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Deltoid muscle contribution to shoulder flexion and abduction strength: an experimental approach

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Background: The rotator cuff (RC) and the deltoid muscle are 2 synergistic units that enable the functionally demanding movements of the shoulder. A number of biomechanical studies assume similar force contribution of the force couple (RC and deltoid) over the whole range of motion, whereas others propose position-dependent force distribution. There is a lack of in vivo data regarding the deltoid's contribution to shoulder flexion and abduction strength. This study aimed to create reliable in vivo data quantifying the deltoid's contribution to shoulder flexion and abduction strength throughout the range of motion.

Methods: Active range of motion and isometric muscle strength of shoulder abduction and flexion in 0°, 30°, 60°, 90°, and 120° of abduction/flexion as well as internal and external rotation in 0° and 90° of abduction were obtained in 12 healthy volunteers on the dominant arm before and after an ultrasound-guided isolated axillary nerve block. Needle electromyography was performed before and after the block to confirm deltoid paralysis. Radiographs of the shoulder and an ultrasonographic examination were used to exclude relevant shoulder pathologies.

Results: Active range of motion showed a minimal to moderate reduction to 94% and 88% of the preintervention value for abduction and flexion. Internal and external rotation amplitude was not impaired. The abduction strength was significantly reduced to 76% at 0° ($P = .002$) and to 25% at 120° ($P < .001$) of abduction. The flexion strength was significantly reduced to 64% at 30° ($P < .001$) and to 30% at 120° ($P < .001$) of flexion. The strength reduction was linear, depending on the flexion/abduction angle. The maximal external rotation strength showed a significant decrease to 53% in 90° ($P < .001$) of abduction, whereas in adduction no strength loss was observed ($P = .09$). The internal rotation strength remained unaffected in 0° and 90° of abduction ($P = .28$; $P = .13$).

Conclusion: The deltoid shows a linear contribution to maximal shoulder strength depending on the abduction or flexion angle, ranging from 24% in 0° to 75% in 120° of abduction and from 11% in 0° to 70% in 120° of flexion, respectively. The overall contribution to abduction strength is higher than to flexion strength. The combination of deltoid muscle and teres minor contributes about 50% to external rotation strength in 90° of abduction. The internal rotation strength is not influenced by a deltoid paralysis. This study highlights

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the position-dependent contribution of the shoulder muscles to strength development and thereby provides an empirical approach to better understand human shoulder kinematics.
Level of evidence: Basic Science Study; Kinesiology

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The merging rotator cuff (RC) muscles and the deltoid muscle are 2 position-dependent synergistic units that enable the functionally demanding movements and the dynamic stabilization of the shoulder.[6] Some biomechanical studies assume similar force contribution of the ''abduction'' force couple (RC and deltoid) over the whole range of motion,[2,3] whereas others propose position-dependent force distributions.[20] Biomechanical investigations show angle-dependent moment arms and a greater activation of the deltoid during load compared with the supraspinatus.[23] This might lead to the conclusion that the deltoid contributes more to shoulder abduction and flexion strength and that the RC mainly centers the humeral head in the glenoid.[10] Data about the quantitative effect of the deltoid muscle contribution on the movement of the glenohumeral joint are sparse. Colachis et al[3] described a 35%-80% loss of abduction strength after selective blockade of the axillary nerve. Gerber et al,[5] on the other hand, found a loss of abduction strength of 73%-86% after blockade of the suprascapular nerve. These results are partly contradictory but might be explained by different techniques that were used to record the strength. Colachis et al[3] used a dynamometer that was held by the examiner and pressed against the subject's force, whereas Gerber et al[5] used a device that was fixed to a table or wall. We believe that an in vivo investigation before and after an axillary nerve block similar to the work of Colachis et al but using the same recording device that was used in the study by Gerber et al and with a larger number of volunteers would improve comparability and add relevant knowledge. The quantitative data obtained in this study could also be used to improve computational models, which in the future may help to predict shoulder function after injury or surgery. We hypothesize that the deltoid muscle has a joint position–dependent impact on the maximal shoulder strength in flexion and abduction that increases with an increasing flexion/abduction angle and that its impact on external and internal rotation is only marginal.

