CURRENT CONCEPTS

Sagittal Band Injuries: A Review and Modification of the Classification System

Brahman Sivakumar, MBBS, BSci, MS,*† David J. Graham, MBBS, BPhty,†‡ Mark Hile, BE, MBBS,* Richard Lawson, MBBS*

Sagittal band injuries, although relatively uncommon, can be difficult to treat. This review provides a contemporary perspective on this pathology, as well as a modification to the classification system. This modification aims to incorporate the spectrum of disease seen, guide treatment, and allow standardization when documenting and describing injuries. *(J Hand Surg Am. 2021;■(■):■−■. Copyright © 2021 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Classification, extensor tendon instability, sagittal band.Sagittal band injuries, while relatively uncommon, can be difficult to treat, particularly in the setting of delayed presentations. This article provides a topical review and describes a modification to the most prevalent classification system.

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ANATOMY

The sagittal band (SB) comprises a portion of the retinacular system of the extensor apparatus to the digits, which also incorporates the triangular ligament between the lateral bands, the transverse and oblique retinacular ligaments, and the transverse aponeurotic fibers between the central tendon and lateral bands at the level of the proximal phalanx.[1] Along with the volar plate, the SB forms a closed cylindrical tube surrounding the metacarpal head and metacarpophalangeal (MCP) joint (Fig. 1). The SB demonstrates a free proximal edge at the level of the metacarpal neck; distally, it merges with the interosseus tendon, which forms the free palmar border of the extensor retinacular system. The SB of the middle finger extends further proximally and distally than those of other digits.[2]

The SB splits dorsally to envelop the extensor tendon via thin superficial and thicker deep layers. The deep layer has a groove in which the extensor tendon sits, and contributes fibers to a ridge on the dorsal base of the proximal phalanx.[1,3] Based on a small number of histopathological sections, Kettelkamp et al[4] found that the dorsal component of the SB was thicker in the central digits than the border digits, which was likely due to the presence of accessory extensor tendons in the little and index fingers. The middle finger also demonstrated relatively fewer fibrous connections between the tendon and enveloping SB, and a more rounded cross-sectional tendon shape, which may partially account for its propensity toward injury and instability (Figs. 2 and 3).[4,5]

Lateral to the extensor tendon, the superficial and deep layers coalesce into radial and ulnar laminae, which may be divided into palmar and dorsal portions by the interossei tendons.[1] The radial lamina is often thinner and longer than the ulnar component; the dorsal portion of each lamina is larger and more

*From the *Department of Hand & Peripheral Nerve Surgery, Royal North Shore Hospital, St Leonards, Australia; the †Australian Research Collaboration on the Hand (ARCH), Palm Beach, Australia; and the ‡Department of Orthopaedic Surgery, Gold Coast University Hospital, Southport, Australia.*

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Corresponding author: Brahman Sivakumar, MBBS, BSci, MS, Department of Hand & Peripheral Nerve Surgery, Royal North Shore Hospital, Reserve Rd, St Leonards, NSW, Australia 2065; e-mail: brahman.sivakumar@gmail.com.

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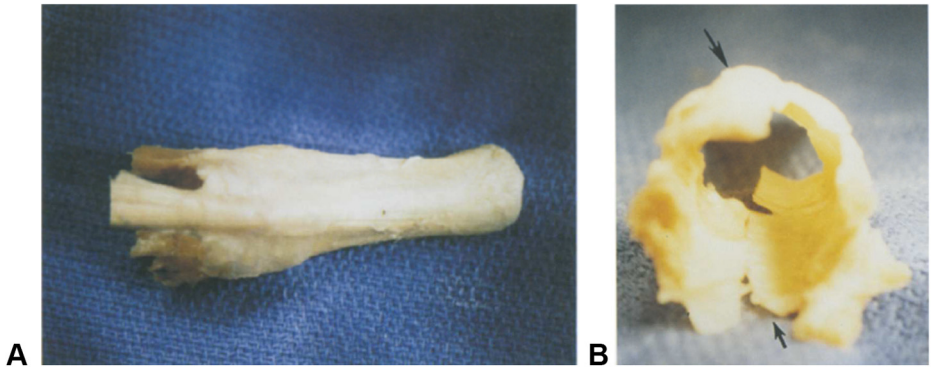

