Nerve transfers for treatment of isolated axillary nerve injuries

Margie Wheelock MD[1], Tod A Clark MD FRCSC[2], Jennifer L Giuffre MD FRCSC[1]

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BACKGROUND: The most common neurological defect in traumatic anterior glenohumeral dislocation is isolated axillary nerve palsy. Most recover spontaneously; however, some have persistent axillary neuropathy. An intact rotator cuff may compensate for an isolated axillary nerve injury; however, given the high rate of rotator cuff pathology with advancing age, patients with an axillary nerve injury are at risk for complete shoulder disability.

OBJECTIVE: To review reconstruction of the axillary nerve to alleviate shoulder pain, augment shoulder stability, abduction and external rotation to alleviate sole reliance on the rotator cuff to move and stabilize the shoulder.

METHODS: A retrospective review of 10 patients with an isolated axillary nerve injury and an intact rotator cuff who underwent a triceps nerve branch to axillary nerve transfer was performed. Patient demographics, surgical technique, deltoid strength, donor-site morbidity, complications and time to surgery were evaluated.

RESULTS: Ten male patients, mean age 38.3 years (range 18 to 66 years), underwent a triceps to axillary nerve transfer for isolated axillary nerve injury 7.4 months (range five to 12 months) post-traumatic shoulder dislocation. Deltoid function was British Medical Research Council grade 0/5 in all patients preoperatively and ≥3/5 deltoid strength in eight patients at final follow-up (14.8 months [range 12 to 25 months]). There were no complications and no donor-site morbidity.

CONCLUSION: A triceps to axillary nerve transfer for isolated axillary neuropathy following traumatic shoulder dislocation improved shoulder pain, stability and deltoid strength, and potentially preserves shoulder function with advancing age by alleviating sole reliance on the rotator cuff for shoulder abduction and external rotation.

Key Words: *Isolated axillary nerve injury; Nerve transfer*

Le transfert des nerfs pour traiter les lésions isolées du nerf axillaire

HISTORIQUE : La paralysie isolée du nerf axillaire est la principale anomalie neurologique après une dislocation gléno-humérale traumatique antérieure. La plupart guérissent spontanément, mais certains souffrent de neuropathie axillaire persistante. Une coiffe des rotateurs intacte peut compenser une lésion isolée du nerf axillaire. Cependant, compte tenu du fort taux de pathologies de la coiffe des rotateurs liées au vieillissement, les patients ayant une lésion du nerf axillaire risquent une invalidité complète de l'épaule.

OBJECTIF : Examiner la reconstruction du nerf axillaire pour soulager la douleur de l'épaule et en accroître la stabilité, l'abduction et la rotation externe afin d'éviter de se fier uniquement à la coiffe des rotateurs pour bouger et stabiliser l'épaule.

MÉTHODOLOGIE : Les chercheurs ont procédé à l'analyse rétrospective de dix patients ayant une lésion isolée du nerf axillaire et une coiffe des rotateurs intacte qui ont subi un transfert de la branche du nerf du triceps sur le nerf axillaire. Ils ont évalué la démographie des patients, la technique chirurgicale, la force du deltoïde, la morbidité du site du donneur, les complications et le délai avant l'opération.

RÉSULTATS : Dix patients de sexe masculin, d'un âge moyen de 38,3 ans (plage de 18 à 66 ans), ont subi un transfert du nerf du triceps sur le nerf axillaire en raison d'une lésion isolée du nerf axillaire, et ce, 7,4 mois (plage de cinq à 12 mois) après une dislocation traumatique de l'épaule. Chez tous les patients avant l'opération, la fonction du deltoïde était de 0 sur une échelle de 5 selon le *British Medical Research Council*, tandis que la force du deltoïde était d'au moins 3 sur 5 chez huit patients au suivi final (14,8 mois [plage de 12 à 25 mois]). Il n'y a eu aucune complication et aucune morbidité au site du donneur.

CONCLUSION : Le transfert du nerf du triceps sur le nerf axillaire pour soigner une neuropathie axillaire isolée après une dislocation traumatique de l'épaule soulageait la douleur et la stabilité de l'épaule et la force du deltoïde et assurait la préservation potentielle de la fonction de l'épaule malgré le vieillissement, car la coiffe des rotateurs n'était plus l'unique mode d'abduction et de rotation externe de l'épaule.