Materials and methods

Muscle strength measurements of the deltoid muscle were obtained in 12 healthy volunteers on the dominant arm before and after isolated ultrasound-guided axillary nerve block. Needle electromyography (EMG) was performed before and after the block to assess the neurologic status.

Inclusion criteria were healthy volunteers of both sexes between 18 and 65 years old.

Exclusion criteria were any reported major trauma, previous shoulder dislocation or fracture involving both shoulders, motor or sensory abnormalities, as well as rotator cuff tears on ultrasonography and significant arthritis on radiographs.

General exclusion criteria were severe coagulopathy or intake of anticoagulants, history of alcohol abuse or the intake of psychotropic drugs, pregnancy or breast feeding, infection at the injection site or a systemic infection, and any neuromuscular disorders. Moreover, we excluded subjects with any comorbidity that could interfere with this study (eg, stroke, pacemaker, and cardiac disease) and with contraindications for peripheral regional anesthesia or local anesthetics used in the study (eg, known hypersensitivity or allergy to the used class of drugs).

Radiologic assessment

In order to exclude relevant deformities, arthritis, or rotator cuff pathologies, we performed shoulder radiography in 3 planes and ultrasonography of the rotator cuff carried out by a musculoskeletal radiologist before conducting the experiment.

Axillary nerve block

Standard noninvasive monitoring and a peripheral venous access were obtained for all volunteers.

The axillary nerve block was performed with the participant in the sitting position using ultrasonography (SonoSite SII; FujiFilm SonoSite, Inc., Bothell, WA, USA) with a 6- to 13-MHz linear probe and nerve stimulation (Stimuplex HNS 11; B. Braun Melsungen AG, Melsungen, Germany) with a setting of 0.5 mA current intensity, 0.1 ms impulse duration, and 2 Hz impulse frequency for double guidance. We used the technique to block the nerve selectively as described by Rothe et al.[18]

The ultrasound probe was placed parallel to the longitudinal axis of the shaft of the humerus and approximately 2 cm below the posterolateral corner of the acromion on the dorsal side of the arm. Then we identified the surgical neck and the shaft of the humerus and the cross section of the posterior circumflex humeral artery. The axillary nerve is located cranially to and in close relation to the posterior circumflex humeral artery.

Using an out-of-plane approach, we inserted a 90-mm insulated, short-bevel needle (UPC 90; RM Temena GmbH, Felsberg, Hesse, Germany) from the cranial end of our probe and placed the needle tip cranial to the posterior circumflex humeral artery under the muscle fascia caudal to the teres minor muscle. We slowly injected 20 mL of mepivacaine 1%. The location of the axillary nerve block at the common trunk of the axillary nerve is proximal to the teres minor branch, which consistently originates from the

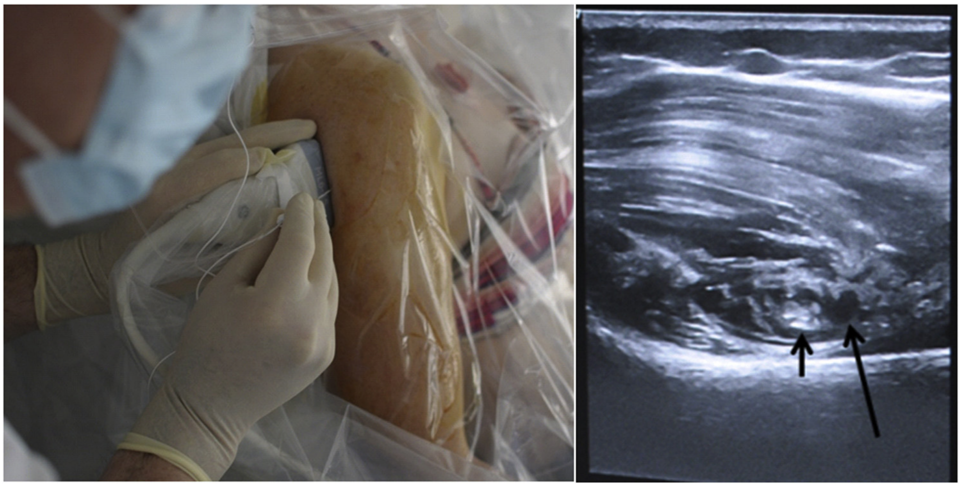

Figure 1 Execution of the ultrasound controlled axillary block. The volunteer is lying in beach chair position, and the anesthetist is standing behind. The ultrasound probe is held vertically. The ultrasonographic image on the right shows the axillary nerve (→) in a short axis view directly cranial to the posterior circumlex humeral artery (⟶) at the surgical neck of the humerus.