FIGURE 1: The extensor retinacular system is shaped as a hollow tunnel, incorporating the extensor digitorum (large arrow) and interossei tendons. Along with the palmar plate (small arrow) the SB forms a tube which surrounds the metacarpal head. Reprinted with permission from Rayan et al.[1]

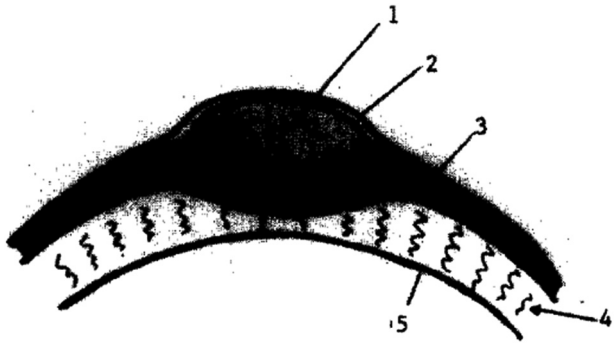

FIGURE 2: Schema of the extensor apparatus of the middle finger at the level of the MCP joint, where 1 indicates the extensor tendon; 2 the superficial layer of the SB; 3 the deep layer of the SB; 4 the loose connective tissue between the SB and dorsal capsule; and 5 the dorsal capsule. Reprinted with permission from Ishizuki.[3]

substantial than the palmar portion. The lamina bifurcate to envelop the superficial interosseous tendon, and are separated from the collateral ligament by the attachment of the interosseous tendon (its medial tendon) onto the lateral tubercle on the proximal phalangeal base.[1]

The SB blends volarly with the volar plate and accessory collateral attachment; the flexor sheath and proximal annular pulley; and the deep transverse metacarpal ligament. Despite this confluence, the SB and collateral ligaments are discrete structures, moving independently and gliding across each other in flexion and extension.[2] The confluence differs in the border digits, as the deep transverse metacarpal ligament is not present on the radial aspect of the index finger or ulnar aspect of the little finger.[1]

The extensor retinacular system contains dense collagen fibers with no elastic tissue.[1] These fibers are orientated somewhat perpendicular to the digital axis, running dorsally from proximal to distal at the MCP joint and more obliquely in the distal SB. The oblique fibers also intersect a group of fibers running volar from the lateral slip of the extensor digitorum tendon, forming a crisscross lattice.[1,6] The dorsal fibers of the SB move proximally when the MCP joint hyperextends through extensor pull, and move distally with MCP joint flexion.[2] On average, the fiber orientation changes 30° through the MCP joint range, with the greatest change noted in the little finger.

FUNCTION

A number of functions have been attributed to the SB, including stabilization of the extensor tendon, extension of the proximal phalanx, prevention of extensor tendon bowstringing, and limitation of extensor excursion.[1]

Using cadavers, Young and Rayan[2] assessed the effects of SB injuries on extensor tendon stability. Sectioning of the ulnar lamina was not found to cause instability. Division of the proximal half of the radial lamina resulted in subluxation, with complete radial SB division leading to tendon dislocation (Fig. 4). This difference may be due to asymmetry of metacarpal head geometry, the placement of extrinsic and intrinsic muscle insertions, the normal digital resting position of slight ulnar deviation at the MCP joint, and more substantial ulnar-sided juncturae tendinum resulting in both prevention of radial instability and increased pull toward ulnar dislocation.[7,8] Division of the distal half of the radial SB did not produce instability. Instability was found to increase with MCP joint flexion (particularly between 45° to 90°) and wrist flexion (past neutral), which has implications for rehabilitation.[2] The middle finger had the

