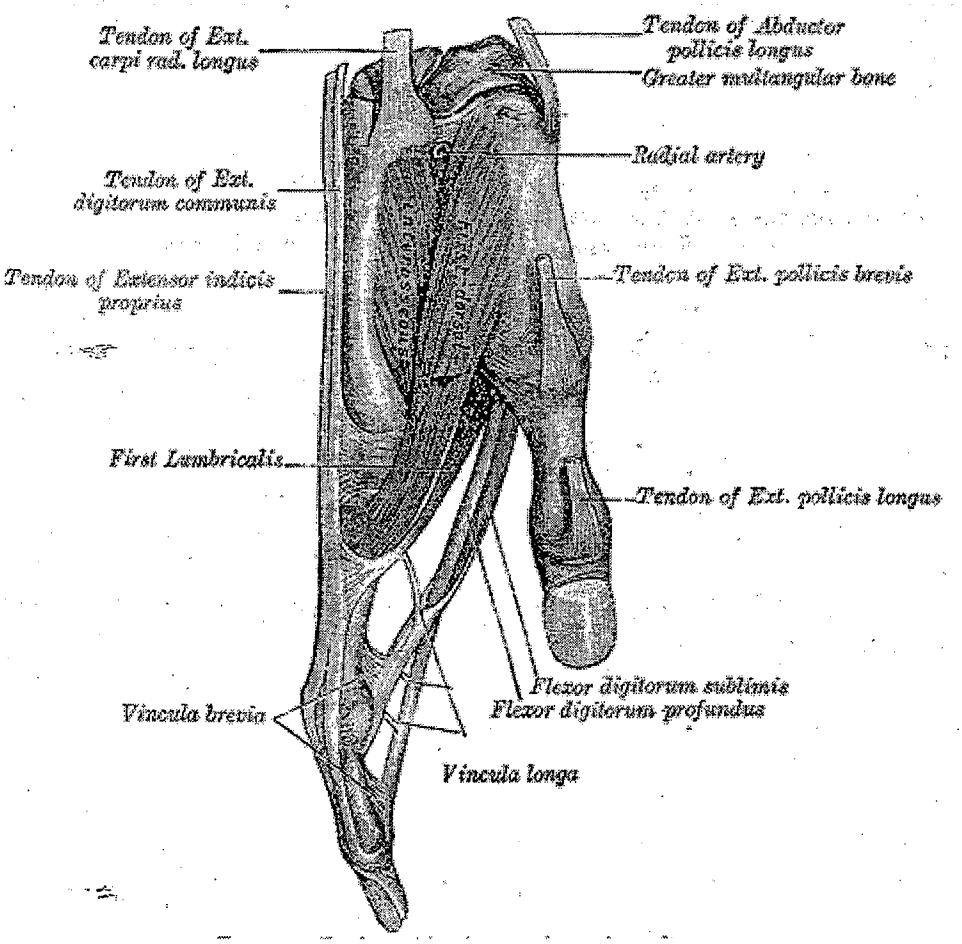
Trigger Finger:

Contributing Factors, Diagnostic Approach and Conservative Means of Treatment

A Literature Review



By: Brian K. Marquez
Trimester 10 #10032

Advisor: Dr. David Stewart

Abstract

Background:

Trigger finger or stenosing tenosynovitis of the digit is a begnign lesion manifested as painful locking or triggering of the involved finger. Notta in 1850 was the first to describe stenosing tenosynovitis of the finger flexors or the trigger finger or the Notta-Neelaton syndrome. (1) Most cases are secondary to thickening of the digit's A1 pulley but other pathogeneses include tendon abnormalities at the level of the carpal tunnel, thickening of other pulleys, and abnormalities of the metacarpal-phalangeal joint.

Objective:

The focus of this literature review is to present an overall view of the factors contributing to trigger finger, its diagnostic approach and the conservative means of treatment. Conservative treatment in regards to this paper does not include steroid injection or surgery.

Data Source:

A computerized search was conducted at the Logan College of Chiropractic Gym. Using Pubmed database, a search was performed using trigger finger as the keyword. A search for texts on tendinitis was then conducted at Logan College of Chiropractic Gym.