posterior division of the axillary nerve or the common trunk.[14] Therefore, the teres minor muscle was always blocked, too. Block success was evaluated assessing the degree of sensory block (cold test) over the distribution area of the axillary nerve every 5 minutes until anesthesia was present in the innervated skin area. After a clinically complete axillary nerve block was assumed, electrophysiological testing was performed as outlined in the next section. Execution of the axillary nerve block is demonstrated in Figure 1.

Needle EMG recordings

Needle EMG recordings (Dantec Keypoint software 2.0, Skovlunde, Denmark) of the 3 parts of the deltoid muscle as well as supra- and infraspinatus muscles were performed before and 30 minutes after the axillary nerve block. This test specifically examined for spontaneous activity to exclude nerve pathology and determine maximal voluntary activation (ie, interference pattern analysis).

Interference patterns during maximal voluntary contraction were recorded and compared between the 2 time points. Paralysis was rated successful if full extinction of the deltoid EMG response was obtained during maximal voluntary contraction. In order to rule out inadvertent paralysis of the supra- and infrascapular nerves due to aberrant fiber course or diffusion of the anesthetic agent, EMG recordings were obtained from the supra-/infraspinatus muscles to show that recruitment was normal after the anesthetic axillary block.

Strength measurements

Strength measurements were carried out before and after axillary nerve block on both arms. The measurements after axillary nerve palsy were started 45 minutes after the block was set. Strength testing (Fig. 1) was conducted with an electronic isometric strength dynamometer (IsoForceControl V1.1; Medical Device Solutions AG, Oberburg, Switzerland) that has been shown to produce validated strength results in healthy adult volunteers with an anesthetic nerve block.[5] First, the active shoulder movement against gravity was documented for abduction, flexion, external rotation, and internal rotation. For abduction and flexion measurements, the volunteers were positioned with the back to a wall so that the heels and the scapula touched it, the arms straight at the side with the thumbs facing forward. Then active abduction and flexion was performed and the measurement was carried out with a standard goniometer. For external rotation measurement, a 90° flexion in the elbow was added and the thumb was facing up. For measuring internal rotation, the subject was positioned directly facing a wall to avoid bending forward, and internal rotation to the back with the thumb up was actively done. The height of the thumb on the back was documented. Elevation and abduction strength was tested in 0°, 30°, 60°, 90°, and 120° of flexion/abduction in standing position. While testing elevation, the subject was positioned with the back exactly perpendicular to the axis of the measuring device, and the isometric testing was carried out exactly in the sagittal plane. For abduction testing at 0° and 30°, the loop was elongated so that the dynamometer could be attached to the contralateral side, and from 60° to 120°, the dynamometer was attached to a table at the ipsilateral side and tests were carried out exactly in the coronal plane. External and internal rotation strength was tested in the standing position with a 90°-bent elbow and the arm in neutral position (dynamometer fixed to a wall on the side and isometric testing in the horizontal plane) as well as in 90° of abduction. For external rotation testing in the latter position, the device was attached to a table directly under the subject's wrist and the direction of force was in the coronal plane. The measurements of internal rotation in 90° of abduction were conducted with the patient lying on the back and the device being attached to the wall behind on wrist height. All angles were determined with a standard goniometer and maintained by adjusting the loop of the dynamometer to the wrist of the patient. Retroversion strength was tested with the arm on the side and the palm facing forward, and the dynamometer was attached to a wall in front of the subject at wrist height. A minimum force of 10 N was necessary to start the electronic measurement. All

Figure 2 Exemplary presentation of some measurement positions. The measuring device was attached to the wall or a table and the angle of the unit was always chosen so that the pulling force was perpendicular. **a**) 90° of abduction; **b**-**d**) 0°, 30°, and 90° of flexion.

measurements were recorded 3 times and were obtained during a 5-second measuring cycle with 30 seconds of rest between each testing. The resultant force was documented as the mean force in newtons obtained during each cycle. To avoid fatigue, testing was performed alternately on both arms for every joint position, giving the other arm an adequate break of 2 minutes. Figure 2 shows some examples of the measurement positions.

