LETTER TO THE EDITOR

Letter to the Editor regarding Westphal T et al: “Axillary nerve lesions after open reduction and internal fixation of proximal humeral fractures through an extended lateral deltoid-split approach: electrophysiological findings”

To the Editor:

We read with much interest the article, “Axillary nerve lesions after open reduction and internal fixation of proximal humeral fractures through an extended lateral deltoid-split approach: electrophysiological findings” by Westphal et al.1

The goal of this study was to determine the frequency of axillary nerve lesions resulting from the operative treatment of proximal humeral fractures using a locking plate through a deltoid split approach. In this retrospective study, neurologic and electrophysiological examinations were performed in 40 of the 76 patients to detect axillary nerve lesions. We have the following comments to the article:

1. We question the authors’ statement that the nerve lesions were caused by the operation.
2. The percentage of nerve lesions found is low compared with the literature. This raises questions about the timing and quality of the electrophysiological examination.
3. The authors’ statement that axillary nerve lesions are rare in proximal humeral fractures is not supported by any evidence.
4. We do agree with the statement that clinical examination alone falls short in detecting axillary nerve lesions.

Several questions were raised after we read this article:

1. Were the fractures caused by a low- or high-velocity trauma?
   To understand the nature of the trauma population studied, knowing what kind of trauma we are dealing with is important. In high-energy traumas, lesions of the brachial plexus, including complete axillary nerve lesions, can be seen. Nerve lesions in low-energy traumas are more likely to occur in the cords of the plexus (distally) or in the deriving nerves.2,9

2. When was the electrophysiological examination performed?
   According to the classification of Seddon,6 neurapraxia is a conduction velocity disorder that recovers within 4 weeks and thus might be missed when the investigation is performed after 4 weeks. As we know from studies about this topic,2,3,10 most of these lesions can be classified as axonotmesis, with spontaneous recovery within several months depending on its severity. Denervation cannot be seen before 3 weeks, so an electromyelogram is only of value after that time. On the one hand, an investigation that is too early can miss the injury. On the other hand, the probability to detect the injury and its severity diminishes as time passes.

3. How was the clinical examination performed?
   The clinical examination can be misleading concerning axillary nerve lesions in these traumas.10 In complete lesions (ie, ruptures of the more proximal part of the nerve), nerve injury can be easily detected by testing the sensibility of the lateral skin of the upper arm (dysesthesia). Partial lesions of the axillary nerve are more commonly located in the anterior branch because of its close relationship to the humeral shaft and its vulnerability in traumas with the arm abducted and rotated. No dysesthesia is found in these cases because the sensory branch, deriving from the posterior branch of the axillary nerve, is still intact.

4. How did the authors distinguish between injury caused by the trauma or by the operation?
   The authors performed a needle examination of the posterior and anterior part of the deltoid. However, no differentiation in outcome is described between the denervation of the parts of the deltoid muscle, which could have helped differentiate between lesions of the posterior or the anterior branch of the axillary nerve.

Several studies have investigated the frequency of lesions of the axillary nerve caused by shoulder trauma.1,3,5,7,9 In a prospective follow-up study, a frequency of 72% was found in displaced fractures (according to the Neer classification). The present study lacks a preoperative electrophysiological examination, so it cannot be
excluded that a number of the nerve lesions already existed preoperatively. Finally, we fully agree with the conclusion that the percentage of permanent axillary nerve lesions in your cohort is higher than expected based on the clinical examination and that an electrophysiological assessment is more sensitive than clinical evaluation in the detection of axillary nerve lesions in shoulder trauma or surgery. Both findings can be easily explained by and supported from the existing knowledge about nerve lesions in these shoulder traumas.

Disclaimer

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