 to 9 months of incomplete recovery. *(J Hand Surg Am. 2022;47(1):91.e1-e8. Copyright © 2022 by the American Society for Surgery of the Hand. All rights reserved.)*

Type of study/level of evidence Therapeutic IV.

Key words Acute flaccid myelitis, brachial plexus, elbow flexion, nerve transfers.

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SINCE 2012, ACUTE FLACCID MYELITIS (AFM), a polio-like disease characterized by the acute onset of asymmetric flaccid paralysis, has gained national attention because its incidence has been increasing.[1] Typically affecting children 4 to 12 years of age,[2] a prodrome of fever and usually a respiratory illness are present before the onset of asymmetric flaccid paralysis along with accompanying symptoms such as headache, neck pain, cranial nerve findings (facial palsy, diplopia, dysphagia), and pain in the affected limb(s).[3] Spinal cord involvement, characterized by magnetic resonance imaging

findings of longitudinal lesions in the gray matter, and an association with severe respiratory illness point toward a viral link. The viral disease is most frequently enterovirus D68 (EV D68)[4] but there may be others, including EV-A71 and West Nile virus.[5] Supportive therapy is the mainstay of acute treatment, and intensive care unit admission should be considered given the potential for autonomic nervous system involvement and subsequent respiratory deterioration. Acute flaccid myelitis is often confused with other neurologic disorders, such as Guillain-Barre, acute disseminated encephalomyelitis, and transverse myelitis. Medications such as steroids, intravenous immunoglobulin, and plasma exchange, which are often successful in the autoimmune disorders mentioned earlier, have not been shown to provide clear improvement in AFM, further separating it from these conditions and suggesting a viral etiology.[6]

Many patients affected by this illness will demonstrate only modest improvements in motor function in the months and years following initial presentation, often with the presence of persistent deficits.[7,8] The limited natural history data suggest that patients with motor deficits who do not recover at least partially by 6 to 9 months are unlikely to recover in the long term.[9] In this population, early results of various nerve and/or tendon transfer procedures have been shown to be promising.[10–12] The advantage of nerve transfers over tendon transfers is that they preserve anatomic muscle and joint biomechanics, require less iatrogenic soft-tissue trauma, require less rehabilitation, and allow for the transfer of expendable donor nerves from nontransferable muscles. The traditional window of opportunity for nerve transfers may or may not apply, owing to the potential for sympathetic and motor afferent fibers to assist in maintaining the motor endplates.[13] The importance of surgical timing for nerve transfers in patients with AFM has yet to be clarified.

It has been reported that several types of nerve transfers improve upper extremity function in patients with AFM.[10–12] Those involving the restoration of elbow flexion have demonstrated favorable results in 87% to 100% of patients.[10–12] Our study focuses on evaluating the restoration of elbow flexion in patients with AFM. To date, little has been published on nerve transfer surgery for patients suffering from this uncommon, yet increasingly more prevalent, condition. To our knowledge, this is the largest study to date specifically assessing nerve transfer surgery to restore elbow flexion in patients with AFM.

MATERIALS AND METHODS

The medical records of all patients from January 2008 to July 2020 with a diagnosis of AFM, as defined by the Centers for Disease Control and Prevention (CDC) criteria, were reviewed for inclusion in this study. All included patients underwent nerve transfer surgery performed by the 2 senior authors (S.H.K, D.A.Z) for the goal of restoration of elbow flexion. Hospital charts were reviewed, and patient demographic information was obtained. Information on the levels of paralysis before surgery, date of diagnosis, date of first visit, and ventilatory status was also obtained. Surgery was indicated in patients with (1) persistent motor deficits with an inability to flex the elbow (Active Movement Scale [AMS] less than 5) for a minimum of 6 months from the onset of symptoms and (2) available donor nerves to perform nerve transfer to the musculocutaneous nerve. All included patients demonstrated 5/5 motor grade strength of musculature innervated by the donor nerves, as per the Medical Research Council (MRC) scale.[14] Electrodiagnostic studies were not routinely performed, as their utility in this patient population has yet to be established.