All volunteers were contacted the day after the block by the anesthetist and questioned regarding full recovery of sensory and motor function.

Statistics

The 3 test repetitions were averaged for subsequent analysis. Then the relative values (in percentage) of postintervention strength normalized to preintervention strength was computed. As normal distribution was not fulfilled by the data, nonparametric testing was applied. An exact Wilcoxon signed-rank test comparing preintervention values with postintervention values for both sides was used. Ordinary least squares linear regression was used to examine associations between strength and testing angle with

Table I Demographics (N = 12)

	Median (range) or n (%)
Female sex	6 (50)
Age, yr	25 (21-40)
Weight, kg	69 (55-87)
Height, m	1.75 (1.68-1.86)
Right-handed	10
Left-handed	2

R-squared test adjustment for goodness of fit. Statistical analyses were conducted using SPSS, version 26.0, for Windows (IBM, Armonk, NY, USA). If not otherwise specified, median and range are reported. Statistical significance was set at $\alpha = 0.05$.

Results

We included 12 healthy volunteers with an equal number of male and female participants. The median age was 25 years (21-40), the weight 69 kg (55-87), and the height 1.75 m (1.68-1.86), and 10 participants were right-handed. Demographics are given in Table I.

In all volunteers, complete paralysis of the 3 parts of the deltoid muscle could be electromyographically confirmed. In 2 subjects, the initial anesthetic block was incomplete (ie, preserved EMG activity in the deltoid muscle). In these cases, a proximal bifurcation of the axillary nerve was shown by ultrasonography. An additional neurolysis of the proximal branches then resulted in complete deltoid paralysis in these subjects. Supra- and infraspinatus muscle activity was unaffected in all subjects.

Active range of motion without any additional weight showed a significant reduction to 94% ($P = .004$) and 88% ($P < .001$) of the preintervention value for abduction and flexion, respectively, on the intervention side (Fig. 3, *a*). Internal and external rotation was not impaired ($P = .25$, $P = .69$).

The maximal abduction strength showed lower values compared with preintervention values, ranging from 76% (50%-107%) at 0° of abduction to 25% (0%-47%) at 120° of abduction. The reduction was significant over the whole range of motion between 0° and 120° ($P = .002$ at 0°; $P < .001$ at 30°-120°; Fig. 3, *b*).

In contrast to the abduction, forward flexion showed no significant reduction at the starting position of 0° with a median strength of 89% (62%-125%) of preintervention values ($P = .12$). From 30° to 120° of flexion, a significant strength reduction ($P < .001$) to 64% (at 30°) and 30% (at 120°) of the initial values was observed (Fig. 3, *c*).

The relative reduction in maximum strength appears to have a linear dependence on the flexion angle as well as the abduction angle with a strength decrease of 0.50% (standard error: 0.05%, R^2-adjusted: 0.669) and a 0.38% (standard error: 0.05%, R^2-adjusted: 0.509) per degree flexion or abduction according to an ordinary least squares linear regression, respectively.

The internal rotation strength showed no significant reduction in 0° ($P = .28$) and 90° ($P = .13$) of abduction (Fig. 3, *d*).

The maximal external rotation strength showed a trend ($P = .09$) to decrease to 84% in 0° of abduction and a significant decrease ($P < .001$) to 53% in 90° of abduction (Fig. 3, *e*).

Finally, a significant reduction ($P < .001$) to 64% was observed regarding the retroversion strength on the intervention side (Fig. 3, *f*). A subgroup analysis did not reveal any difference with gender (data not shown).

The control side showed no changes in range of motion or strength during all pre- and post-intervention tests. All the obtained data are summarized and clearly displayed in Table II.

Discussion

This experimental study in healthy volunteers shows a systematic reduction of abduction and flexion strength with a linear dependence on joint position following selective neurolysis of the axillary nerve. The loss of strength was greatest at 120° of abduction and flexion. Internal rotation strength was not impaired, whereas external rotation showed a significant strength reduction at 90° of abduction.

