RESEARCH ARTICLE

Abnormal Patterns of Biceps and Triceps Co-Contraction Following Elbow Surgery May Result in Elbow Stiffness

Pedro K. Beredjiklian, MD; Amir R. Kachooei, MD, PhD; Greg Gallant, MD; Jack Abboudi, MD; Moody Kwok, MD; Robert Takei, MD; Robert N. Hotchkiss, MD

Research performed at Hospital for Special Surgery in NY, USA and The Rothman Orthopedic Institute, Philadelphia, PA, USA

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Abstract

Objectives: This study examines the pattern of muscular contraction and the intensity of this contraction of the biceps and triceps following elbow surgery.

Methods: We performed a prospective electromyographic study of 16 patients undergoing 19 surgical procedures on the elbow joint. We measured the resting EMG signal intensity of the biceps and triceps of the operated and the normal sides at 90 degrees. We then calculated the peak EMG signal intensity during passive elbow flexion and extension of the operated side.

Results: Seventeen of 19 elbows (89%) displayed a co-contraction pattern of the biceps and triceps near the end of flexion and extension during the passive range of motion. The co-contraction pattern was observed near the end of the range of motion in both flexion and extension. In addition to the observed co-contraction patterns, we detected higher contraction intensities for the biceps and triceps muscles in all patients in both flexion and extension for the elbows, which had been treated surgically. Further analysis suggests an inverse correlation between the biceps contraction intensity and the arc of motion measured at the latest follow-up.

Conclusion: The co-contraction pattern and increased contraction intensity of periarticular muscle groups may result in internal splinting mechanisms, contributing to the development of elbow joint stiffness, which is frequently observed following elbow surgery.

Level of evidence: III

Keywords: Biceps, Co-contraction, Elbow contracture, EMG, Heterotopic Ossification, Instability, Triceps

Introduction

The propensity of the elbow joint for developing capsular fibrosis has been explained in several ways. It includes hematoma formation and subsequent heterotopic ossification in the brachialis muscle, collateral ligaments shortening following immobilization, and articular surface irregularities.[1-4] early elbow range of motion is advocated to prevent capsular fibrosis for the aforementioned reasons. The therapeutic dilemma is the immediate loss of active and passive motion following surgery or contracture release, implying a possible muscle imbalance around the elbow. Even elbows with a full motion under anesthesia may show resistance to physical therapy and continuous passive motion.

Clinically, we have noted abnormal muscle activity in the biceps and triceps muscle groups following injury or surgery to the elbow joint. Abnormal co-contraction of the biceps and triceps may contribute to immediate postoperative or post-traumatic elbow stiffness, interfering with postoperative rehabilitation protocols. Even an aggressive therapy to move the elbow in the presence of muscle co-contraction might result in a vicious cycle of resistance-contracture, which contradicts the advocacy of

Corresponding Author: Pedro K. Beredjiklian, Department of Orthopedic Surgery, Sidney Kimmel Medical College, Thomas Jefferson University, Rhothman Orthopedic Institute, Philadelphia, PA, USA

Email: Pedro.Beredjiklian@rothmanortho.com

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early motion exercise protocols. The dynamic loss of motion because of muscle co-contraction decreases the therapy effectiveness leading to a rigid static loss of motion due to scar formation. While co-contraction firing patterns may be related to normal reflexive and protective mechanisms, they may have a harmful effect on the range of motion. We hypothesized that there would be an abnormal co-contraction pattern of the biceps and triceps muscles postoperatively compared to the normal contralateral elbow.

Materials and Methods

We prospectively assessed the surface electromyography (sEMG) in 16 consecutive patients who underwent elbow joint surgery by the senior author. The average age of the patients was 48 years (range, 21–64 years).The preoperative indications were post-traumatic contracture in eight patients, acute fracture-dislocation of the elbow joint in two, osteoarthritis in two, osteomyelitis in two, distal humerus nonunion in one, chronic valgus instability in one, and rheumatoid arthritis in one patient. Three patients required a second surgical procedure during the study period, two for recurrence of post-traumatic contractures and one for recurrence of deep infection, yielding 19 surgical procedures in the study period. None of the patients underwent a triceps split or triceps turndown approach. The patients were followed for an average of 6 months after the surgical procedure (range, 4–8 months). Approval from the internal review board at our institution was granted, and informed consent was obtained from all patients.

