SCIENTIFIC ARTICLE

Biomechanics of the Acute Boutonniere Deformity

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Purpose To demonstrate which structures of the extensor mechanism create a boutonniere deformity, when damaged, in a cadaver model. An analysis of how damage to these anatomical structures affects the biomechanical performance of the extensor mechanism was also performed.

Methods We secured 18 fresh cadaveric hands onto an apparatus consisting of a computer-controlled motor and tensiometer attached in series to the extensor communis tendon of the ring and middle digits. The central slip, transverse, and oblique fibers of the interosseous hood and the triangular ligament were sequentially divided. After each structure was divided, the motors were activated to provide a constant tendon displacement force. The angular displacement at the proximal interphalangeal (PIP) and distal interphalangeal joints was recorded.

Results In all digits, detachment of the central slip from the middle phalanx produced a decrease in extension of the PIP joint. When the transverse and oblique fibers of the interosseous hood were also divided, extension at the PIP joint was further decreased. A boutonniere deformity occurred only when all 3 structures were damaged.

Conclusions The boutonniere deformity requires subluxation of the lateral bands volar to the axis of rotation of the PIP joint. This study demonstrates that damage to the central slip alone does not cause the deformity. Combined injury of the central slip, triangular ligament, and transverse and oblique fibers of the interosseous hood causes a boutonniere deformity.

Clinical relevance Division of the central slip leads to loss of extension at the PIP joint. A more substantial loss of extension after injury or development of a boutonniere deformity should alert clinicians that other structures of the extensor mechanism are also damaged. *(J Hand Surg Am. 2017;■(■):1.e1-e6. Copyright © 2017 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Boutonniere, biomechanics, hand, finger, acute.

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THE BOUTONNIERE DEFORMITY IS characterized by flexion at the proximal interphalangeal (PIP) joint with hyperextension at the distal interphalangeal (DIP) joint. This occurs as a result of lateral band (LB) subluxation below the axis of rotation of the PIP joint, where the LBs function as paradoxical flexors. The deformity may be caused by open or closed traumatic injuries of the central slip (CS),[1–4] rheumatoid arthritis,[5] or a congenital abnormality.[6] The current study focused on traumatic injuries.

Using a cadaveric model, Grundberg and Reagan[7] found that a surgically created central slip laceration

did not cause subluxation of the LB and an acute boutonniere deformity to occur. Another study by Mercer et al[8] showed that contributions from the LB largely governed PIP extension. They reported that extensor mechanism repair with the LBs placed in a dorsal position, without reattachment of the central slip, prevented the development of a boutonniere deformity.

The purposes of our study were to create a reproducible model for boutonniere deformity in a cadaver and to analyze quantitatively how sequential damage to key anatomical structures of the extensor mechanism affects the biomechanics of extension. We hypothesized that damage to the CS, triangular ligament, and transverse and oblique fibers of the interosseous hood running between the central slip and the LBs is necessary to create an acute boutonniere deformity.[9] We also hypothesized that damage to each of these structures decreases the biomechanical efficiency of the extensor mechanism leading to decreased PIP extension.

MATERIALS AND METHODS

Anatomic study

We thawed 10 fresh cadaveric hands in normal saline, sectioned them at the distal third of the forearm, and fixed them palmar side down on the test apparatus. We randomly selected and used either the middle or ring digit in each cadaveric hand and excluded digits with poor passive range of motion. The ring and the middle digits are more commonly injured than other digits, accounting for more than 50% of the cases in a large case series.[10] We elected against using the index and little digits because of contributions of the extensor indicis and extensor digiti quinti, respectively. Skin and subcutaneous tissue were removed from the digits tested. We then dissected and identified the CS, LB, transverse and oblique fibers of the interosseous hood, and the triangular ligament (Fig. 1A).

For all 10 digits we divided the CS at its insertion to the base of the middle phalanx. In group A (5 digits in total), we sequentially lacerated the triangular ligament and the transverse and oblique fibers of the interosseous hood running between the CS and LB. In group B (5 digits in total), we reversed the sequence, lacerating the transverse and oblique fibers of the interosseous hood before the triangular ligament. Figure 1A is a schematic of the anatomy of the extensor mechanism; Figure 1B depicts the sequence of structures damaged in groups A and B.

Based on previous descriptions in the literature, we simulated the action of the flexors and intrinsic muscles by applying loads to the superficial (100 g) and deep (100 g) flexor tendons as well as the radial (400 g) and ulnar (400 g) interosseous tendons by sutures through a low-friction pulley system.[11] The extensor communis tendon was then sutured to a computer-controlled, winch-type servomotor and tension was varied to obtain multiple initial flexion angles at the PIP joint. A constant tendon displacement force of 30 mm/min was then applied. Consistent with physiological loads described in the literature, the force never exceeded 20 N.[12,13] Two separate investigators independently used goniometers to measure the relative flexion-extension at the PIP and DIP joints after extensor tendon excursion.

Biomechanical study

Eight additional intact, fresh cadaveric hands were thawed in normal saline, sectioned at the distal third of the forearm and fixed palmar side down on the test apparatus. After randomly selecting either the ring or middle digit, we made a midline dorsal incision over each digit beginning 1 cm distal to the DIP joint to 4 cm proximal to the metacarpal head. We dissected and identified the CS, LB, transverse and oblique fibers of the interosseous hood and the triangular ligament (Fig. 1C). We then sutured the extensor communis tendon to the same computer-controlled, winch-type servomotor with a tensiometer connected in series, 4 cm proximal to the metacarpal head. To measure angular displacement of the PIP relative to the starting position of 35° flexion, we used a potentiometer, which acted as a rheostat (Fig. 2). The potentiometer was secured to the soft tissues of the PIP joint by sutures (Fig. 2). As rotation at the PIP joint rotated the potentiometer, a precalibrated angular displacement was displayed and recorded.

Four total configurations were tested for each digit. In configuration 1, all structures remained intact. In configuration 2, the CS was detached from its insertion on the middle phalanx. In configuration 3, the CS was detached and the transverse and oblique fibers of the interosseous hood were sectioned on either side of the CS. In configuration 4, the triangular ligament was sectioned longitudinally, the CS was detached, and the transverse and oblique fibers of the interosseous hood were sectioned on both sides of the CS. Figure 1A is a schematic of the anatomy of the extensor mechanism; Figure 1C depicts the sequence of structures divided in each of the configurations.

Using the same apparatus as before, a computer recorded data from the potentiometers and a modified version of LabVIEW Software (National Instruments Corporation, Austin, TX) calculated angular displacement at the PIP joint.[13] The data