Shoulder dislocations are the most common major joint dislocations in the human skeleton, accounting for up to 45% of all dislocations (1). Several injuries associated with anterior glenohumeral dislocations include neurological defects, rotator cuff injuries and fractures of the greater tuberosity (2). These injuries can influence immediate recovery of function or reduce functional reserve, leading to upper extremity disability with subsequent injury. In a large-scale study of anterior glenohumeral dislocations by Robinson et al (2), 5.8% (210 of 3633 patients) were found to have an associated neurological injury and 7.8% (282 of 3633 patients) had combined injuries (neurological injury and either rotator cuff injury or greater tuberosity fracture). The most common neurological deficit following an anterior glenohumeral dislocation is an isolated axillary nerve injury. While most recover spontaneously over the subsequent three to four months, some experience persistent axillary neuropathy.

The axillary nerve innervates the deltoid and teres minor muscles, which provide humeral stability within the glenoid fossa, shoulder external rotation and shoulder abduction. The axillary nerve also enables sensation over the lateral shoulder. Both the rotator cuff and deltoid muscles contribute to shoulder abduction: the rotator cuff muscles are more effective abductors at low abduction angles (0° to 30°) and the deltoid at higher abduction angles (>30°) (3). The functional deficit in a patient with an axillary neuropathy depends on nerve recovery, associated rotator cuff injuries and the patient's premorbid rotator cuff function. Individuals with intact rotator cuffs may be able to compensate for the axillary nerve dysfunction; however, without the deltoid, their shoulder fatigues easily, limiting their activities. Additionally, sole reliance on the rotator cuff may be problematic in later years, given high rates of partial or complete rotator cuff tears in the aging population (4,5).

Management of persistent axillary neuropathy has traditionally consisted of axillary nerve grafting, with 73% to 88% of patients regaining useful deltoid strength (6-9). Success of this procedure is dependent on the length of graft required, determination of the distal extent of the axillary nerve injury and has the added donor-site morbidity. Triceps branch to axillary nerve transfer was originally described by Leechavenvongs et al (10) for C5 and C6 brachial plexus injuries. This transfer has been effective for restoring shoulder abduction, external rotation and stability. It has the advantages of a single neurorrhaphy in close proximity to the target muscle and a single operative site.

[1]*Section of Plastic Surgery, Department of Surgery;* [2]*Department of Orthopedic Surgery, University of Manitoba, Winnipeg, Manitoba*
Correspondence: Dr Jennifer L Giuffre, University of Manitoba, Section of Plastic Surgery, Department of Surgery, 75 Poseidon Bay, Winnipeg, Manitoba R3M 3E4. Telephone 204-927-2835, e-mail jgiuffre@panamclinic.com

TABLE 1
Patient demographics, injury time, time to surgery, pre- and postoperative deltoid strength and duration of final follow-up

Patient	Age, years/sex	Smoker	Injury type	Associated injuries	Delay from injury to surgery, months	Duration of follow-up, months	Deltoid BMRC score	
							Preoperative	Postoperative
1	63/male	No	Trauma		5	15	0	4
2	50/male	Yes	Motor vehicle collision		8	12	0	4
3	37/male	No	Snowmobile	Humerus, scapula, rib spinous process, sternum fractures	12	20	0	3
4	66/male	Yes	Motor vehicle collision	Humerus fracture	7	25	0	0
5	24/male	Yes	Sports trauma		6	14	0	5
6	18/male	No	Motorcross		7	14	0	5
7	19/male	No	Motorcross	Clavicle fracture	7	12	0	0
8	64/male	No	Trauma		8	12	0	5
9	22/male	Yes	Motor vehicle collision	Rib fractures	8	12	0	4
10	20/male	No	Motor vehicle collision	Rib fractures	6	12	0	4

BMRC British Medical Research Council

Figure 1) *A 24-year-old man (patient 5) with an isolated left axillary nerve injury following an anterior shoulder dislocation while playing rugby. Note the wasting in the left deltoid*

While axillary neuropathy in brachial plexus patients has been well described in the literature, little has been published on isolated axillary nerve injuries (11,12). We propose reconstruction of the isolated axillary nerve injury following anterior shoulder dislocation, despite an intact rotator cuff, to augment shoulder stability, abduction and external rotation to alleviate sole reliance on the rotator cuff to move and stabilize the shoulder.

METHODS

A retrospective chart review of all patients presenting with an isolated axillary injury following shoulder dislocation between July 2011 and July 2013 was performed. Inclusion criteria included adult patients (≥18 years of age) with an isolated axillary nerve injury and intact rotator cuff muscles treated with a triceps to axillary nerve transfer and minimum follow-up of one year. Exclusion criteria included patients with nontraumatic causes of axillary neuropathy or multiple nerve injuries. A total of 10 male patients who met both the inclusion and exclusion criteria were identified.