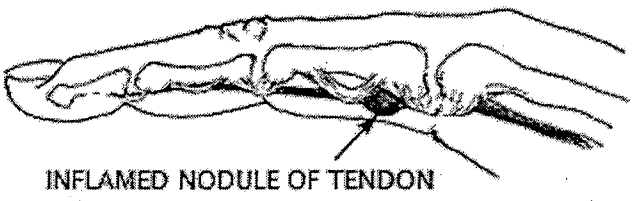
Conclusions:

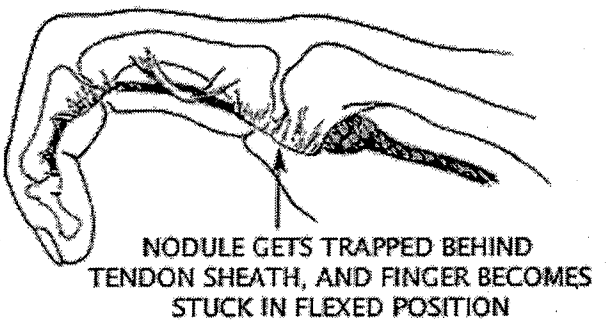
With proper splinting and elimination of provocative movements such as repetitive grasping or the use of tools that apply pressure over the area conservative management of trigger finger may be considered a reasonable treatment option. Patients with trigger finger should have their hands thoroughly examined to rule out carpal tunnel syndrome. This is due to the strong correlation found between carpal tunnel and trigger finger. It may be appropriate to screen all patients with trigger finger for diabetes. (44) This is especially true when the patient presents with bilateral or multiple trigger fingers.

Key Indexing Terms:

Trigger finger, stenosing tenosynovitis, Notta-Neelaton syndrome, Trigger digit

Factors Contributing to Trigger Finger





Trigger finger is classified as an overuse injury. An overuse injury is defined as a long-standing or recurring othropedic problem and pain in the musculoskeletal system, which begins during exertion due to repetitive tissue microtrauma. (2,3). Repetitive microtrauma results in microscopic injury. A single acute trauma is normally not involved in the pathogenesis of an overuse injury. In tendinous injuries, overuse implies a repeated strain of 4% to 8% on a tendon that results in the inability of the tendon to endure further tension, whereby injury occurs. (4,5,6,7) The structure of the tendon is disrupted micro- or macroscopically by this repetitive strain causing breakage of their cross-linked structure, and denature, causing inflammation, edema, and pain. Thus, trigger finger is the earliest clinical manifestation of tendon injury due to overuse (5)

Stenosing tenosynovitis in finger flexors occur in adults and rarely in children. In 1994 Rogers and Waters researched the incidence of trigger finger in newborns. They found that trigger finger to be uncommon in infants. They prospectively examined 1046 newborns to determine the congenital incidence of this condition. No trigger fingers were identified among these children. This correlates by power calculation to an

incidence of 0-3 trigger digits per 1000 live births. They then retrospectively reviewed the records of all children who had undergone surgical release of trigger finger at Boston Children's Hospital from July 1989 to July 1992. Seventy-three children underwent 89 trigger thumb release and five children had eleven trigger finger releases. This study raises the possibility that trigger finger represents lesions acquired after birth (8).

In children the highest incidence can be found at the age of two to four without a clear preponderance in either gender (9). The flexor pollicis longus tendon at the level of the metacarpophalangeal joint is most commonly involved, followed by the third, fourth, fifth and second finger (10). Macroscopically, the sheath of the flexor pollicis longus tendon is thickened at the level of the metacarpophalangeal joint, with nodular swelling of the tendon distally from the stenosis (9) Microscopically, fibrosis and proliferation of the capillaries of the sheath, and hyperplasia and nonspecific inflammation of the synovial villi can be observed. In the tendon tissue, large areas of hylinosis, mucoid degeneration and chondral metaplasia have been seen (11, 12).