This confirmed our hypothesis that the deltoid muscle's contribution to maximal shoulder strength in flexion and abduction depends on the joint position and increases with increasing flexion/abduction. Furthermore, we assumed that the effect on external and internal rotation is only marginal. We were able to generally confirm our hypothesis with the exception that external rotation showed a significant loss of strength in 90° of abduction, which might strongly be related to the additional palsy of the teres minor muscle. Although not readily verifiable by EMG because of methodologic challenges with potential cross-talk arising from the infraspinatus muscle,[15] we assume a concurrent paralysis of the teres minor muscle due to the ultrasonographically verified anesthetic block proximal to the teres minor branch of the axillary nerve.[14]

Clinical data regarding the deltoid's contribution to shoulder function is sparse, and at times contradictory. In 1969, Colachis et al[3] performed axillary nerve blocks on 5 adult male students and found a force loss of 30%-60% for abduction strength measured in 0°-150° of abduction, which showed a stable plateau between 60° and 120°. For flexion, a force loss of 35%-80% was described, with a plateau at 60% of force loss between 60° and 120° of flexion.[3]

Our study revealed a more linear force loss of 24%-75% for abduction strength measured in 0°-120° of abduction with no plateau visible. The same was found for flexion strength, with a linear force loss of 11%-70% with