The charts were reviewed for patient demographics, surgical technique, preoperative British Medical Research Council (BMRC) deltoid and triceps strength, time to surgery and length of final follow-up. Outcome measures included postoperative shoulder pain, deltoid and triceps BMRC strength, donor deficits and complications. All patients underwent electromyography (EMG) and nerve conduction studies (NCS) at six weeks to confirm an isolated axillary nerve injury, and magnetic resonance imaging to confirm the absence of a rotator cuff injury. All patients were scheduled for a triceps to axillary nerve transfer if they failed to show clinical or electrophysiological (EMG/NCS) evidence of reinnervation by a minimum of three months after injury. Patients were reevaluated clinically before surgery to ensure no return of axillary nerve function. The procedure was performed as described by Leechavengvongs et al (10).

RESULTS

Patient demographics and individual outcomes are presented in Table 1. All patients were male and were a mean age of 38.3 years (range 18 to 66 years). All 10 patients sustained an anterior glenohumeral dislocation confirmed by radiographs. The reported mechanism of injury was motor vehicle collision (including motorcycle injuries, snowmobile injuries and motorcross injuries) in seven patients and trauma (one was pinned between two barrels, one was injured playing rugby and one by lifting heavy machinery) in three.

Two patients had associated fractures of the humerus, which underwent fixation before nerve transfer. One patient had an associated clavicle fracture, one had associated scapular, rib, sternum and spinous process fractures, and two had associated rib fractures that were all treated conservatively.

Although all patients underwent at least one attempt at shoulder relocation, one required relocation under general anesthetic, one underwent two attempts at shoulder relocation and one underwent three attempts. All of the dislocations were identified acutely following the trauma: no patient presented with a chronic shoulder dislocation. No patient exhibited clinical or magnetic resonance imaging evidence of a rotator cuff tear. Preoperative electrophysiological testing (EMG/NCS) confirmed an isolated axillary nerve injury in all patients.

BMRC deltoid strength on preoperative testing was 0 in all patients (Figures 1, 2 and 3). In the standing position, patients were examined for deltoid wasting, humeral sag within the glenoid fossa, loss of sensation over the lateral aspect of the shoulder and any muscle contraction while palpation of the deltoid during attempts at shoulder forward flexion and abduction. The BMRC triceps strength preoperatively was 5 in all patients. The average time to nerve transfer was 7.4 months (range five to 12 months). With no evidence of motor unit potentials on EMG/NCS at three months postinjury, patients were scheduled for surgery. Patients were re-examined before surgery to ensure no return of deltoid muscle contraction on attempts at shoulder forward flexion and abduction. The branch to the medial head of triceps was transferred into the axillary nerve in seven cases, the branch to the lateral head of triceps was transferred in one and the branch to the long head of triceps was transferred in two (Figure 4). In all cases, the triceps nerve branch was transferred into the axillary nerve alone, the branch to teres minor was dissected out and excluded from the transfer (all patients had functioning infraspinatus muscle to externally rotate the shoulder).

Figure 2) *With the patient attempting to abduct the left shoulder, he is using scapulothoracic motion. Note the prominent left humeral head*

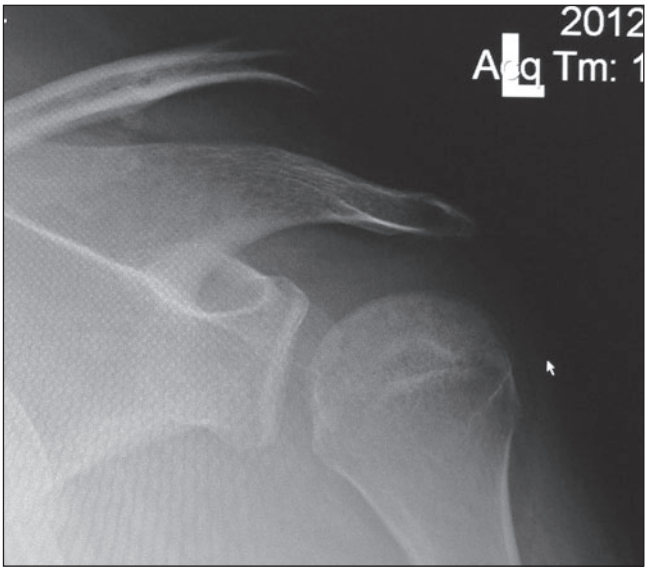

Figure 3) *With loss of the left deltoid muscle, the humerus sags within the glenoid fossa, causing lack of stability*