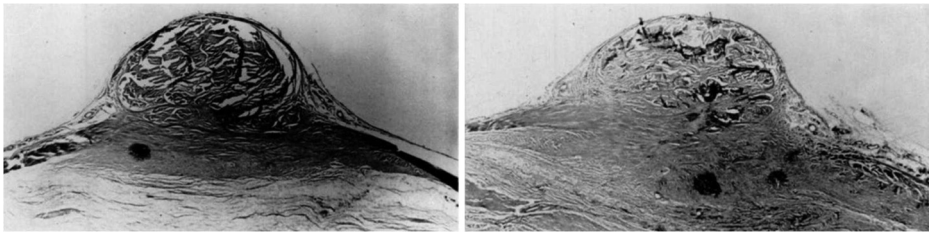

FIGURE 3: Histological cross-section over the MCP joint, showing the tendon sitting on top of transverse fibers with few interconnections in **A** the middle finger as compared to **B** the ring finger, which has many. Reprinted with permission from Kettelkamp et al.[4]

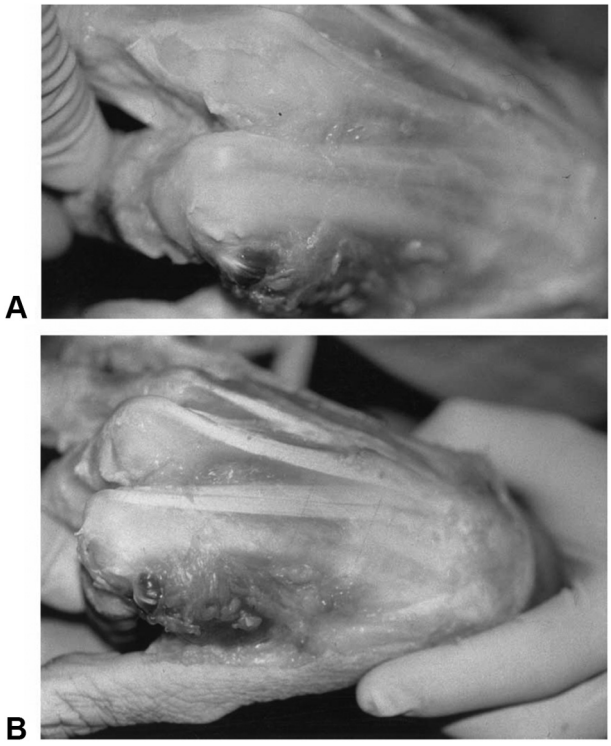

FIGURE 4: A Partial proximal sectioning and **B** complete sectioning of the radial SB of the index finger. The wrist and the MCP joint are flexed. With proximal partial sectioning, the extensor tendons are subluxed ulnarly and ride over the ulnar border of the metacarpal head; with complete sectioning, the tendons dislocate. Reprinted with permission from Young and Rayan.[2]

FIGURE 5: Complete sectioning of the radial SB of the little finger, with flexion of the wrist and MCP joint and abduction of the digit. The extensor tendons are only moderately subluxed, due to the stabilizing effect of the junctura tendinum. Reprinted with permission from Young and Rayan.[2]

greatest propensity to develop instability, likely due to the tendon blending into the extensor hood more distally than in other digits, as well as the relatively fewer fibrous connections between the tendon and enveloping SB.[3,4] In contrast, tendon instability did not occur in the little finger despite complete radial SB division with the wrist and MCP joint flexion, due to the stabilizing effect of the juncturae and the greater frequency of multiple shared extensor tendons to the ulnar 2 digits (Fig. 5). Instead, patients tend to develop abduction deformities of the little finger, due to the unrestricted pull of the abductor digiti minimi. Young and Rayan's[2] findings were in concordance with those of Koniuch et al[8] from an earlier study. Normal tensioning of the radial SB is thought to be assured by 2 anatomical factors of the intact metacarpal head—the cam effect and radial prominence—which in full flexion make the radial SB sufficiently taut to draw the tendon 2 to 3 mm radial.[9]