In Adults Trigger finger usually occurs during the fifth decade of life (10). Sixty percent to one hundred percent of the patients are women. The dominant hand is more frequently affected and bilateral affection can be found in five to twenty-five percent of the cases. The thumb is the most frequently affected finger (50%-70% of the cases), followed by the third and fourth fingers (10%-20%, respectively), the little finger (about 5%) and the index finger (about 2%) (1). The exact etiology and pathogenesis of the trigger finger in adults are not clear. Local friction is important because the stenosis is constantly found at the same anatomic site, the metacarpophalangeal joint. Macroscopically, both thickening of the fibrotic tendon sheath and nodular thickening of

the tendon belly occur in 70% of the cases; in 10%, only the fibrotic tendon sheath is thickened, and in 20 % only the nodular thickening o the tendon belly occurs (10). Microscopically, both chronic, nonspecific inflammation as well as hyaline degeneration can be found in both the tendon and the sheath (13).

Repetitive overuse following microtrauma is probably part of the process since certain manual occupations, such as shoemakers, dressmakers, carpenters, and cutters represent a majority of the patient population (9) Zelle and schnepp (34) discovered cases case occurring among occupations thought to involve prolonged, frequent, vigorous movements of the involved tendons, including pianists, typists, bookkeepers, maids, factory workers, tailors and clerks. In the majority of the patients cumulative irritation of the sheath by forceful back and forth gliding of the tendon during repeatedly performed identical operations was the producing factor of trigger finger rather than acute trauma. (35) A study was conducted by Gorsche et. al. (37) to determine the prevalence and incidence of trigger finger in a meat-packing plant and explore the relationship between hand-tool use and the development of trigger finger. A cross-sectional study was competed wherein 665 workers were interviewed and examined to determine the point prevalence. Subsequently, 454 trigger finger negative workers were followed up and examined twice at a median interval of 255 days. The point prevalence of trigger finger was 14%. The person-year incidence rate was 12.4% and 2.6% for tool use and non-tool use workers, respectively. This indicates an increased prevalence of trigger finger in this meat-packing plant and high worker turnover may underestimate the true prevalence rates. Rosenthal stated that pinching placed greater forces on the pulley system than grasping because all of the forces were concentrated on the fingertip during pinch but

dissipated across the skin and other phalanges during grasp. (38) In a study done by Talsania et al a computerized digital dynamometer was used to assess the contribution of individual fingers to total grip strength in100 hands from 50 randomly selected healthy subjects. The dynamometer simultaneously recorded force data from each digit (index, long, ring, and small) and cumulative grip directly to a laptop computer. The percentage contribution of each finger force to total grip force was calculated at three successive handle sizes for dominant and non-dominant hands. It was found that individual digital contributions to total grip strength were approximately 25%, 35%, 26% and 15% for the index, long, ring and small fingers respectively. (39)

Trigger Finger as Prodrome to Carpal Tunnel

It is thought that subclinical carpal tunnel syndrome can manifest as trigger finger. In a study done by Garti et al an association between symptomatic compression neuropathy of the median nerve at the carpal tunnel and trigger finger has been reported. They assessed the incidence of increased median nerve latency in subjects with trigger finger. 62 consecutive patients with trigger finger and no signs of symptoms of median nerve compression underwent nerve conduction studies of the median nerve. 63% of the patients had increased distal motor latency in the median nerve. Only 6% of the control group had a borderline value of distal motor latency. (40) An earlier study done by Loong found that 11% of his symptomatic carpal tunnel syndrome patients also presented with symptomatic trigger finger. (41)

Trigger Finger in Neck Injury

One study found a correlation between trigger finger and neck injury in automobile accidents. Among 450 patients examined for neck pain following automobile accidents, carpal tunnel syndrome was diagnosed in four and trigger finger in three. All seven patients sustained cervical sprains and were drivers or passengers in automobiles, which received impacts from the rear. All seven patients reported the onset of hand symptoms during or shortly after the accident. This unusual occurrence, not previously reported, appears to be related to the events, which occurred at the time of the accident. (42)

Trigger Finger in Diabetes

The association between diabetes and trigger finger was first confirmed by Strom in 1977. (43) Strom felt that it was a manifestation of the diabetic hand syndrome, which includes Dupuytren's contracture, carpal tunnel syndrome, and finger joint stiffness. In a study conducted by Blyth et al eighteen of 100 patients undergoing surgery for trigger finger were diabetic. In addition one further patient was diagnosed as suffering from the diabetes 2 years after surgery. Trigger finger was found to be unilateral in 90.2% of the non-diabetic group and in only 61.1% in the diabetic group. The diabetic group tended to have more fingers involved than the non-diabetics. (44) This study confirms the association between diabetes and trigger finger.