The average postoperative deltoid BMRC score was 3.4, with a range from 0 to 5 at final follow-up (mean 14.8 months; range 12 to 25 months). The deltoid muscle was palpated during attempts at shoulder forward flexion and abduction. There were two patients with a postoperative deltoid MRC grade of 0/5 (no palpable deltoid muscle contraction), while the remaining eight had a postoperative deltoid MRC grade of ≥3 (palpable contraction of the deltoid against gravity was grade 3, palpable contraction of the deltoid against resistance was grade 4 and equal strength to the contralateral deltoid was grade 5). The mean postoperative triceps BMRC score was 5 (Figures 5A and 5B, and Figure 6). All patients reported resolution of pain.

The average follow-up was 14.8 months (range 12 to 25 months). There were no complications and no donor deficits. All patients had 5/5 triceps postoperatively. Similar to previous studies (11), shoulder range of motion was not included as an outcome measure because full normal shoulder range of motion is still possible with intact rotator cuff muscles and a paralyzed deltoid, and stiffness of the glenohumeral joint may limit movement despite a strong deltoid. BMRC grading of the deltoid muscle was determined by palpating the isolated deltoid muscle in shoulder forward flexion and abduction.

At final follow-up, three patients returned to their previous occupations, three returned to school, three had retired and one died in a motor vehicle collision.

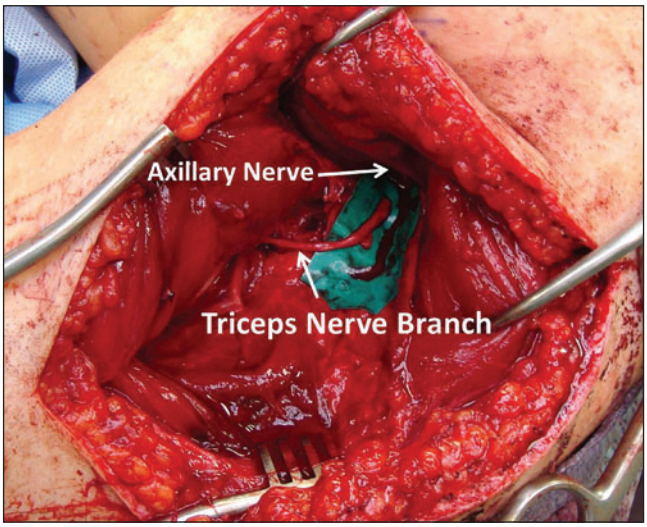

Figure 4) *Intraoperative photograph of the triceps nerve branch juxtaposed to the axillary nerve*

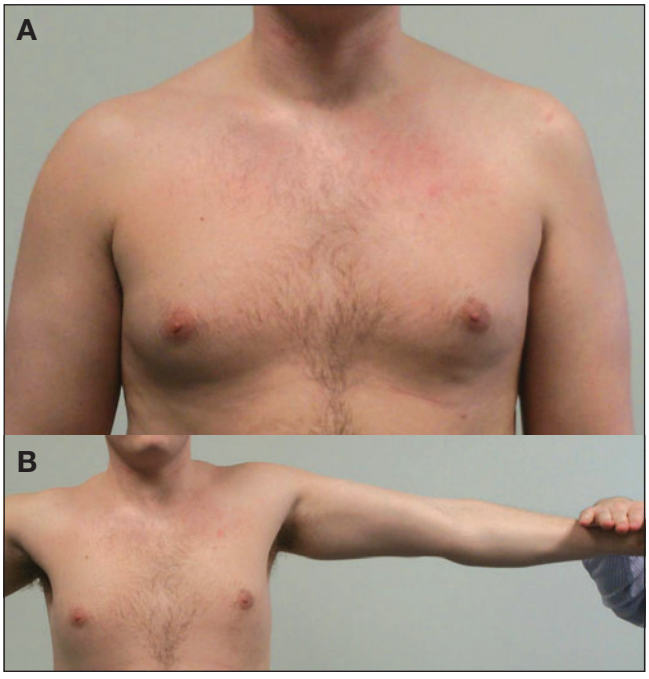

Figure 5) A *Follow-up of patient 5 one year after a left triceps nerve branch to axillary nerve transfer. Note the restoration of the left deltoid contour compared with the preoperative photograph in Figure 1.* **B** *The patient achieved 5/5 British Medical Research Council left deltoid strength. He is being tested against resistance*