Motor function was assessed preoperatively (within 1 month before surgery) and postoperatively (minimum 12 months after surgery) using the AMS. All clinical and motor function evaluations were completed by licensed hand therapists and the senior authors. AMS scores were recorded for elbow flexion and determined to be poor (AMS 0–4), good (AMS 5), or excellent (AMS 6–7) based on the strength and range of motion (ROM) of the elbow. Achievement of elbow flexion against gravity was deemed a good result, while antigravity movement of $>$ 50% of the passive ROM was deemed an excellent result. Postoperative nerve conduction studies and electromyography were not routinely performed. Patients with less than 1 year of postoperative follow up, insufficient documentation, or procedures other than nerve transfers to restore elbow flexion were excluded.

Surgical technique

Ulnar or median nerve fascicle transfers to the common or motor branches of the musculocutaneous nerve were performed with the patient in the supine position and the operative arm on a hand table. The entire arm was prepared and draped; depending on additional concomitant nerve transfers, the ipsilateral neck and thorax were prepared and draped as well. The procedure was performed through a midline longitudinal incision along the middle third of the

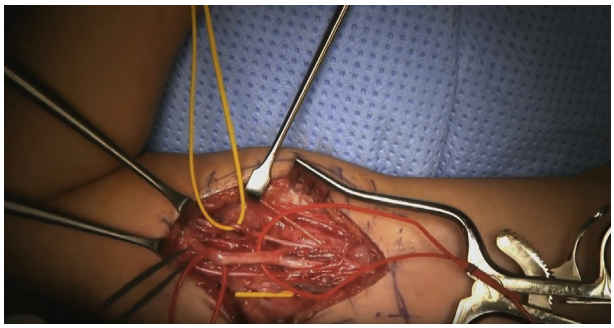

FIGURE 1: Intraoperative image of the medial approach to the brachium. The musculocutaneous nerve has been identified and tagged with 1 yellow vessel loop, and the median nerve is, tagged with 2 red vessel loops.

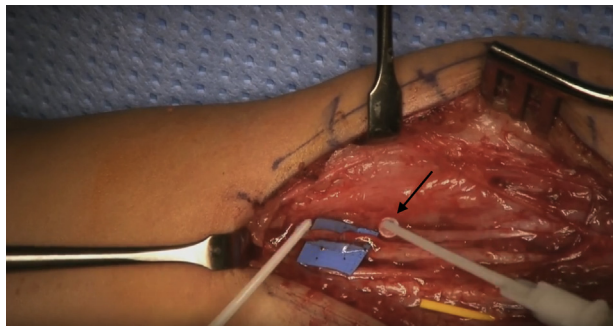

FIGURE 2: Intraoperative image of the same patient as in Figure 1 showing coaptation of the transferred median nerve motor fascicle to the brachialis branch of the musculocutaneous nerve. Coaptation is achieved using fibrin glue, identified by the black arrow.

medial brachium, as described by Little et al.[15] In brief, once the common musculocutaneous nerve and/or its motor nerve fascicles to the biceps or brachialis were mobilized as proximally as possible, the ulnar or median nerve and its expendable motor branches were identified (Fig. 1). These were also mobilized, distally enough to allow for a tension-free coaptation to the musculocutaneous nerve or its branches. A handheld electrical nerve stimulator (Checkpoint Surgical) was utilized to confirm correct identification of the nerves being transferred. Upon deciding which motor fascicles to transfer, we aimed to utilize the best stimulating fascicles on the anterior margin of the ulnar nerve and posterior margin of the median nerve. Prior to taking these fascicles, we confirmed that the rest of the nerve was able to perform all of its functions. Coaptation of the nerve ends was performed under loupe magnification with fibrin glue (Tisseel, Baxter Inc), as shown in Figure 2. Sutures were not used, as the transfers were without tension. After wound closure, a sterile dressing was applied, and the elbow was splinted at 90° of flexion.