Diagnosis of Trigger Finger

In Children

Clinically, the characteristic sign of this condition is difficulty in extending the thumb; snapping of the thumb is not a characteristic sign in children. A tendon nodule may be palpable, but palpation does not produce pain. Trigger finger is found rarely in children, but the highest incidence in children can be found at the age of two to four without a clear preponderance in either gender.(10)

In Adults

Clinically the condition develops gradually; the first signs of stenosis are increased difficulty in flexing and extending the digit as well as pain and discomfort at the flexor surface of the metacarpophalangeal joint. Palpation of the area may increase the pain and on palpation a small tendon nodulus may be felt. When the stenosis is advances, the flexion or extension movement causes the typical snapping feeling or sound as soon as the tight stenotic place has been overcome. A case definition might consider the following.

1. Onset is usually gradual, but some cases may follow trauma or carpal tunnel release surgery. (14-18) The abrupt onset of locking following a specific incident was commonly reported among cases owing to atypical anatomical factors (19-22)
2. Sperling (14) describes the sensations of snapping or locking as "spasmodically arrested, irregular movement." Snapping, clicking, locking, stiffness, and difficulty extending a flexed digit, often with associated

discomfort or pain, are the most prominent symptom qualities. Some cases reported difficulty flexing the affected digit (11,23)

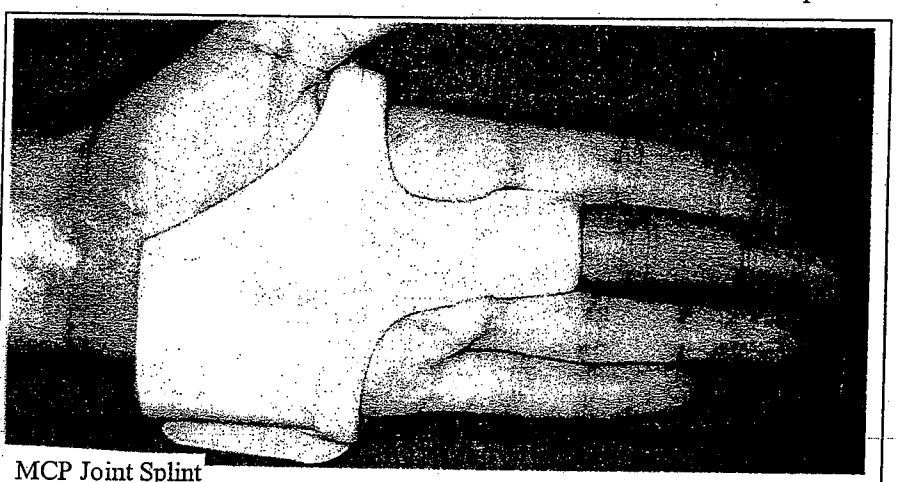
3. In terms of intensity, the snapping sensation may be barely perceptible without any actual triggering, or it May be painful, especially when a triggered digit is forcefully extended (24-26)
4. Localization is to the Ai pulley on the palmar side of the MP joint. (24-26)
5. Radiation has not been reported.
6. There is no reliable symptom pattern. Some patients report more difficulties in the morning compared with other times. (27,28) Symptoms may be intermittent. (11,27)
7. For physical findings, inspection is usually unremarkable unless the digit triggers in the presence of the examiner. Palpation may reveal a clicking or snapping sensation, with range of motion of the digit or detection of tenderness of a nodule on the tendon in the region of the A1 pulley. (29-31) Pain along the flexor tendon with resisted digital flexion or passive stretch has been mentioned rarely. (27)
8. There are no objective confirmatory tests. Laboratory tests and x-rays are generally not done. In a study done by Katzman et. al. (32) They performed a combined retrospective and prospective study on the utility of obtaining radiographs in patients with trigger finger. Ninety-three patients with a total of 110 involved fingers were reviewed. The average age of the patients were 52 years. There were 54 women and 39 men. There were no abnormal findings in 62% of the radiographs. Thirty-one percent had radiographic

