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Axillary motor nerve conduction study: Description of technique and provision of normative data

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ABSTRACT

Background: Axillary nerve lesions can commonly occur secondary to trauma or brachial plexopathy. Our aim was to describe our technique of axillary nerve motor conduction studies and provide the respective normal values.

Methods: Active electrode was positioned over the most prominent portion of the middle deltoid, approximately 5–7 cm distal to the acromion. Reference electrode was positioned over the acromion. Ground electrode was placed between the active and the reference electrodes. Supramaximal stimulation was at the Erb's point.

Results: A total of 154 participants (61% male, age range 18–84) were included. There was a significant positive correlation between the subjects' age and the onset latency (Spearman's rho 0.312, $p < 0.001$) and a significant negative correlation between the participants' age and the CMAP (Spearman's rho -0.481, $p < 0.001$).

For the total male population the lower normal value for the CMAP was 7.6 mV and the higher normal value for the onset latency was 5.0 ms. For the total female population the respective normal values were 6.5 mV and 3.5 ms.

In order to detect an axillary nerve lesion, asymmetry of > 40% in the CMAPS between the symptomatic and the asymptomatic side show a sensitivity of 95.2% and a specificity of 96.6%.

Conclusion: We described our technique of axillary nerve motor conduction studies and provided the respective normal values stratified for age and gender. When suspecting an axillary nerve lesion it is always worth performing axillary motor NCS bilaterally and compare the CMAPs.

1. Introduction

The axillary nerve originates from the brachial plexus (upper trunk, posterior division, posterior cord) and arises in the axilla. The fibers are derived from the fifth and sixth cervical ventral rami (C5 and C6 motor roots). The axillary nerve divides into anterior and posterior terminal division. The former innervates the middle and anterior fibers of the deltoid muscle when the latter supplies the teres minor muscle and the posterior fibers of the deltoid (Leis and Schenk, 2013).

An axillary nerve lesion can commonly occur secondary to trauma (Berry and Bril, 1982). Fracture of the surgical neck of the humerus and inferior dislocation of the humerus at the shoulder joint are common traumatic causes of axillary nerve lesion (Cutts et al., 2009). Neuralgic amyotrophy (idiopathic brachial plexopathy) is another well-recognized cause of axillary nerve lesion (van Alfen, 2011).

The aim of our paper is to describe our technique for axillary nerve motor conduction studies (NCS), provide normative data and determine the extent of asymmetry in the NCS between the two sides, that should be considered as abnormal.

2. Methods

2.1. Participants

Data were collected from all consecutive patients with suspected axillary nerve palsy, secondary to shoulder dislocation or suspected idiopathic brachial plexopathy, who were referred for neurophysiological studies to our department. All patients had NCS bilaterally and needle electromyographic (EMG) examination of the deltoid muscle on the symptomatic side. The NCS from the asymptomatic side were used to establish the normal values. The normal inter-side variability of the compound motor action potential (CMAP) and the onset latency (OL) were calculated in a sub-group of patients who had normal EMG findings of the deltoid muscle.

Exclusion criteria were (1) having history of any form of polyneuropathy (Zis et al., 2016, 2017a), (2) suffering from diseases

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associated with polyneuropathy, such as diabetes mellitus, renal failure and cancer (Zis et al., 2017b, 2017c), (3) receiving or having received neurotoxic medication, and (4) having experienced a shoulder dislocation or brachial plexopathy on the asymptomatic side in the past.

The local Ethics Committee has confirmed that no ethical approval is indicated given that all investigations were clinically indicated and did not form part of a research study.

2.2. Description of the technique

Electrophysiological examinations were performed using a Nicolet EMG machines and Viking select software.

Active electrode was positioned over the most prominent portion of the middle deltoid, approximately 5–7 cm distal to the acromion. Reference electrode was positioned over the acromion. Ground electrode was placed between the active and the reference electrodes. Supramaximal stimulation was at the Erb's point. The following settings were also applied; filter 3 Hz–10 kHz, stimulus duration 0.2 ms (up to 1 ms where needed), stimulus intensity (up to 100 mA) sweep speed 5 ms (0.5 ms/division).

Base-to-peak CMAP and onset latency (OL) were obtained.

2.3. Statistical analyses

A database was developed using the Statistical Package for Social Science (version 23.0 for Mac; SPSS). Frequencies and descriptive statistics were examined for each variable.

The distribution of variables was tested for normality using the Kolmogorov–Smirnov test. Assumption of normality was met in all variables apart from the axillary nerve CMAP and the OL.

We calculated the absolute difference of the CMAP amplitude and the OL between the two sides and we then converted it to a percentage, using the following formulas: CMAP decrease = (difference between the CMAPs/ higher CMAP) and OL increase = (difference between the OL/ lower OL).

Comparisons between groups were made by using the Student's *t*-test for the normally distributed continuous variables and by using the Mann-Whitney's *U* or test for the non-normally distributed continuous variables. Spearman correlation coefficients were calculated for non-parametric variables.