When intercostal nerves were used as donors, supine or lateral decubitus positioning of the patient was used, depending on the additional nerve transfers being performed. Since respiratory embarrassment could be present in some patients, we routinely obtained fluoroscopic imaging of the right and left diaphragm. If both lungs moved symmetrically and ventilator support was not needed, we harvested as many intercostal nerves as we needed. If 1 diaphragm was paralyzed, we only harvested 3 intercostal nerves. A curved incision was made from the anterior axillary fold to the sternum following the lower edge of the pectoralis major muscle, with superficial dissection carried down to its fascia while taking care not to injure the intercostobrachial nerve. After incising the fascia and retracting the pectoralis major, the ribs were identified, the soft tissue and periosteum were dissected off, and the intercostal nerve motor branches were harvested, as shown in Figure 3, similar to the technique described by Wahegaonkar et al.[16] Maximal length of the nerves was achieved prior to transection at the most distal end of each nerve. The harvested intercostal nerves were then shuttled through a tunnel made within the serratus anterior muscle and into the medial arm wound. Coaptation to the musculocutaneous nerve or its branches was performed, as previously described.

RESULTS

Upon chart review, a total of 52 patients with AFM were identified based on CDC criteria, 25 of whom had undergone nerve transfers for restoration of elbow flexion. Ten patients were excluded from analysis, owing to either insufficient documentation or postoperative follow up of less than 10 months. This left 15 patients who met inclusion criteria. Eleven of the 15 patients were in the patient cohort of our group's previous study with shorter follow up by Pino et al.[12] Patient demographics are presented in Table 1. Males were predominantly affected (73%), and the average age at disease onset was 56.4 months (range, 5 months to 12 years and 4 months). The right upper extremity was affected in the majority of patients (60%). All but 2 patients had also undergone nerve transfers to the suprascapular and/or axillary nerves for shoulder reanimation. One patient, who was ventilator dependent, had also undergone thoracoscopic intercostal to phrenic nerve transfer for improvement of diaphragmatic function. All donor nerves were assessed intraoperatively using a nerve stimulator, and all demonstrated tetany of their target

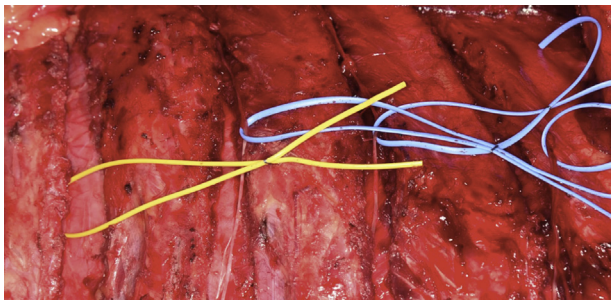

FIGURE 3: Intraoperative image demonstrating the inframammary approach to the ribs. Intercostal nerves have been identified and tagged with 1 yellow and 2 blue vessel loops.

muscle at 0.5 mA and 100-microsecond pulse width. Postoperative immobilization was maintained for 3 weeks and included an elbow flexion splint at 90° and a sling. Patients undergoing intercostal or long thoracic nerve transfers were also limited from abduction in a shoulder immobilizer with restriction of passive shoulder abduction beyond 90° for 3 months. Mean time from disease onset to the first visit at our institution was 10.5 months (range 4 to 20 months) and to surgery was 12.3 months (range 8 to 22 months). Median time between disease onset and surgery was 12.5 months. There were no intraoperative complications, and all patients remained in the hospital overnight with discharge the following day.

All patients had a minimum of 10 months and mean of 17.3 months of follow up (range 10 to 40 months). Evaluation of elbow flexion motor function preoperatively and postoperatively at the time of latest follow up is presented in Table 1. Several different combinations of nerve transfers were performed to restore elbow reanimation. Transfer of ulnar nerve fascicles to the musculocutaneous nerve occurred in 7 patients: 3 to the common musculocutaneous nerve itself and 4 to the biceps motor branch. Median nerve fascicles were transferred to the biceps motor branch in 1 patient and to the brachialis in 3 patients. Two patients underwent transfer of 3 intercostal motor nerves to the common musculocutaneous nerve. Most patients (80%) achieved excellent elbow flexion (AMS 6 or 7) with antigravity movement of at least 50% of the passive ROM; 9 of these patients had full active ROM (AMS 7). Two patients achieved good elbow flexion (AMS 5) with antigravity movement to less than 50% of the passive ROM, while 1 patient did not show recovery of motion against gravity (AMS 1) at final follow up. No cases of superficial or deep infection were reported, and all patients maintained identical motor function, relative to preoperative status, of the functions innervated by the donor nerves.