abnormalities that were not currently clinically significant. Four percent had radiographic findings that correlated with other clinical problems. No radiographic finding changed their management. This indicates that patients with trigger finger without a history of injury of inflammatory arthritis do not need routine radiographs. Blood tests may be helpful in detecting of verifying the presence of related conditions, such as diabetes, hypothyroidism, of rheumatoid arthritis.(27)

It is important to note that According to Hadler (33) Trigger finger may be confused with the early stages of Dupuytren's contractures. Dupuytren's contractures however can be differentiated due to the fact that the palpable nodules in the palm initially present as painless while in trigger finger they are painful

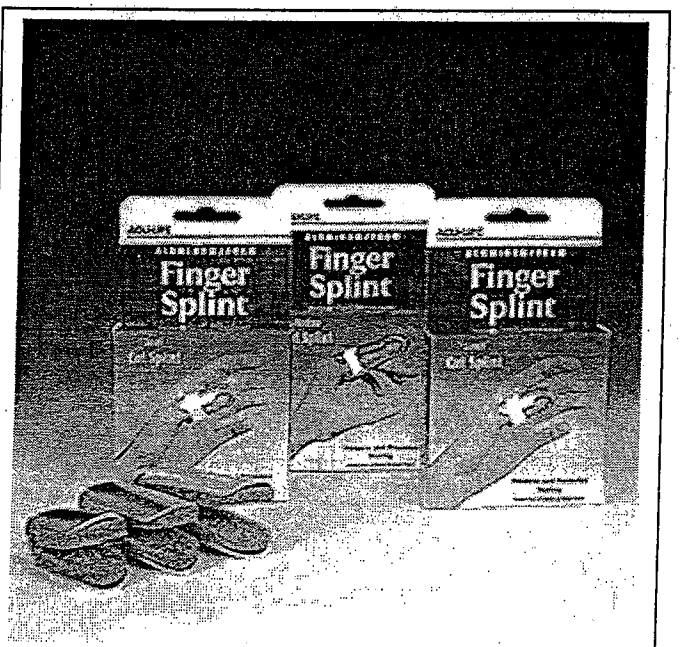
Conservative Treatment of Trigger Finger

1. Ice for five to fifteen minutes at a time on the areas which are most swollen and tender.
2. The patient should eliminate such provocative movements as repetitive grasping or the use of tools that apply pressure over the area. Rosenthal stated that use of palmar pads to distribute forces over a larger area and the use of broader tool handles to reduce the degree of finger flexion can reduce the incidence of trigger finger.(38)



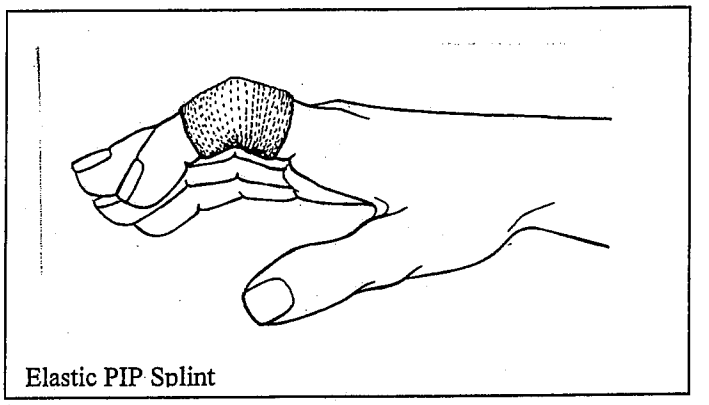
MCP Joint Splint

3. Trigger finger can be managed by immobilizing the MCP joint in extension.(36) The patient is allowed to remove the splint while bathing, while doing twice-daily flexibility exercises, and also while sleeping. Splinting longer than twelve weeks is unlikely to be beneficial and may