Marshall et al[10] challenged the theory that the SB drives MCP joint extension via a sling effect around the base of the proximal phalanx, by comparing reductions in extension force following division of either the SB or the extensor tendon distal to it on cadavers. Although extension force diminished by 8% after SB division with the MCP joint at neutral and by 7% when the MCP joint was placed in 45° flexion, this result was not significant. Division of the extensor tendon, however, resulted in decreases in extension force of 85% at neutral and 35% with MCP joint flexion. The authors concluded that the role of the SB in MCP joint extension is negligible; rather,

the extensor tendon has a moment arm, by way of its more distal insertion on the composite digital structure of articulated phalanges, and generates an extensor moment at the MCP joint.

The importance of the SB in limiting extensor tendon bowstringing and excursion, and subsequent alterations in digital range of motion, have not been elucidated. This may be an interesting subject for future cadaveric research, particularly given the presence of loose connective tissue attachments from the volar surface of the extensor tendon to the dorsal MCP joint capsule and base of the proximal phalanx, which have been found to limit hyperextension of the proximal phalanx.[11]

PATHOMECHANICS

Acute injury to the SB can occur via closed or open mechanisms. Colloquially known as a "boxer's knuckle", blunt trauma can result in disruption of either laminae of the SB and occurs most frequently in the middle finger due to the previously mentioned anatomical peculiarities, as well as the relative prominence of the third metacarpal head.[4,12] Young and Rayan[2] determined that the radial SB receives more tensile stress with MCP joint ulnar deviation and flexion, and hypothesized that full ulnar deviation against resistance in extension (or less commonly, flexion) predisposes the radial SB to traumatic disruption. Full flexion of the MCP joint during trauma also increases the risk of associated collateral ligament injury, which has prognostic implications.[2,7] More severe injuries with associated osseous, capsular, or ligamentous damage increase the risk of failure with nonoperative measures, perhaps due to retraction and scarring of the damaged capsule leading to the establishment of a fistula between the MCP joint and the subcutaneous space, with persistence of joint fluid in the disrupted interval.[12,13]

An atraumatic acute SB injury can also occur during common, low-energy daily activities, such as flicking the finger or crumpling paper.[3] Interestingly, Ishizuki[3] noted during exploration of a series of SB injuries that while traumatic disruption resulted in tearing of both superficial and deep layers of the SB lamina several millimeters lateral to the extensor tendon, atraumatic injuries result in rupture of the superficial layer adjacent to the tendon (Fig. 6). Thus, the technique of repair should be guided by the mechanism of injury.

Penetrating or open injuries can disrupt either of the tendinous or retinacular components of the extensor apparatus at the level of the MCP joint and should be explored appropriately.

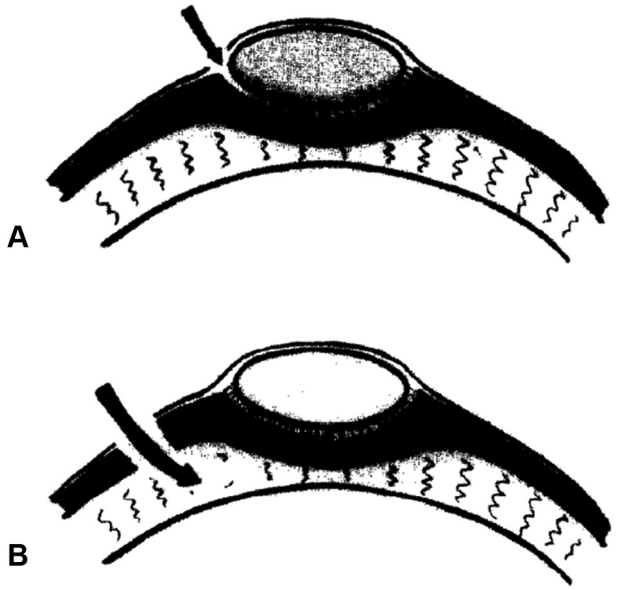

FIGURE 6: **A** While atraumatic injuries cause disruption of the superficial layer adjacent to the extensor tendon, **B** traumatic injuries result in disruption of both superficial and deep lamina several millimeters lateral to the tendon. Reprinted with permission from Ishizuki.[3]