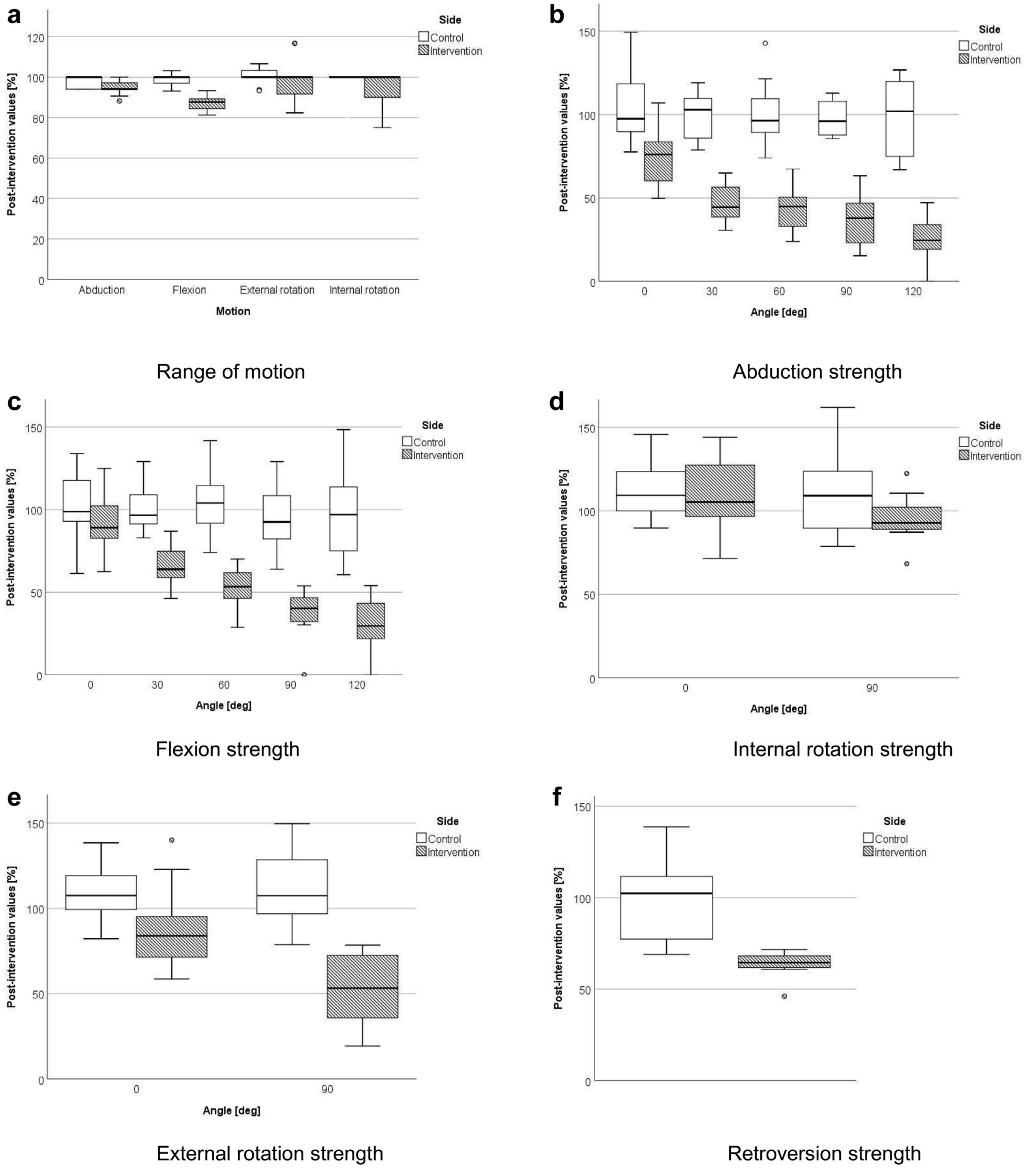

Figure 3 Post-intervention values (in percentage) normalized to preintervention values: **a**) range of motion, **b**) abduction, **c**) flexion, **d**) internal rotation, **e**) external rotation, and **f**) retroversion strength. Medians are represented by the *bold black horizontal line*. The *white* boxplots show the control side values whereas the striped ones stand for the intervention side. When testing was done in different positions, the abduction/flexion angle is given.

increasing flexion angles. In our opinion, the plateau phase described by Colachis et al[3] may be a result of vulnerable methodology. For example, the magnetic scale used to assess the subject's force was held by an examiner, which presumably made measurements prone to motion artifacts. In contrast, our measurement device was always fixed to a table or wall, giving the most stable position possible. Furthermore, the linearly increasing loss of strength with increasing abduction and flexion found in our study is more plausible than the plateau described previously, because the

Table II Postintervention values (in percentage) normalized to preintervention values regarding range of motion and strength measurements (N = 12)

Motion	Dominant arm		Nondominant arm	
	Median (range)	*P* value*	Median (range)	*P* value*
Range of motion, %				
Abduction	94 (88-100)	**.004**	100 (94-100)	.06
Flexion	88 (50-93)	**<.001**	100 (93-103)	.25
Internal rotation	100 (75-100)	.25	100 (80-100)	>.99
External rotation	100 (82-117)	.69	100 (93-114)	.44
Maximal abduction force, %				
0° of abduction	76 (50-107)	**.002**	98 (78-149)	.79
30° of abduction	44 (31-65)	**<.001**	103 (79-119)	.81
60° of abduction	45 (24-67)	**<.001**	96 (74-143)	.93
90° of abduction	38 (15-63)	**<.001**	96 (86-113)	.42
120° of abduction	25 (0-47)	**<.001**	102 (67-127)	.91
Maximal flexion force, %				
0° of flexion	89 (62-125)	.12	99 (61-184)	.85
30° of flexion	64 (46-87)	**<.001**	97 (83-129)	.53
60° of flexion	53 (29-70)	**<.001**	104 (74-142)	.64
90° of flexion	40 (0-54)	**<.001**	93 (64-129)	.34
120° of flexion	30 (0-54)	**<.001**	97 (61-148)	.57
Maximal internal rotation force, %				
0° of abduction	105 (72-144)	.28	109 (90-146)	.06
90° of abduction	93 (68-122)	.13	109 (79-162)	.34
Maximal external rotation force, %				
0° of abduction	84 (59-140)	.09	108 (82-138)	.08
90° of abduction	53 (19-79)	**<.001**	107 (79-150)	.15
Maximal retroversion force, %	64 (46-93)	**<.001**	102 (69-139)	>.99

* Wilcoxon signed-rank test.
Bold values indicate significance level < .05.

moment arm of the supraspinatus shortens linearly with higher flexion or abduction.[1,7,9]