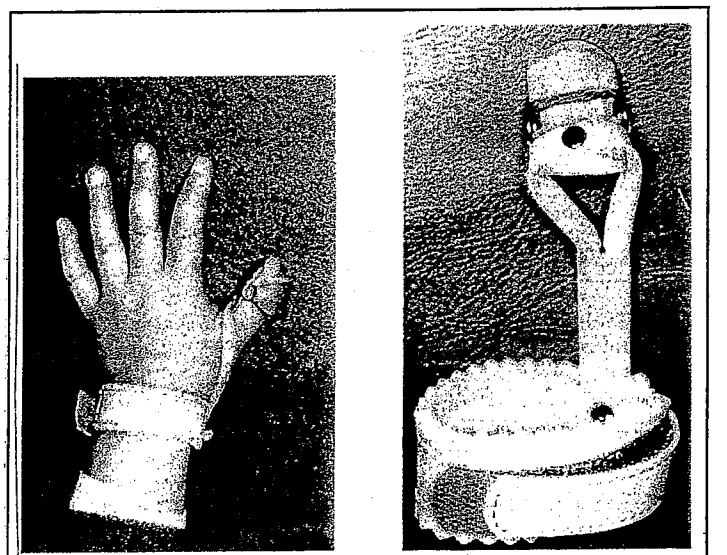
Alumafoam Splint for DIP Joint

lead to joint stiffness. In a second study by Rodgers et al evaluated the efficacy of functional distal interphalangeal joint (DIP) splinting for the treatment of trigger finger. Thirty-on fingers from 21 meat packing plant workers were treated with DIP splinting. All workers returned to work immediately. Eighty-one percent of the digits were treated successfully. They concluded that DIP Alumafoam splinting provided a reliable and functional means of treating work-related trigger finger without lost time from work. This is due to the fact that the Alumafoam splint significantly decreased Flexor digitorum profundus excursion. (45) Alumafoam splints are also inexpensive and easily obtained over the counter at any local Wal-Mart or drug store.



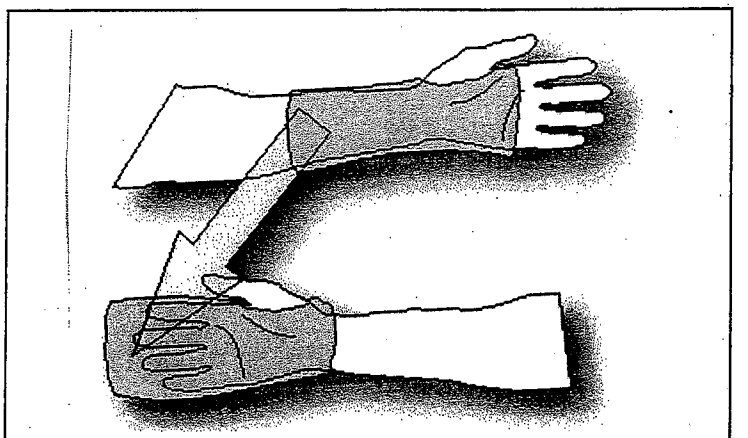
Elastic PIP Splint

In another study done with children 83 trigger digits in 65 children were treated using a modified coil spring splint, which maintains the interphalangeal(IP) joint in neutral extension or hyperextension. Sixty-two digits (75%) were completely healed following splint therapy alone, after an average period of splinting



Modified Coil Spring Splint

for 9.4 months. This shows that splint therapy to maintain the IP joint in neutral extension or hyperextension proved markedly effective in this series.(46) Another form of splinting outlined by Swezey involves a 1-inch-wide elastic band sewn to make a snug sleeve overlying the PIP joint. This permits flexion of the PIP joint to about 60 to 70n degrees. This mild restriction of PIP flexion is compatible with most functions but restricts the full excursion of the flexor tendons and thus minimizes tenosynovial irritation and triggering. (47) If splinting is done only when sleeping one may use a wrist support splint. A wrist splint designed to be worn on the opposite hand may be worn with the forearm end turned out to form a palm side support for the fingers as shown.