The electrical activity of the biceps/brachialis complex and the triceps muscle groups was recorded simultaneously using noninvasive, surface electromyographic sensors (Amtron, Thought Technology Ltd., Montreal, Canada) at a sampling rate of 1000 Hz [5]. Electrodes were attached to the skin and oriented parallel to the muscle fibers. The electromyographic assessment was performed on the affected extremity and the contralateral, normal extremity, which served as an internal, paired control seven days postoperatively. All measurements were obtained with the forearm in neutral rotation with the upper extremity at the side resting on an arm table. The sEMG data was processed to eliminate any offset, integrated over 40-ms intervals, and rectified a full wave.

The electromyographic assessment was performed by modifying previously described techniques.[6-8] First, the EMG signal of the muscles at rest (R_{EMG}) and during passive range of motion (P_{EMG}) was determined for each patient in the affected and the normal, contralateral extremity. The R_{EMG} for the biceps was established by placing the surface triode 7 cm proximal to the elbow crease anteriorly. The EMG signal at rest (R_{EMG}) was recorded by passively maintaining the elbow at 90° of flexion. Once the rest signal was recorded, the elbow was passively ranged at about 10 degrees per second from the tolerated end extension to the tolerated end flexion and vice versa with the forearm in neutral rotation. The electromyographic signal (P_{EMG}) was recorded during the elbow range of motion. The peak EMG signal in relation to the position of the elbow (start/mid/end range of motion) was assessed qualitatively. The duration of the EMG recording varied in each case depending on the range of motion tolerated by the patient. The elbow was maintained at the end of the range of motion in both flexion and extension until the EMG signal returned to the rest signal.

In addition to measuring the biceps muscle electrical activity, similar measurements were calculated for the triceps muscle. For measurement of the triceps signal, the sensor was placed 7 cm above the elbow joint posteriorly on the triceps, and the rest and passive EMG signals were set as described above for the biceps muscle. As mentioned previously, the occurrence of the peak EMG signal during passive motion with relation to the position of the elbow (start/mid/end range of motion) was recorded, and a comparison of the timing of biceps and triceps EMG activity compared to assess for the presence of co-contraction. The end range of motion in flexion and extension was measured with a standard office goniometer. In all patients, the elbow joint was ranged within the pain limits.

To normalize the signal during the passive motion for both the experimental and normal sides, the maximum isometric voluntary contraction (MVIC) for the biceps and triceps of the normal side was recorded by having the patient actively flex and extend the elbow against resistance in 90 degrees of flexion. The peak ratio of the REMG and MVIC EMG signals was calculated for the experimental side by and the percentages for the biceps and triceps signals in flexion and extension were tabulated and compared between the sides [Table 1]. Statistical analyses were performed using a paired Student's t-test for parametric data (Microsoft Excel, Microsoft Corp., Redmond, WA).

$$Experimental\ Peak\ Ratio = \frac{Experimental\ \text{REMG}}{Control\ MVIC} \times 100$$

$$Noraml\ Peak\ Ratio = \frac{Control\ \text{REMG}}{Control\ MVIC} \times 100.$$

Results

At an average follow-up six months postoperatively (range 4-8 months), the average arc of motion was 89°, with a mean of 29° lack of extension (range, 5°–60°) and 118° of flexion (range, 90°–145°). No adverse effects were observed as a result of the electromyographic testing. None of the patients experienced undue pain during testing the affected and the control elbows.