DISCUSSION

Over the last decade, AFM has become more widely recognized as a polio-like disease that often leads to permanent functional disability among pediatric patients.[2,3] Typically affecting children younger than 15 years of age, AFM is marked by a clinical pattern of flaccid paralysis without spasticity or sensory involvement. Many questions regarding this disease entity remain unanswered. The exact mechanism of action of the disease is unknown, although a link to a virally mediated process of anterior horn cell destruction has been suggested. Enteroviruses A-71 and D-68 have been speculated to be viral causes owing to their tropism for the spinal cord, muscles, and respiratory system.[17,18] Medical management of the acute viral illness is mainly supportive, with questionable benefits coming from the use of plasma exchange, immunomodulating agents, and steroids.[3] The sequela of anterior horn cell destruction is variable, leaving most patients with incomplete recovery and long-term muscle weakness. Partial recovery of muscle groups can be exhibited up to 6–9 months following onset of the disease,[9] but many patients are left with complete loss of function of 1 or more muscle groups in no specific pattern. A consensus regarding treatment for persistent muscle paralysis and loss of upper limb function has not been established. A proposed option with early encouraging results is nerve transfer surgery. If no improvement in symptoms is demonstrated within 3 months of disease onset, surgical intervention at the 6-month mark should be strongly considered, particularly if the patient's AMS score is 0 or 1.

Nerve transfers for restoration of upper limb function among patients with AFM have been reported, although they have been limited to a small number of cases. Liao et al[11] demonstrated successful results in 5 of 6 pediatric patients with brachial plexus polio-like paralysis due to the less virulent A-71 strain, all of whom had complete absence of shoulder function (Gilbert stage 0) and achieved Gilbert stage 4 shoulder abduction (greater than 160°). All 5 patients underwent spinal accessory to suprascapular nerve transfer. Three of 5 patients also underwent concomitant transfer of the phrenic nerve to either the axillary nerve or the posterior division of the upper trunk. All 5 patients had surgery within 1 year of disease onset. One patient with stage 0 shoulder abduction had a poor result (remained at stage 0) after

TABLE 1. Patient Demographics and Outcomes

Case	Sex	Laterality	Age at AFM Onset (y)	Time Until Surgery (mo)	Preop AMS/Toronto	Procedure	Follow Up (mo)	Postop AMS/Toronto
1	F	R	1	12	1 / 0.3	• CN 11 to SSN • Median n. fascicle to MSC (brachialis) • Ulnar n. fascicle to radial n. (triceps) • IC (x3) to axillary n.	40	6 / 1.6
2	F	R	12	16	2 / 0	• Ulnar n. fascicle to MSC (biceps)	14	5 / 1.3
3	M	R	<1	17	3 / 0	• LTN to axillary n. • CN 11 to SSN • Ulnar n. fascicle to MSC	19	7 / 2
4	M	L	1	22	1 / 0	• LTN to axillary n. • CN 11 to SSN • Ulnar n. fascicle to MSC	12	5 / -
5	F	R	7	13	1 / 0	• CN 11 to SSN • Median n. fascicle to axillary n. • Ulnar n. fascicle to MSC	13	7 / 2
6	M	R	5	8	3 / 0	• SSN to IC (x2) • IC (x3) to axillary n. • Median n. fascicle to MSC (brachialis) • Ulnar n. fascicle to radial n. (triceps)	19	7 / 2
7	M	R	6	9	0 / 0	• Ulnar n. fascicle to radial n. (triceps) • Median n. fascicle to MSC (biceps) • IC (x2) to SSN • IC (x3) to axillary n.	21	7 / 2
8	M	L	2	8	0 / 0	• IC (x4) to SSN • Ulnar n. fascicle to MSC (biceps) • Radial n. (triceps) to axillary n.	17	6 / -
9	M	L	3	10	3 / 1	• CN 11 to SSN • Ulnar n. fascicle to MSC (biceps) • IC (x3) to axillary n.	14	7 / 2
10	M	L	2	14	2 / 0.6	• IC (x2) to SSN • IC (x3) to MSC • SSN notch release	19	7 / 2
11	M	L	2	20	3 / 0.6	• CN 11 to SSN • Median n. fascicle to MSC (biceps) • Radial n. (triceps) to axillary n.	26	7 / 2