Wrist Splint Modification For Trigger Finger

4. Patients with trigger finger should have their hands thoroughly examined to rule out carpal tunnel syndrome. This is due to the strong correlation found between carpal tunnel and trigger finger.
5. It may be appropriate to screen all patients with trigger finger for diabetes. (44) This is especially true when the patient presents with bilateral or multiple trigger fingers.

Conclusion

Trigger finger or stenosing tenosynovitis of the digit is a begnign lesion manifested as painful locking or triggering of the involved finger. Clinically the condition develops gradually; the first signs of stenosis are increased difficulty in flexing and extending the digit as well as pain and discomfort at the flexor surface of the metacarpophalangeal joint. With proper splinting and elimination of provocative movements such as repetitive grasping or the use of tools that apply pressure over the area conservative management of trigger finger may be considered a reasonable treatment option. Spontaneous recovery has occurred in some patients but in most cases the symptoms persist for years if untreated. For patients who have had symptoms less than six weeks, conservative treatment can produce favorable results in up to 81%. Patients with trigger finger should have their hands thoroughly examined to rule out carpal tunnel syndrome. This is due to the strong correlation found between carpal tunnel and trigger finger. It may be appropriate to screen all patients with trigger finger for diabetes. (44) This is especially true when the patient presents with bilateral or multiple trigger fingers.

Bibliography

1. Jozsa L, Kannus P (1997) Human tendons anatomy physiology and pathology. Champaign: Braun-Brumfield

2. Orava S (1980). Exertion inuries due to sports and physical exercise. Academic Dissertation, Kokkola: Osterbottningen Press, Kokkola, Finland, pp. 1-95

3. Renstrom P, Kannus P (1991). Prevention of sports injuries. In: Strauss RH (ed) Sports Medicine. Philadelphia: W.B. Saunders, pp. 307-329.

4. Curwin S, Stanish WD (1984). Tendinitis: Its etiology and treatment.. Lexington: Collamore Press

5. Hess GP, Capiello WL, Poole RM, Hunter SC (1989). Prevention and treatment of overuse tendon injuries. Sports Med 8, 371-384

6. Micheli LJ, Fehlandt AF (1992). Overuse injuries to tendons and apophyses in children and adolescents. Clin Sports Med 11, 713-726.

7. Renstrom P, Johnson RJ (1985). Overuse injuries in sports: a review. Sports Med 2, 316-333.

8. Rodgers WB, Waters PM (1994). Incidence of trigger digits in newborns. J Hand Surg [Am] 19(3), 364-368.

9. Ippolite E (1986b). Insertional tendinopathies, stenosing tenosynovitis, exudative hypertrophic tenosynovitis, peritendinitis, and tendinosis and metaplasia. In: Perugia L, Postacchini F, Ippolito E (eds). The Tendons. Milano:editrice Curtis, pp. 113-213.

10. Weilby A (1970). Tigger finger: Incidence in children and adults and the possibility of predisposition in certain age groups. Acta Orthop Scand 41, 419-423.

11. Fahey JJ, Bollinger JA (1954). Trigger finger in adults and children. J Bone Joint Surg (Am) 36, 1200-1202

12. Grob M, Stockmann M (1951). Stenosing tenosynovitis: A typical affection of early childhood. Acta Pediatr Helvet 6, 112-118.

13. Meachim G, Roberts C (1969). The histopathology of stenosing tendovaginitis. J pathol 98, 187-192.

14. Compere EL. (1933). Bilateral snapping thumbs. Am J Surg. 97,773-777

15. Sperling WP. (1951) Snapping finger: roengten treatment and experimental production. Acta Radiol 37: 74-80

16. Pirotta T. (1964) Locked finger. Med J Aust 34:924-925

17. Hombal JWR. , Owen R. (1970) Carpal tunnel compression and trigger digits. Hand 2:192-196

18. Nasca RJ. (1980) Trigger finger: a common hand problem. J Ark Med Soc 76:388-390

19. Alldred A (1954) Alocked index finger. J Bone Joint Surg 36B:102-103

20. Flatt A (1958) Recurrent locking of an index finger. J Bone Joint Surg. 40A:128-1130

21. Aston JN (1960) Locked middle finger. J Bone Joint Surg. 42B:75-79

22. Janecki CJ. (1976) Triggering of the finger caused by flexor tendon laceration: report of two cases. J Bone Joint Surg. 58A:1174-1175