DISCUSSION

Normal shoulder range of motion is possible following an axillary nerve injury if the suprascapular nerve and rotator cuff muscles are intact (13). Patients can be deceptively functional on simple examination; however, without the deltoid, their shoulder fatigues easily, limiting their activities (12). Shoulder range of motion is a combination of movement at both the glenohumeral joint and the scapulothoracic-gliding plane, with contributions from the rotator cuff, deltoid, pectoralis, latissimus and serratus muscles. These same muscles, in addition to the glenohumeral ligaments, contribute to shoulder stability. Although studies investigating static shoulder stability point to the role of the glenohumeral capsule, dynamic shoulder stability relies significantly more on the rotator cuff and deltoid for stabilization of the glenohumeral joint during

Figure 6) *Additional example of a patient shown preoperatively* (**left**) *with an isolated left axillary nerve injury following an anterior shoulder dislocation and one year postoperatively left triceps nerve branch to axillary nerve transfer* (**right**). *Note the left deltoid*

arm range of motion (14). Shoulder range of motion is a complex interaction between the deltoid and rotator cuff muscles. The deltoid and rotator cuff both contribute to arm abduction, with differing contributions depending on the degree of abduction. In biomechanical studies investigating the deltoid, the abductor moment arm at 0° abduction (and neutral rotation) is 0 cm for the anterior deltoid and 1.4 cm for the middle deltoid (15). The moment arm progressively increases with increasing abduction; thus, by 60° abduction, the moment arm is 1.5 cm to 2 cm for the anterior deltoid, and 2.7 cm to 3.2 cm for the middle deltoid. At low abduction angles (0° to 40°), the moment arm of the deltoid is less than the moment arms of the supraspinatus, subscapularis and infraspinatus muscles, implying that the deltoid is a more effective abductor at higher abduction angles than the rotator cuff muscles (15). The lateral deltoid abducts the arm in the plane of the scapula while the anterior deltoid flexes the arm and rotates it medially – no other muscle can perform these specific functions (16).

Patients with disabling brachial plexus injuries are likely to be protective of their shoulder given the already limited function. Unlike patients with a brachial plexus injury, patients with an isolated axillary nerve injury are functional, albeit weaker and have less endurance (12); thus, they are likely subject to rotator cuff injuries. With a statistically significant increase in asymptomatic and symptomatic rotator cuff tears with advancing age, (54% rate among subjects >60 years of age) (4,5), there is concern that individuals with an axillary neuropathy will develop complete shoulder disability if the axillary nerve is not reconstructed due to the potential of a future rotator cuff tear.

The success of triceps to axillary nerve transfers in brachial plexus injuries has been well documented (11,17,18). Our results show that with this transfer, functional deltoid strength is recovered (3.4/5). Lee et al (11) found similar BMRC deltoid strength (mean [± SD] 3.5±1.1) when investigating triceps to axillary nerve transfer in 21 patients with isolated axillary nerve injuries. Eight of the 10 patients in our study achieved a postoperative deltoid BMRC grade of ≥3 of 5. There were two patients who did not regain any deltoid function. The failure to regain deltoid function may have been due to several factors including: the severity of injury to the axillary nerve including injury to the motor end plates within the deltoid muscle (both patients were involved in high speed motor vehicle collisions); the older age and smoking history of patient 4; and the obese habitus of patient 7 (body mass index >40 kg/m^2). In a multiple linear regression model, Lee et al (11) reported that delay from injury to surgery, age of the patient and body mass index were factors affecting the outcome of the triceps to axillary nerve transfer.

Within the brachial plexus patient population, the risk associated with surgery is well tolerated given the patient's devastating functional limitations. In isolated axillary nerve injury, patients have near normal shoulder range of motion; therefore, to undergo a nerve transfer to the axillary nerve, the morbidity of the procedure must be low. The present study, similar to those in the literature, did not document any complications or donor-site morbidities (11,17,18).

Limitations of the present study include its retrospective design and small sample size.

CONCLUSION

With no donor-site morbidity or complications, a triceps to axillary nerve transfer to restore deltoid function appears to be reasonable in an isolated axillary nerve injury, despite an intact rotator cuff. While the rotator cuff does provide some shoulder abduction for these patients, the contribution of the deltoid as well as the potential for future rotator cuff injury supports nerve transfer in this patient population. This transfer improves shoulder stability, external rotation and abduction and has the potential to prevent complete shoulder disability in a large number of patients who may develop rotator cuff injures with advancing age.

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