(Continued)

TABLE 1. Patient Demographics and Outcomes (Continued)

Case	Sex	Laterality	Age at AFM Onset (y)	Time Until Surgery (mo)	Preop AMS/Toronto	Procedure	Follow Up (mo)	Postop AMS/Toronto
12	M	L	6	9	1 / 0.3	• CN 11 to SSN • Median n. fascicle to MSC (biceps). • Ulnar n. fascicle to axillary n. • Radial n. fascicle (distal to common triceps br. point) to common triceps br. (radial n.)	12	6 / 1.6
13	F	R	< 1	7	0 / 0	• CN 11 to SSN • Radial n. (triceps) to axillary n. • Median n. (fascicle) to MSC (brachialis) • Ulnar n. fascicle to MSC (biceps)	10	7 / 2
14	M	R	6	10	0 / 0	• IC (x3) to SSN • IC (x2) to axillary n. • Ulnar n. (fascicle) to radial n. (triceps) • Median n. fascicle to MSC (biceps)	12	7 / 2
15	M	L	11	10	0 / 0	• IC (x3) to MSC	12	1 / 0

br., branch; CN 11, cranial nerve 11 (or spinal accessory nerve); IC = intercostal nerve; mo, month; LTN, long thoracic nerve; MSC, musculocutaneous nerve; n., nerve; SSN, suprascapular nerve; y, year.

undergoing a phrenic to suprascapular nerve transfer 3 years after onset of paralysis. Although excellent shoulder abduction was restored in all but 1 patient in this series, other studies have reported more variable return of shoulder function after shoulder nerve transfers in patients with a mixed viral etiology. Pino et al[12] reported on 11 patients with AFM who underwent various nerve transfers for restoration of shoulder, elbow, and hand function. All patients had significant shoulder involvement, with the great majority exhibiting an AMS of 0 for shoulder abduction and external rotation preoperatively. Shoulder external rotation against gravity was achieved in 70% of patients, 50% of whom recovered excellent function (AMS $>$ 5). Shoulder abduction recovery was less favorable, with 70% of patients having a poor outcome. Similar results were noted by Saltzman et al[10] in a report of 2 patients with absent shoulder abduction. One patient, who underwent thoracodorsal and radial nerve transfer to the axillary nerve, recovered 135° of shoulder external rotation with MRC scale 3/5 strength at 35 months postoperatively. Although the patient regained deltoid activation and bulk, no appreciable shoulder abduction was noted. The second patient, who had unilateral shoulder involvement with absent abduction, underwent medial pectoral and radial fascicle transfer to the axillary nerve. At 32 months postoperatively, only 45° of active shoulder abduction was regained. Restoration of elbow flexion has been more predictable and better achieved than restoration of shoulder function. The reason for this has not been fully elucidated; however, investigation in neonatal mice suggested that central muscles, such as those of the shoulder girdle, were the primary sites of viral replication (in addition to anterior horn cells) and could be more of a target than more distal extremity musculature.[19] Pino et al[12] reported excellent results (AMS 6 or 7) in 7 of 8 patients who underwent either ulnar or median nerve fascicle transfer to the musculocutaneous nerve or its branches at a mean of 14.5 months of follow up. Saltzman et al[10] also demonstrated marked improvement in elbow flexion in their 2 patients after over 30 months of follow up after transfer of an expendable median nerve fascicle to the brachialis branch of the musculocutaneous nerve. Both patients achieved full elbow ROM with MRC scale 5/5 strength. Preoperatively, one patient had MRC 0/5 elbow flexion strength while the other had MRC 2/5 elbow flexion strength.