23. Lapidus PW. (1952) Stenosing tenovaginitis at the wrist and fingers: report of 423 cases in 369 patients with 354 operations. AMA Arch Surg. 64:475-487

24. Webber JB. (1981) Common pain syndromes: upper extremities. Geriatrics. 36:59-69

25. Burger MR. (1982) Painful tendon problems of the hand: trigger thumb, trigger finger, and deQuervain's syndrome. Orthop Nursing. 20-33

26. Tsuyuguchi T. (1983) Splint therapy for trigger finger in children. Arch Phys Med Rehabil. 64: 75-76

27. Gottlieb NL. (1991) Digital Flexor tenosynovitis: diagnosis and clinical significance. J Rheum. 18:954-955.

28. Lenggenhager K. (1969) The genesis and therapy of trigger finger. Minn Med. 11-14

29. Griggs SE. (1995) Treatment of trigger finger in patients with diabeties mellitus. J Hand Surg. 20A:797-789

30. Chammas M. (1995) Dupuytren's disease, carpul tunnel syndrome, tigger finger and diabetes mellitus. J Hand Surg. 20:109-114.

31. Ranney D. (1995). Upper limb musculoskeletal disorders in highly repetitive industies: Precise anatomical physical findings. Ergonomics. 38:1408-1423.

32. Katzman BM, Steinberg DR, Bozentka DJ, Cain E, Caligiuri DA, Geller J. (2000). Utility of obtaining radiographs in patients with trigger finger. Am J Orthop. Dec;28(12):703-5

33. Hadler NM. (1999). Occupational Musculoskeletal Disorders. 2nd ed. Philadelphia: Lippincott, Williams & Wilkins; 154

34. Zelle OL, Schnepp KH. (1936) Snapping thumb: tenovaginitis stenosans. Am J Surg. 33:321-322.

35. Lapidus PW. (1953) Stenosing tenovaginitis. Surg Clin North Am. 33;1317-1347

36. Patel MR, Bassini L. (1992) Trigger finger and thumb: When to splint, inject or operate. J Hand Surg. 17;110-113

37. Gorsche R, Wiley JP, Renger R, Brant R, Gemer TY, Sasyniuk TM. (1998) Prevalence and incidence of stenosing flexor tenosynovitis in a meat-packing plant. 40(6);556-560

38. Rosenthal EA. (1987) Tenosynovitis: tendon and nerve entrapment. Hand Clin. 3;585-609

39. Talsania JS, Kozin SH. (1998) Normal digital contribution to grip strength assessed by a computerized digital dynamometer. J Hand Surg. 23B;162-163

40. Garti A, Velan G, Moshe W, Hendel D. (2001) Increased median nerve latency at the carpal tunnel of patients with trigger finger. Acta Orthop Scand. 72(3);279-281

41. Loong SC. (1977) The carpal tunnel syndrome: a clinical and electrophysiological study of 250 patients. Clin Exp Neurol. 14;51-65

42. Guyon MA, Honet JC. (1977) Carpal tunnel syndrome or trigger finger associated with neck injury in automobile accidents. 58(7);325-327

43. Stom L. (1977) Tigger finger in diabetes. J Med Soc New Jersey. 74;951-954

44. Blyth MJG, Ross DJ. (1996) Diabetes and trigger finger. J Hand Surg.(BR) 21B(2);244-245

45. Rodgers JA, McCarthy JA, Tiedeman JJ. (1998) Functional distal interphalangeal joint splinting for trigger finger in laborers: a review and cadaver investigation. Ortho. 21(3); 305-309

46. Tsuyuguchi Y, Tada K, Kawaii H. (1983) Splint therapy for trigger finger in children. Arch Phys Med Rhabil. 64(2);75-76

47. Swezey RL. (1988) Essential Therapies for joint, soft tissue and disk disorders. Hanley & Belfus. Philadelphia