We identified significantly increased contraction intensity of the biceps muscle compared to controls during passive both flexion and extension. We identified an average peak electromyographic signal of 43% (range, 0-100%) of the control MVIC in the biceps muscle during passive flexion of the post-surgical elbows. In contrast, it was 2% (range 0–12%) of the control MVIC in the normal biceps, and the difference was significant ($P < 0.001$).

During passive extension of the post-surgical elbows, we identified an average peak electromyographic signal in the biceps muscle at 18% (range, 0–25%) of the control MVIC, which was 6% (range, 0–12%) of the control MVIC in the normal biceps. The difference was significant ($P < 0.001$).

We identified similar patterns in the triceps muscle. During passive flexion of the post-surgical elbows, we identified an average peak electromyographic signal in the triceps at 21% (range, 0–38%) of the control MVIC. In

Table 1. Patients' data

Patient No.	Age	Diagnosis	Procedure
1	21	Distal humerus nonunion	ORIF distal humerus nonunion with ICBG
2	43	Elbow fracture-dislocation	LUCL reconstruction; hinged fixator placement
3	64	Degenerative arthritis	Total elbow replacement
4	58	Post-traumatic contracture	Elbow contracture release; hinged fixator placement
5	63	Degenerative arthritis	Total elbow replacement
6	40	Post-traumatic contracture	Elbow contracture release
7	55	Chronic valgus instability	Radial head allograft, LUCL reconstruction
8	51	Post-traumatic contracture	Elbow contracture release
9	32	Post-traumatic contracture	Elbow contracture release
		Recurrence of post-traumatic contracture	Elbow contracture release; hinged fixator placement
10	60	Post-traumatic contracture	Elbow contracture release
11	44	Post-traumatic contracture	Elbow contracture release
12	34	Post-traumatic contracture	Elbow contracture release; hinged fixator placement
13	43	Post-traumatic contracture	Elbow contracture release
		Recurrence of post-traumatic contracture	MUA elbow
14	45	Distal humerus osteomyelitis post ORIF	Hardware removal; I&D elbow
		Recurrence of osteomyelitis	I&D elbow
15	55	Elbow fracture-dislocation	Prosthetic radial head replacement; ligament repair; hinged fixator
16	55	Rheumatoid arthritis	Fascial interposition arthroplasty; hinged fixator placement

ORIF - Open reduction and internal fixation; ICBG - Iliac crest bone graft; LUCL- Lateral ulnar collateral ligament
MUA - Manipulation under anesthesia; I&D - Irrigation and debridement

contrast, it was 5% (range, 0–12%) of the control MVIC in the normal triceps, which was statistically significant ($P < 0.001$). During passive extension of the post-surgical elbows, we identified an average peak electromyographic signal in the triceps at 8% (range, 0–25%) of the control MVIC. In contrast, it was 0% of the control MVIC in the normal triceps, and the difference was statistically significant ($P < 0.001$) [Figure 1].

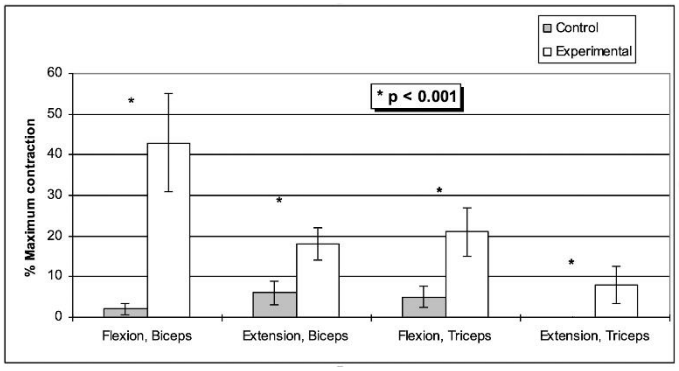

Figure 1. The graph shows the experimental and the control biceps and triceps muscle contraction intensity during passive range of motion of the elbow joint as a percentage of maximum voluntary contraction of the control side

In 17 of 19 elbows (90%), the pattern of increased contraction intensity was observed within the end of the range of motion in both flexion and extension for both the biceps and the triceps. The observed electrical activity yielded a co-contraction pattern of the biceps and triceps at the end of motion during the passive range of motion of the elbow joint. The muscles returned to a relaxed baseline state as the elbow joint was maintained at the end of motion for an average of 6 seconds (range, 3–8 sec) after the onset of the increased contraction intensity. Further analysis suggests an inverse correlation between the biceps contraction intensity and arc of motion measured at the latest follow-up [Figure 2].

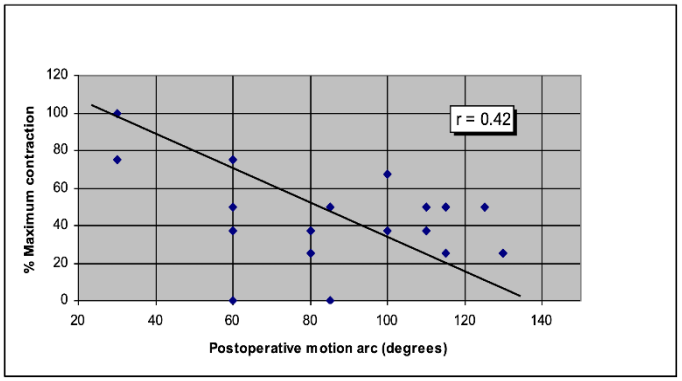

Figure 2. Biceps contraction during passive flexion is shown

Two of the 19 elbows (10%) did not display abnormal muscle firing. We did not detect any electromyographic evidence of excessive muscle activity in the biceps and triceps of these two elbows, both of whom were acute elbow fracture-dislocations, and the measurements were done acutely after the injury. Both of these patients had otherwise normal neurovascular exams.

Discussion

We have noted abnormal muscle activity patterns in the biceps and triceps muscle groups following elbow surgery. For this reason, we decided to examine the patterns of muscular contraction of the biceps and triceps following surgical trauma, and sixteen consecutive patients were recruited for the study regardless of the diagnosis or surgical procedure. Interestingly, we detected firing patterns involving biceps and triceps co-contraction and increased contraction intensities of these muscle groups following surgery to the elbow joint. Specifically, we found that 89% of the elbows under study displayed a pattern of abnormal co-contraction of the biceps and triceps during the passive range of motion. The co-contraction pattern was observed near the end of the range of motion in both flexion and extension. The biceps/triceps co-contraction at the end of passive flexion was sustained for an average of 6 seconds, after which the muscles returned to a relaxed state while the elbow joint was maintained in that position.

Our study showed a correlation between co-contraction and elbow stiffness because co-contraction was more intense in chronic patients but showed less abnormal firing in acute patients. Another speculation is that co-contraction firing patterns probably occur due to the elbow joint's internal, reflexive muscular splinting, and it may contribute to the development of post-traumatic and postoperative contractures.

During surgery, temporary interruption of blood flow to muscles may occur due to the use of tourniquets, surgical technique, stress related sympathetic stimulation,[9] leading to ischemia. Similarly, traumatic injuries to muscles, such as fractures or crush injuries, can also disrupt blood flow and cause ischemia. It is speculated that chronic muscle involuntary contractions and spasm results from ischemia causing spontaneous electrical activity. It is analogous to cardiac muscle ischemia which might result in automatism and arrhythmia. It is hypothesized that chronic muscle spasms and EMG changes result from skeletal muscle micro-ischemia.[10] The probable mechanisms are impairment of the normal functioning of motor neurons and sensory receptors,[11] and disruption of the normal regulation of calcium ions in skeletal muscles, leading to imbalances in intracellular calcium levels. Dysregulation of calcium homeostasis can result in increased sensitivity of contractile proteins to calcium, leading to increased muscle tone, stiffness, and dysregulated muscle contraction.[12] Moreover, muscle relaxation requires energy even more than contraction and ischemia can disrupt the normal metabolic processes within muscles. This leads to impaired muscle relaxation, generation of autonomous signals and muscle contraction.[13] Page and colleagues analyzed and compared the electromyographic activity of the periarticular muscle groups in stiff and normal elbows in ten patients. These authors also identified increased contraction intensities in the muscle groups in stiff elbows compared to normal controls.[14] Osu et al. estimated a linear relationship between the muscle co-contraction around the joint and joint stiffness.[15] Ischemia reduces short latency (SL) EMG reflexes. SL EMG activity in muscles recruit the force responsible for terminating the torque reduction coincident with decreasing stretch velocity.[16]

We speculate that injury to the distal biceps and triceps tendon results in a constant stimulus to elicit deep tendon reflex and contraction, resulting in hyperreflexia. This theory can be supported by studies that used botulinum toxin (Botox) with superior results after contracture release.[17] Moreover, biceps contraction was reduced in stroke patients by eliminating biceps stretch in forearm supination, showing that tendon stretch may contribute to eliciting deep tendon reflex.[18]

Notably, the two patients in the series who were treated acutely for fracture-dislocations of the elbow joint displayed flaccidity of the biceps and triceps muscle groups. No measurable electromyographic signals were detected in these two patients. The absence of electrical activity of these muscles may result from reflex muscular inhibition in the acute post-injury period. It could potentially explain recurrent elbow joint instability following elbow dislocations. While it is challenging to conclude only two patients, it is also possible that the initial inhibition may eventually lead to the abnormal co-contraction patterns observed in the rest of the elbows under study.

Observational studies have described the presence of abnormal periarticular muscle co-contraction in stiff elbows. Page and colleagues analyzed and compared the electromyographic activity of the periarticular muscle groups in stiff and normal elbows in ten patients. These authors also identified increased contraction intensities in the muscle groups in stiff elbows compared to normal controls.[14]

There are several limitations to our study. First, only the biceps and triceps muscle groups were studied. Other muscles which cross the elbow joint may also exhibit abnormal patterns of contraction, which may contribute to the development of contractures. Second, the testing was performed under an ideal extension-flexion passive range of motion. Muscle firing patterns may change according to forearm rotation changes, factors that were not accounted for in this study. Third, it is possible that some of the electromyographic changes. Finally, electromyographic analysis with needle electrodes may render more accurate contraction intensity and timing information. Despite these, we are unaware of any published studies evaluating the patterns of periarticular muscle contracture following surgery on the elbow joint.

Postoperative and post-traumatic elbow contractures often result in substantial functional impairment. Seventeen of 19 elbows in this study displayed a biceps and

triceps co-contraction pattern during passive flexion, independent of pain. In light of these findings, we have altered our routine postoperative rehabilitation protocols, emphasizing passive motion at a slow rate of progression and allowing a period of relaxation after maximum flexion has been achieved. Moreover, forearm supination might help reduce the deep tendon reflex discharge by eliminating tendon stretch. The irregular pattern and increased contraction intensity of periarticular muscle groups may result in internal splinting mechanisms, contributing to postoperative and post-traumatic elbow joint contractures.

Conclusion

The co-contraction pattern and increased contraction intensity of periarticular muscle groups may result in internal splinting mechanisms, contributing to elbow joint stiffness, which is frequently observed following elbow surgery.

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Pedro K. Beredjiklian MD [1]
Amir R. Kachooei MD, PhD [2,3]
Greg Gallant MD [1]
Jack Abboudi MD [1]
Moody Kwok MD [1]
Robert Takei MD [1]
Robert N. Hotchkiss MD [4]

1 Rothman Orthopedic Institute at Thomas Jefferson University, Philadelphia, PA, USA
2 Rothman Orthopedic Institute, Adventhealth, Orlando, FL, USA
3 University of Central Florida, Orlando, FL, USA
4 Division of Hand and Upper Extremity Surgery, Hospital for Special Surgery, New York, NY, USA

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