ASSESSMENT

When acute SB injury is being considered, a thorough history ascertaining the mechanism of injury is paramount. Patients may present complaining of pain and swelling or of a sensation of snapping or pseudotriggering at the MCP joint. Careful physical examination will allow confirmation of the diagnosis and sequelae, as well as exclusion of differentials.

On examination, subluxation or dislocation (and subsequent relocation) of the extensor tendon to the border of the metacarpal head or into the gully between the metacarpals may be visualized. An extensor lag, and decreased flexion secondary to pain, may be present.[14] Extensor quadriga may also be present, as effective gliding amplitude of the lesser digits is linked due to the common muscle belly of the extensor digitorum communis, with a lack of extension in 1 digit resulting in decreased excursion in the adjacent tendons through distal tension on the juncturae tendinum. Long-standing quadriga can lead to intrinsic muscle tightness and secondary swan-neck deformity.

The patient's capacity to initiate and maintain MCP joint extension will differentiate between SB injury and extensor tendon dysfunction: if the patient cannot actively extend the involved digit from a flexed position, but can maintain full extension when passively placed, SB disruption is likely. In the

setting of extensor tendon disruption, the patient may be able to initiate extension from a flexed position (due to pull transmitted through the juncturae tendinum) but will be unable to maintain active extension. Stenosing flexor tenosynovitis can be excluded by a lack of tenderness over the proximal annular pulley. Confirmation of innervation to the extensor pollicus longus or extensor indicus proprius tendons renders a posterior interosseous nerve palsy unlikely.

Although the diagnosis is often apparent through physical examination, further imaging will allow confirmation of SB disruption and visualization of associated injuries. In addition to a standard hand series of radiographs, the Brewerton view (where an anteroposterior beam is directed in a 15° radial angle at a fully supinated hand with the MCP joint flexed 65°) may discern subtle osseous pathology in the MCP joint region (Fig. 7). Dynamic ultrasonography can define tendon instability; magnetic resonance imaging will confirm disruption of the SB. Arai et al[13] recommended arthrograms to diagnose capsular disruption and aid in the consideration of operative intervention.

CLASSIFICATION

Gladden[16] initially classified boxer's knuckles into 4 types based on intraoperative findings, ranging from thickening of soft tissues (including the tendon and capsule over the distal metacarpal) with no tear to a disruption of these tissues with the defect extending into the joint. Rayan and Murray[12] later divided closed SB injuries into those with a contusion, but no tear of the retinacular structures or instability (type 1), those with tendon subluxation (type 2), and those with tendon dislocation (type 3; Fig. 8). Neither classification system is inclusive of the spectrum of pathology seen clinically, guides treatment, allows prognostication, or affords enough detail to permit a comparison of treatment methods.

The authors of this review have proposed a modification to Rayan and Murray's classification that may be better suited (Table 1). Types 2 and 3 have been modified to focus on acute injuries with tendon subluxation or dislocation (those within a 6-week period of the index insult, due to the high rate of failure with nonoperative measures thereafter, as will be discussed later). New categories have been added to include chronic injuries (patients seeking treatment after 6 weeks); SB disruptions in combination with bony, capsular, or ligamentous injuries (as determined on imaging or intraoperative findings); and open injuries. The new classification incorporates the spectrum of disease seen, guides treatment (with the new categories more likely to require operative intervention), and allows standardization when documenting and describing injuries, to allow comparison between treatment modalities. A higher grade of injury increases the likelihood of failure with nonoperative management and progression to surgery. Type 1 injuries can be treated symptomatically, and do not require splinting; patients with types 2 and 3 injuries should be treated with an appropriate splinting protocol as first-line management. Types 4 and 5 injuries are unlikely to be successfully managed via splinting, and treating clinicians should advise the patient accordingly and have a low threshold to surgically intervene. Patients with type 6 injuries will require an operation.