The 5th percentile was used to determine the overall lower normal value for the CMAP and the 95th percentile was used to determine the overall higher normal value for the OL.

Receiver operator characteristics (ROC) analysis was conducted to assess the utility of the inter-side CMAP decrease and OL increase to distinguish the diagnosis of an axillary nerve lesion and establish the optimum cut-off on each occasion. Area under the curve (AUC) and its 95% confidence intervals (CI) for the ROC curve were calculated. The AUC is a measure of the diagnostic power of the test, independent of cut-off points. An AUC < 0.60 is considered "negative", 0.61–0.80 as "doubtful", 0.81–0.90 as "good" and > 0.91 as "very good" (Altman, 1999). The Youden Index was calculated as the sum of sensitivity plus specificity minus 1 for all possible cut-off points to identify the most relevant cut-off values (Youden, 1950).

A value of $p < 0.05$ was considered to be statistically significant.

3. Results

3.1. Demographics and normative data

A total of 154 participants (61.0% male) met the above mentioned inclusion criteria. The ages ranged from 20 to 84 years (mean 50.2 ± 15.7 years). Comparisons between males and females showed no statistically significant differences regarding age (49.7 ± 16.2 versus 51.0 ± 15.0 respectively, p = 0.616). However, males showed significantly higher CMAP in comparison to females (12.8 ± 3.7 versus 11.3 ± 3.6 mV, p = 0.016) and significantly prolonged OL (3.2 ± 0.9 versus 2.8 ± 0.5 ms, p = 0.001).

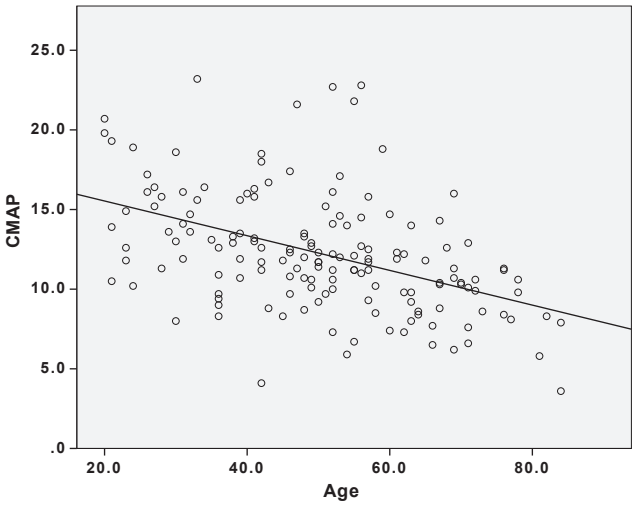

Fig. 1a. Scatterplot and superimposed regression line of relationship between base-to-peak CMAP (in mV) and age (in years). CMAP, compound motor action potential.

There was a significant positive correlation between the subjects' age and the OL (Spearman's rho 0.312, $p < 0.001$) and a significant negative correlation between the participants' age and the CMAP (Spearman's rho −0.481, $p < 0.001$). Scatter plots with superimposed regression lines of relationship between CMAP and age and also between distal latency and age are shown in Figs. 1a and 1b.

Table 1 summarizes the CMAP and OL normal values of axillary NCS, stratified by age group and gender. For the total male population the lower normal value for the CMAP, based on the 5th percentile is 7.6 mV and the higher normal value for the OL, based on the 95th percentile is 5.0 ms. For the total female population the lower normal value for the CMAP, based on the 5th percentile is 6.5 mV and the higher normal value for the OL, based on the 95th percentile is 3.5 ms.

3.2. Inter-side variability

Eighty-seven patients (56.5%) did not have EMG abnormalities in the deltoid and these data were used to establish the normal inter-side variability in the CMAP and the OL. The mean inter-side variability for the CMAP was 16.0 ± 11.0%, ranging from 0% to 58.4% and the mean inter-side variability for the OL was 26.0 ± 26.4%, ranging from 0% to

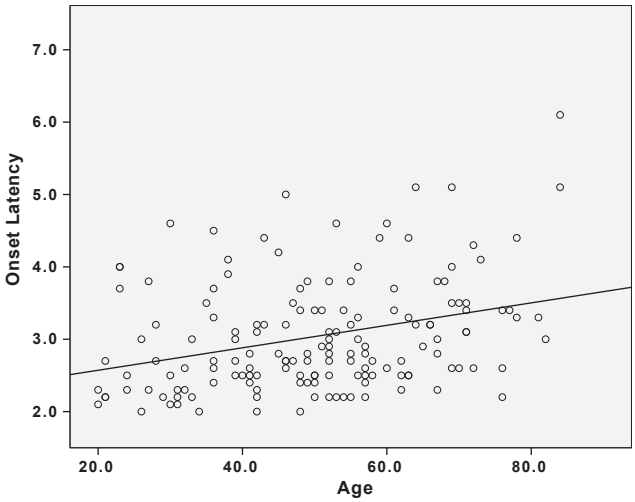

Fig. 1b. Scatterplot and superimposed regression line of relationship between onset latency (in ms) and age (in years).