In 2007, Gerber et al published the results of a suprascapular nerve block investigation that showed a loss of abduction strength by 45% if the infraspinatus alone was paralyzed and by 75% if both infra- and supraspinatus were paralyzed. This effect was stable between 30° and 90° of flexion.[5] Consequently, this would suggest that the deltoid contributes only 25% or less to total shoulder strength in this distinct range of abduction. The possible reason for these seemingly contradictory findings is the central role of the rotator cuff, which functions as a pivotal counterbalance to the deltoid force. If the centralizing rotator cuff is not sufficient, the humeral head moves cranially, which significantly shortens the deltoid moment arm and leads to the phenomenon described above.[8,11]

We found a significant strength loss of about 50% for external rotation in 90° of abduction, which partly confirms the findings published by Walch et al.[21] They showed a correlation between degeneration of teres minor and the hornblower sign, which describes the inability to keep the elbow at 90° of abduction with the forearm in a horizontal position. With our study, we cannot determine the exact contribution of the teres minor muscle to external rotation, because the posterior part of the deltoid acts synergistically with the external rotators.[16] The abduction strength showed the greatest force loss beginning already at 0° of abduction, in contrast to the fact that flexion strength was not significantly reduced in this position. These results support the anatomic and biomechanical findings that the middle portion of the deltoid is not only the anatomically largest part but also functionally most important for shoulder abduction over the whole range of motion, whereas flexion seems to be sufficiently supported by the pectoralis major at the neutral position.[22] Although in a cadaveric study, Otis et al[12] found the moment arm of the deltoid to be less than that of the supraspinatus for abduction angles up to 40°, our findings show a significant contribution of the deltoid muscle at such a low angle of abduction. A possible explanation would be that the supraspinatus was found to have a variable contribution to shoulder motion independent of the moment arm. This was found in a study examining 23 volunteers who had to lift the arm isometrically against a force of 30 N. Load was applied alternately at the proximal and distal humerus to generate different moment arms. The deltoid showed an increased activation with increased moment arm, whereas the supraspinatus showed no consistent increase and a high variability between different subjects. The underlying cause for that is not known.[23]

A lot of studies used computed models or cadaveric testing, which neglected or idealized important secondary force generators and shoulder stabilizers and movers such as the pectoralis minor, latissimus dorsi, or teres major.[4] Results of cadaveric studies are to be interpreted with caution, because neuromuscular input and control are missing and therefore in vivo interactions and compensation mechanisms are absent. [9,17,19,25] In our study, those secondary stabilizers were active and not impaired by the axillary nerve block, but an explicit analysis of these muscles was beyond the scope of this investigation.

Interestingly, abduction and flexion range of motion was only reduced by 6% and 12%, whereas the internal and external rotation showed no decreased range of motion. That would explain why acute paralysis of the deltoid leaves unloaded shoulder function almost intact. Colachis et al[3] showed similar results but added that earlier fatigue occurred in the paralyzed shoulder when little load was applied.

Limitations of this study include the additional paralysis of the teres minor, because of the anesthesia technique used. A reliable interpretation of the results regarding external rotation strength is therefore not possible. Moreover, the reported contribution of the deltoid muscle to shoulder function might be underestimated because of compensation mechanisms of the numerous other shoulder muscles. Although, to date, this study represents the most comprehensive assessment of strength measurements after anesthetic axillary neurolysis, the demographic characteristics of the participants (ie, age distribution and body mass index) might not be fully representative of the typical cohorts encountered in clinical practice. Our results simulate an acute axillary nerve palsy; therefore, we cannot make any statement regarding chronic lesions. Adaption of the rotator cuff and secondary shoulder muscles supposedly leads to better strength and function as has been previously described after axillary nerve injury.[13,24]

Conclusion

In this cohort of 12 healthy individuals, the deltoid muscle shows a linear contribution to maximal shoulder strength depending on the abduction or flexion angle, ranging from 11% of total flexion strength at neutral position to 70% at 120° and from 24% at neutral position to 75% at 120° for abduction. The combination of deltoid muscle and teres minor contributes significantly to external rotation in 90° of abduction. This study suggests that uniform assumptions regarding the distribution of power between shoulder muscles in biomechanical models do not reflect in vivo muscle physiology. The arm position in space, the muscle lever arms, and the individual anatomy must be taken into account. This investigation provides in vivo data that can be used in the development of computational models to promote the understanding of shoulder kinematics and biomechanics.

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