MANAGEMENT

Patients with type 1 SB injuries can be treated symptomatically with buddy strapping and analgesia as required. Closed, acute, and reducible injuries with instability should be treated nonoperatively with appropriate splinting as soon as possible. Early splinting protocols consisting of volar MCP joint splinting in a neutral position for 3 weeks, followed by protected range of motion for a further 3 weeks, were associated with high rates of failure. Rayan and Murray[12] noted failure at 24 months in 4 of 18 digits treated in this manner, although the patients had painless instability with normal function. Arai et al[13] reported ongoing instability in all patients treated this way; however, the mean time to presentation was 7 weeks from injury, and 6 of 8 patients had an associated capsular injury on surgical exploration, both of which the author identified as increasing the risk of failure. They hypothesized that more extensive injuries may result in synovitis and a fistula between the MCP joint and the subcutaneous space, with persistence of joint fluid resulting in failure of nonoperative management.

Contemporary protocols utilize the relative motion splint described by Merritt,[14] whereby the injured digit is placed in 15° to 20° of greater MCP joint extension than adjacent digits, and full flexion at the MCP and interphalangeal joints is encouraged. This splint is based on the rationale that when an injured tendon is placed in 15° to 20° less relative motion than adjacent tendons with a common muscle belly, it experiences markedly less force than those adjacent tendons, regardless of the position from full extension to full flexion (Fig. 9). This concept also reduces tension across the SB, allowing effective

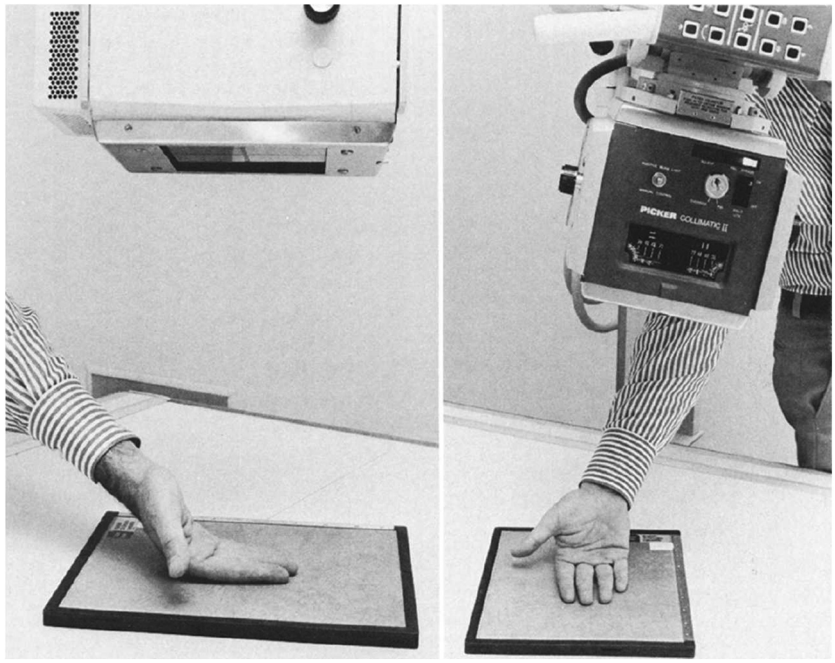

FIGURE 7: The Brewerton view, in which an anteroposterior beam is directed in a 15° radial angle at a fully supinated hand with the MCP joint flexed 65°, may be useful. Reprinted with permission from Lane.[15]

FIGURE 8: Rayan and Murray classification of SB injuries. Reprinted with permission from Rayan and Murray.[12]

