

Case Study

Compiled Date: December 30, 2023

Axillary Nerve Lesions with Deltoid Paralysis

Roger Pillemer OAM, MBBCh(Rand), FRCS(Ed), FRACS, FAOrthA*

Australian Orthopaedic Association, Australia

*Corresponding author: Roger Pillemer, Australian Orthopaedic Association, Australia, E-mail: roger@rpillemer.com.au

Abstract

How is it possible to obtain near normal shoulder function after permanent loss of axillary nerve function and complete deltoid atrophy.

Case Study

A case report and review of the literature written up in the journal Military Medicine (March 2016) [1] presented a patient with a closed axillary nerve injury. The case report describes a 43-year-old male who was involved in a motor vehicle accident at the age of 18 sustaining a dislocation of his left shoulder and an axillary nerve lesion which proved to be complete. Four years after the injury he enlisted in the U.S. Army with no limitation of function in his left shoulder; he was able to perform 65 push up repetitions in 2 minutes (with 100 being the highest score obtainable). There was no pain or dysfunction and on examination there was a full range of shoulder movement with absent sensation in the axillary nerve distribution; X-rays and MRI were normal apart from showing complete atrophy of the deltoid. Electromyogram (EMG) studies confirmed complete deltoid paralysis.

The authors raised the issue of the role of the supraspinatus in the deltoid deficient shoulder. They did not however suggest how this remarkable return of shoulder function had occurred. They also reviewed the literature and, leaving out those cases treated surgically and those without a proper follow-up, they reported 43 cases of loss of deltoid function following axillary nerve lesion injury. Of these 17 had a partial or complete recovery, leaving 26 cases of permanent paralysis. A total of 15 cases with complete permanent paralysis of the deltoid regained full active range of movement with good shoulder function. One case returned to professional football and another to collegiate football. Once again however there is no suggestion of a possible mechanism for the excellent return of shoulder function in these cases.

One of the leading textbooks on the shoulder is acknowledged to be Rockwood & Matsen's The Shoulder [2]. In the third edition the authors note that:

"Young patients may be able to compensate for complete deltoid paralysis and can often perform activities of daily living with only partial disability."

Once again, no explanation is given but they do note that:

"The shoulder can easily maintain a full range of motion with an intact rotator cuff."

These sentences are repeated in the latest edition of the book.

They do not make it clear whether the intact rotator cuff is necessary for there to be a full range of movement or whether they feel that the intact cuff accounts for the recovery of a full range of movement, which seems highly unlikely.

Figure 1 shows the typical appearance of a patient with an axillary nerve lesion and deltoid wasting and prominence of the greater tuberosity of the humerus.



Figure 1: Patient with axillary nerve lesion showing deltoid wasting and prominence of the greater tuberosity of the humerus.

Anatomy of the Axillary Nerve

Reviewing the anatomy, the axillary nerve is a branch of the posterior cord of the brachial plexus which passes backwards through the quadrilateral space together with the posterior humeral circumflex artery as shown in **Figure 2.** The space is bounded superiorly by the teres minor muscle, inferiorly by the teres major, medially by the long head of triceps and laterally by the humeral shaft.

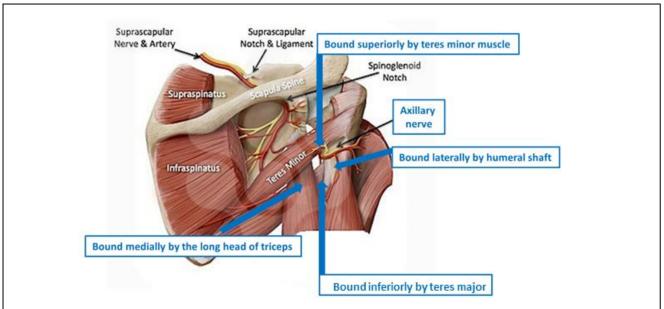
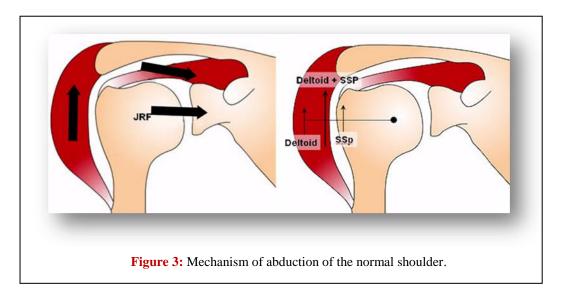


Figure 2: Anatomy of the shoulder showing the axillary nerve and anatomy that bounds the quadrilateral space [adapted from shoulderdoc.co.uk].

Mechanism of abduction

Abduction in the normal shoulder, involves contraction of both the deltoid and supraspinatus such that, as the arm is abducted, the resultant force is directed towards the glenoid. The action of supraspinatus compresses the head of the humerus against the glenoid and maintains stability of the joint while the arm is abducted by the deltoid muscle (Figure 3).



Explanation

So, how can this phenomenon of full range of shoulder movement, with very good power, in the presence of complete axillary nerve damage with deltoid wasting, be explained?

The suggestion is made that deltoid function is taken over by the long head of biceps. Experienced shoulder surgeons occasionally note at operation a biceps tendon that is three to four times its normal size.

This hypothesis can be supported in a patient with deltoid paralysis with a reasonable, but not full, return of function using a three-step test: (Figure 4).

- ➤ With his elbow flexed to 90° and with his forearm in full supination (which shortens the biceps and weakens its efficiency) he was only able to abduct to 90° (Figure 4a).
- ➤ With his elbow flexed and his arm pronated (which slightly lengthens the biceps and increases its efficiency) and he was then able to abduct to 115° (Figure 4b).
- ➤ With his elbow extended (further increasing efficiency) the patient was able abduct his shoulder to 135° (Figure 4c).

And these findings could be readily reproduced.

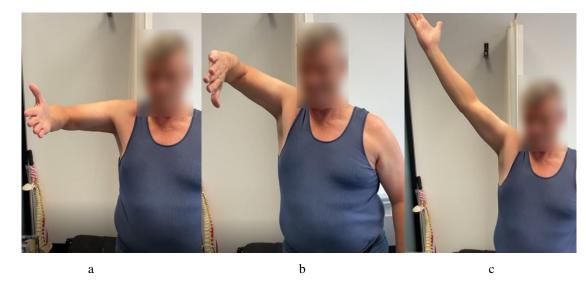


Figure 4: a) Abduction to 90° with forearm in full supination; b) Abduction to 115° with forearm in pronation; c) Abduction to 135° with elbow extended.

Conclusion

These findings strongly suggest that the long head of biceps can compensate for the absent deltoid and that this explains the excellent shoulder function that can occur in cases of permanent deltoid paralysis.

References

- 1. Galvin JW, Eichinger JK. Outcomes Following Closed Axillary Nerve Injury: A Case Report and Review of the Literature. Mil Med. 2016;181(3):e291-7.
- 2. Rockwood CA, Matsen FA, Wirth M, Lippitt, Fehringer EV, Sperling SB. Rockwood & Matsen's The Shoulder, 3rd Edition. 2016.

Citation of this Article

Pillemer R. Axillary Nerve Lesions with Deltoid Paralysis. Mega J Case Rep. 2023;6(12):2001-2005.

Copyright

© 2023 Pillemer R. This is an open-access article distributed under the terms of the <u>Creative Commons</u> <u>Attribution License (CC BY)</u>. The use, distribution or reproduction in other forums is permitted, provided the original author(s) or licensor are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms.