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ORIGINAL ARTICLE

Preventing brachial plexus injury during shoulder surgery: a real-time cadaveric study

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Background: Brachial plexopathy is not uncommon after shoulder surgery. Although thought to be due to stretch neuropathy, its etiology is poorly understood. This study aimed to identify arm positions and maneuvers that may risk causing brachial plexopathy during shoulder arthroplasty.

Methods: Tensions in the cords of the brachial plexuses of 6 human cadaveric upper limbs were measured using load cells while each limb was placed in different arm positions and while they underwent shoulder hemiarthroplasty and revision reverse arthroplasty. Arthroplasty procedures in 4 specimens were performed with standard limb positioning (unsupported), and 2 specimens were supported from under the elbow (supported). Each cord then underwent biomechanical testing to identify tension corresponding to 10% strain (the stretch neuropathy threshold in animal models).

Results: Tensions exceeding 15 N, 11 N, and 9 N in the lateral, medial, and posterior cords, respectively, produced 10% strain. Shoulder abduction >70° and combined external rotation >60° with extension >50° increased medial cord tension above the 10% strain threshold. Medial cord tensions (mean ± standard error of the mean) in unsupported specimens increased over baseline during hemiarthroplasty (sounder insertion [4.7 ± 0.6 N, $P = .04$], prosthesis impaction [6.1 ± 0.8 N, $P = .04$], and arthroplasty reduction [5.0 ± 0.7 N, $P = .04$]) and revision reverse arthroplasty (retractor positioning [7.2 ± 0.8 N, $P = .02$]). Supported specimens experienced lower tensions than unsupported specimens.

Conclusions: Shoulder abduction >70°, combined external rotation >60° with extension >50°, and downward forces on the humeral shaft may risk causing brachial plexopathy. Retractor placement, sounder insertion, humeral prosthesis impaction, and arthroplasty reduction increase medial cord tensions during shoulder arthroplasty. Supporting the arm from under the elbow protected the brachial plexus in this cadaveric model.

Level of evidence: Basic Science Study; Biomechanics

Keywords: shoulder; surgical positioning; nerve injury; brachial plexus injury; neurologic complications; shoulder hemiarthroplasty; reverse total shoulder arthroplasty; cadaveric study

Neurologic complications are not uncommon in shoulder surgery, with an incidence of 1% to 2% in rotator cuff operations, 1% to 8% in anterior instability procedures, 3% in hemiarthroplasties, 0.1% to 4% in total anatomic shoulder arthroplasties, and 2% to 4% in reverse arthroplasties.[1-3,5,17,23] Most of these are injuries to the trunks

This study was conducted after anatomy licensing approval from the Ministry of Health at the New South Wales State Government.

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and cords of the brachial plexus.[10,17,20] Although most brachial plexopathies resolve spontaneously, permanent injuries do occur and are devastating for the patient and the surgeon.[3,10,14,17]

Despite their frequency, the mechanisms of iatrogenic brachial plexus injury are poorly understood. They are thought to occur due to stretching of the plexus secondary to patient positioning or shoulder manipulation during the operation.[7]

Cadaveric studies have demonstrated shoulder abduction of 90° or greater (particularly when combined with shoulder extension or external rotation) and combinations of external rotation and extreme extension cause brachial plexus stretching.[8,11,12,15,27] Intraoperative peripheral nerve monitoring studies have identified a particular association of extreme shoulder movements, particularly those incorporating several anatomic planes (eg, abduction, external rotation and extension), with nerve dysfunction during shoulder surgery, noted as sustained neurotonic electromyographic activity or greater than 50% amplitude reduction in transcranial electrical motor evoked potentials in the upper extremity muscles.[20,22,30]

These findings support the hypothesis that intraoperative maneuvers/positions cause stretching forces that result in iatrogenic brachial plexopathy. Cadaveric studies, however, have not clearly determined whether positions of the shoulder, particularly those involving combined motions (eg, abduction with external rotation), stretch the brachial plexus.[8,11,12,15,27] Furthermore, no studies have determined whether stretching forces sufficient to cause neuropathy are encountered during shoulder surgery. As such, the etiology of iatrogenic brachial plexus injuries—and thus methods to prevent them—remain unclear.

Strains tolerated by human nerves have not been studied in vivo but have been studied in animal models. In a study of rabbit tibial nerves, Lundborg and Rydevik[16] found that strain (percentage elongation of resting nerve length) of 8% reduced intraneural venous blood flow by more than 50%. Arterial impairment began at strains of 12% and complete cessation of intraneural blood flow occurred at 15% strain.[16] Takai et al[25] found that 8% strains caused failure in compound muscle action potentials in rabbit brachial plexuses. Brown et al[4] and Jou et al[9] found that in rabbit and rat models, respectively, strains of 8% caused reversible nerve function derangement that resolved once tension was released.[4,9] Wall et al[29] and Kwan et al[13] determined that strains of 12% caused severe persisting nerve action potential amplitude reduction in rabbit tibial nerves. There is, therefore, consensus in these animal studies that although strains of 8% cause reversible nerve dysfunction, strains of 12% cause nerve injury. That is, if a nerve is exposed to a strain greater than 10%, a nerve injury may occur.

The aims of this study, therefore, were, firstly, to identify arm positions that place tension on the brachial plexus sufficient to cause nerve dysfunction; secondly, to measure tension in the brachial plexus during shoulder hemiarthroplasty and revision reverse arthroplasty to confirm that intraoperative maneuvers/positions cause stretching forces on the plexus; thirdly, to identify surgical steps during shoulder arthroplasty that may place the brachial plexus at risk of stretch injury; and finally, to determine what steps might be taken to relieve these stretching forces and reduce the risk of iatrogenic brachial plexopathy.

Materials and methods

Study design

This was a cadaveric study investigating the stretching forces on the cords of the brachial plexus in different positions of the arm encountered during surgery and during shoulder hemiarthroplasty and revision reverse arthroplasty.

Experimental setup

We obtained 6 fresh frozen cadaveric upper limb specimens extending from the scapula to fingertip, with intact rotator cuffs and complete acromioclavicular and glenohumeral joints. The lateral, medial, and posterior cords of the brachial plexus were identified and tagged. The elbow and forearm of each specimen was secured with the elbow in 90° flexion and the forearm and wrist neutral. The scapula was fixed to a metal backing plate and mounted in a triaxial vice so that the shoulder joint was in the beach chair position.[21,24]

A loop of #2 FiberWire (Arthrex Inc., Naples, FL, USA) was sutured to the end of each cord of the brachial plexus and attached in series to a digital load cell (HFG110; Transducer Techniques, Temecula, CA, USA; Fig. 1). Each load cell was connected to a ratcheting mechanism to adjust the resting tension of each brachial plexus cord to 1.0 N to simulate normal resting forces experienced by peripheral nerves in vivo.[13,26] Because these forces had not been previously studied in the cords of the brachial plexus, a tension of 1.0 N was chosen as an easily standardizable and comparatively small force (in relation to those in biomechanical studies on peripheral nerves in humans and animals) and thus unlikely to exceed normal in vivo forces.[13,18,28] Throughout testing, specimens were kept moist with physiological saline solution (NaCl 0.9%).

Range of motion testing

The shoulder was placed in the neutral position (0° flexion, 0° abduction, and 0° external rotation) with the humeral head in the glenoid fossa. While in this position, a resting tension of 1.0 N was applied to each of the cords via the ratcheting mechanism to simulate physiological resting tensions. Tension in each cord was measured during 3 arcs of movement: (1) every 10° while the shoulder was moved from 30° internal rotation to 90° external rotation and flexion and abduction were kept at 0°; (2) every 20°, from 0° to 80° abduction and at 90° abduction while the angle of external rotation and flexion was kept at 0°; and (3) every 20°, from 60° extension to 100° flexion while the angle of external rotation and abduction was kept at 0°. Shoulder external rotations of 30°, 60°, and 90° were then combined with shoulder extension (every 20° from 0°-60°) and shoulder abduction (every 20° from 0°-100°). Shoulder flexion, extension, and abduction were measured with a P8101 minidigital protractor angle meter (T&E Tools Pty Ltd, Pendle Hill, NSW, Australia). Shoulder internal and external rotation were measured with an M972 digital goniometer (CARBA-TEC, Auburn, NSW, Australia). Tension was