TABLE 1. Proposed Classification System

Type 1	Mild injury with no instability
Type 2	Acute moderate injury with extensor tendon subluxation (<6 weeks)
Type 3	Acute severe injury with tendon dislocation (<6 weeks)
Type 4	Chronic injury with tendon subluxation or dislocation (>6 weeks)
Type 5	Injury to associated structures
Type 6	Open injury

rehabilitation of an injured, repaired, or reconstructed SB. Reported success rates with utilization of this splint range from 71% to 84%. Peelman et al[17] noted resolution of symptoms in 94% of acute injuries (within 3 weeks of injury), 91% of subacute injuries (3 to 6 weeks after the insult), and 62% in chronic injuries (after more than 6 weeks) in a series of 101 digits treated with the splint for 6 weeks. A multivariate analysis in Roh et al's[18] study of 94 digits treated with 5 weeks of full-time splinting, followed by 2 weeks of part-time usage, identified longer times from injury to presentation, manual labor, and frank tendon dislocation (as opposed to subluxation) as predictive of a higher likelihood of nonoperative treatment failure.

Patients who fail nonoperative management, those with delayed presentations, those who cannot tolerate prolonged splinting (such as professional athletes),

FIGURE 9: Relative motion splint, based on the rationale that when an injured tendon is placed in 15° to 20° less relative motion than adjacent tendons with a common muscle belly, it experiences markedly less force than those adjacent tendons. Reprinted with permission from Merritt.[14]

those with open injuries, and those with irreducible tendon dislocations should be considered for operative intervention. The primary goals of surgery should be to relieve pain and restore function, with reversal of instability secondary.[12] In the acute phase, direct repair of the injured structure may be possible, with many authors advocating for the procedure to be performed under wide-awake local anesthesia to confirm stability and tensioning.[7,14] Hong et al[19] recommended a double layer of continuous interlocking suture (running proximal to distal initially, and then back) to repair the superficial layer of the SB to the lateral margin of the tendon in atraumatic ruptures, reporting no failures in a series of 26 patients. In traumatic ruptures with disruption of both the superficial and deep layers, Ishizuki[3] advocated the placement of 2 anchoring sutures between the paratenon of the tendon and the deep layer prior to the superficial repair. Lee et al[20] suggested separating the radial SB from the capsule, suturing the radial aspect of the SB to the radial paratenon, and then draping the remainder over the tendon before securing it to the ulna lamina (Fig. 10). They reported complete resolution of symptoms, full range of motion, and no instability in a series of 13 patients treated in this fashion. The literature is equivocal on whether associated dorsal capsular pathology should be repaired, with some authors advocating for repair, while others recommend against for fear of loss of motion.[7,19] Regardless, if direct repair alone does not centralize the extensor tendon, a combination of release of tight ulnar structures (ulnar SB lamina, juncturae tendinum, and collateral ligament) and reefing or imbricating the radial SB may be required.[7,12,19]

In the setting of insufficient native tissue for SB repair, a reconstruction should be performed. A number of methods have been derived from Kilgore et al's[9] original description of using a distally based slip of the extensor digitorum tendon, which is then passed around the radial collateral ligament and secured onto itself. Criticisms of this technique include that trans-articular passage of the restraint may cause scarring and limit range of motion. To counter this, Watson et al[5] described a variation where the distally based tendon slip is passed through the tendon itself (to prevent further distal migration of the surgically created split) before being looped through a passage in the deep transverse metacarpal ligament on the side of the injured SB and secured back to the tendon (Fig. 11). They reported resolution of pain with no recurrence of instability in a series of 21 SB reconstructions. Modifications using an ulnar-sided tendon slip (to create greater radial tethering force), using a slip from an adjacent digit or free tendon autograft, or securing the check-rein around the interossei or lumbrical tendons or into the metacarpal head have also been described with generally excellent results.[5,7,14] Regardless of the method used, an isometric tether point must be carefully chosen, with placement too proximal limiting full MCP joint extension and placement too distal leading to ongoing instability. Watson et al[5] recommended the ideal position as 8 to 14 mm proximal to the articular surface of the proximal phalanx with the MCP joint in full flexion.[5] Alternative reconstructive methods have been described that aim to reduce the potential of postoperative stiffness. Segalman[21] described the use of the lumbrical tendon to create a dynamic muscle transfer that stabilizes the tendon during MCP joint flexion. Kang and Carlson[22] created a new pulley using autograft passed through a bone tunnel in the distal metacarpal and tied to itself, with careful