Our results are similar to those demonstrated by Pino and Saltzman.[10,12] Additionally, we report a minimum of 1 year of follow-up data on 2 patients with AFM who underwent intercostal to musculocutaneous nerve transfers, which to this date, have not been described for patients with AFM. Although 1 patient (case 10) did exhibit full recovery of elbow flexion at 19 months of follow up, 1 patient (case 15) did not recover elbow flexion after this transfer at the 12-month follow-up date and may not have had viable intercostal nerves to transfer from the start. In that child, we had to use intercostal nerves 3, 5, and 6, skipping intercostal nerve 4 because it did not stimulate with the handheld electrical nerve stimulator. The muscle also appeared pale upon intraoperative assessment. In hindsight, it was likely that the adjoining intercostals were affected as well but below the 80% axotomy level that was clinically measurable.[20] Therefore, there is an argument that pre- or intraoperative electromyography (EMG) of donor nerve-innervated muscles is necessary to assess for a decrease in number and an increase in amplitude of motor units, suggesting partial axotomy with reinnervation. The specificity and sensitivity of EMG or its clinical utility have yet to be defined for AFM. Further investigation is required to accurately understand, interpret, and apply the results obtained by EMG. Similar procedures for reanimation of elbow flexion after brachial plexus birth injuries have also demonstrated excellent results.[21,22] Al-Qattan's experience with Oberlin ulnar nerve transfer to the biceps nerve on 2 late-presenting toddlers with Erb palsy (16 and 18 months after birth) demonstrated "normal" recovery of biceps function (MRC scale 5 strength) at 5 months postoperatively. Little et al[22] were able to obtain an AMS score of greater than 6 in 87% of their 31-patient cohort who underwent median and/or ulnar nerve fascicle transfer to the musculocutaneous nerve. The authors noted a positive correlation between younger age at the time of surgery and improved recovery of elbow flexion and supination. They suggested a relative cutoff of 15 months for achieving excellent results after nerve transfers for the treatment of brachial plexus birth injuries. This relationship of delayed presentation to intervention with worse outcomes in children undergoing late nerve transfers has not been substantiated among patients with AFM. Persistence of sympathetic and afferent motor fibers may delay motor-endplate demise in this efferent motor fiber-specific disease.[13]

To our knowledge, this is the largest report to date on the midterm outcomes of patients with this rare disease who underwent nerve transfer procedures for restoration of elbow flexion. This study, however, is not without its limitations. The retrospective nature is

subject to its inherent biases as is the limited number of patients, short follow-up time, and no comparison to other interventions or more conservative means of treatment. Although our analysis includes a validated outcome measure (the AMS), inclusion of other outcome measures and physical examination findings may better elucidate the function of our patients preoperatively and postoperatively. An advantage of our study is that all procedures are performed by 2 fellowship-trained pediatric hand surgeons specializing in nerve transfer procedures, limiting confounders related to surgical technique.

AFM is a devastating, polio-like condition leading to the acute onset of flaccid muscle paralysis with limited to no recovery of 1 or more muscle groups. Non-operative forms of treatment are mainly supportive and do not provide a demonstrable benefit, especially after 6 to 9 months of monitoring for muscle recovery. Nerve transfer surgery has shown promising midterm results for recovery of nerve and muscle function, particularly for the restoration of elbow flexion. We recommend this treatment option for patients not demonstrating clinical improvement after 6 to 9 months of incomplete recovery.