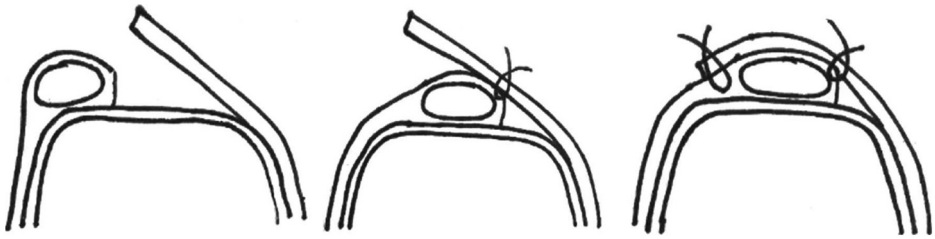

FIGURE 10: Technique of "double breasting" for SB stabilization. Reprinted with permission from Lee et al.[20]

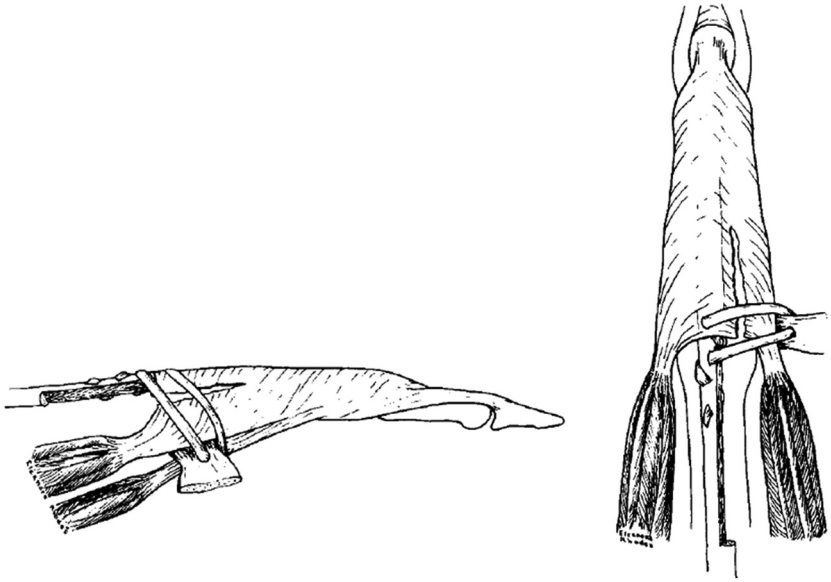

FIGURE 11: Watson et al method of SB reconstruction. Reprinted with permission from Watson et al.[5]

tensioning while the joint is ranged to ensure a stable tendon realignment while minimizing stiffness.

Recommendations on the ideal management of SB injuries are limited by the quality of the available literature. A recent systematic review by Wu et al[7] identified only 7 studies discussing nonoperative management and 14 studies detailing operative treatment of SB injuries, all of which were either retrospective case reports or series. A comparison of outcomes was limited by the low quality and small cohorts of the studies, as well as a lack of standardized reporting on demographics, injury severity, and outcomes. Implementation of a more robust and inclusive classification system, as well as a high-quality prospective study, would be beneficial.

SUMMARY

SB injuries, while relatively uncommon, can be difficult to treat. This review and modification of the classification system may help to guide treatment and allow standardization in documenting and describing injuries.

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