REFERENCES

1. Centers for Disease Control and Prevention. AFM cases in US 2019. Accessed August 24, 2020. https://www.cdc.gov/acute-flaccid-myelitis/cases-in-us.html
2. Sejvar JJ, Lopez AS, Cortese MM, et al. Acute flaccid myelitis in the United States, August-December 2014: results of nationwide surveillance. *Clin Infect Dis*. 2016;63(6):737–745.
3. Hopkins SE. Acute flaccid myelitis: etiologic challenges, diagnostic and management considerations. *Curr Treat Options Neurol*. 2017;19(12):48.
4. Knoester M, Helfferich J, Poelman R, et al. Twenty-nine cases of enterovirus-D68–associated acute flaccid myelitis in Europe 2016: a case series and epidemiologic overview. *Pediatr Infect Dis J*. 2019;38(1):16–21.
5. Helfferich J, Knoester M, Van Leer-Buter CC, et al. Acute flaccid myelitis and enterovirus D68: lessons from the past and present. *Eur J Pediatr*. 2019;178(9):1305–1315.
6. Nelson GR, Bonkowsky JL, Doll E, et al. Recognition and management of acute flaccid myelitis in Children. *Pediatr Neurol*. 2016;55:17–21.
7. Kirolos A, Mark K, Shetty J, et al. Outcome of paediatric acute flaccid myelitis associated with enterovirus D68: a case series. *Dev Med Child Neurol*. 2019;61(3):376–380.
8. Gordon-Lipkin E, Muñoz LS, Klein JL, Dean J, Izbudak I, Pardo CA. Comparative quantitative clinical, neuroimaging, and functional profiles in children with acute flaccid myelitis at acute and convalescent stages of disease. *Dev Med Child Neurol*. 2019;61(3):366–375.
9. Andersen EW, Kornberg AJ, Freeman JL, Leventer RJ, Ryan MM. Acute flaccid myelitis in childhood: a retrospective cohort study. *Eur J Neurol*. 2017;24(8):1077–1083.
10. Saltzman EB, Rancy SK, Sneag DB, Feinberg Md JH, Lange DJ, Wolfe SW. Nerve transfers for enterovirus D68-associated acute flaccid myelitis: a case series. *Pediatr Neurol*. 2018;88:25–30.
11. Liao HT, Chuang DC, Ulusal AE, Schrag C. Surgical strategies for brachial plexus polio-like paralysis. *Plast Reconstr Surg*. 2007;120(2):482–493.
12. Pino PA, Intravia J, Kozin SH, Zlotolow DA. Early results of nerve transfers for restoring function in severe cases of acute flaccid myelitis. *Ann Neurol*. 2019;86(4):607–615.
13. Khan MM, Lustrino D, Silveira WA, et al. Sympathetic innervation controls homeostasis of neuromuscular junctions in health and disease. *Proc Natl Acad Sci U S A*. 2016;113(3):746–750.
14. Curtis C, Stephens D, Clarke HM, Andrews D. The active movement scale: an evaluative tool for infants with obstetrical brachial plexus palsy. *J Hand Surg Am*. 2002;27(3):470–478.
15. Little KJ, Zlotolow DA, Soldado F, Cornwall R, Kozin SH. Median and/or ulnar nerve fascicle transfer for the restoration of elbow flexion in upper neonatal brachial plexus palsy. *JBJS Essent Surg Tech*. 2014;4(2):e8.
16. Wahegaonkar AL, Doi K, Hattori Y, Addosooki AI. Technique of intercostal nerve harvest and transfer for various neurotization procedures in brachial plexus injuries. *Tech Hand Up Extrem Surg*. 2007;11(3):184–194.
17. Messacar K, Asturias EJ, Hixon AM, et al. Enterovirus D68 and acute flaccid myelitis-evaluating the evidence for causality. *Lancet Infect Dis*. 2018;18(8):e239–e247.
18. Hixon AM, Yu G, Leser JS, et al. A mouse model of paralytic myelitis caused by enterovirus D68. *PLoS Pathog*. 2017;13(2):e1006199.
19. Sun S, Bian L, Gao F, et al. A neonatal mouse model of enterovirus D68 infection induces both interstitial pneumonia and acute flaccid myelitis. *Antiviral Res*. 2019;161:108–115.
20. Brown JM, Shah MN, Mackinnon SE. Distal nerve transfers: a biology-based rationale. *Neurosurg Focus*. 2009;26(2):E12.
21. Al-Qattan MM. Oberlin's ulnar nerve transfer to the biceps nerve in Erb's birth palsy. *Plast Reconstr Surg*. 2002;109(1):405–407.
22. Little KJ, Zlotolow DA, Soldado F, Cornwall R, Kozin SH. Early functional recovery of elbow flexion and supination following median and/or ulnar nerve fascicle transfer in upper neonatal brachial plexus palsy. *J Bone Joint Surg Am*. 2014;96(3):215–221.