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by [Kurppa K](#), [Waris P](#), [Rokkanen P](#)

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Peritendinitis and tenosynovitis

A review

by KARI KURPPA, M.D.,¹ PEKKA WARIS, M.D.,¹ and PENTTI ROKKANEN, M.D.²

KURPPA, K., WARIS, P. and ROKKANEN, P. Peritendinitis and tenosynovitis: A review. *Scand. j. work environ. & health* 5 (1979): suppl. 3, 19–24. A critical review on the pathogenesis, etiology and occurrence of peritendinitis and tenosynovitis has been presented.

Key words: etiology, occurrence, pathogenesis, peritendinitis, tenosynovitis.

The muscle-tendon unit may be inflamed in the area of the tendon sheath (tenosynovitis) or outside of it, in the paratenon and the muscle-tendon junction (peritendinitis). Both variants resemble each other with regard to their clinical picture, but the location is different. Although these diseases are often seen at physicians' offices and they are expensive, in economic terms, due to lost workdays, little is known of their occurrence rates, pathogenesis, or etiology. Yet, most investigators think that overexertion of the muscle-tendon unit is the main cause of morbidity. So far, this hypothesis has not been formally tested.

TERMINOLOGY

Terminology on the disorders of the tendon and structures associated with it is still unsettled. As Griffiths (5) remarked

years ago, the word tenosynovitis is used so vaguely that it has come to mean little more than a painful disability arising in or around a tendon or a group of tendons. According to *Dorland's Illustrated Medical Dictionary* (3), the names tenosynovitis, tendovaginitis, tenovaginitis, and peritendinitis are synonymous.

We prefer using the term tenosynovitis as a name for the inflammation of the tendon sheath. After scrutinizing the histological findings described in the literature, we, like some others (8, 22), distinguish it from peritendinitis, which is the name for the inflammation of the paratenon, muscle-tendon junction, and adjacent muscle tissue.

Tendinitis is the name for the inflammation of the tendon tissue which may cause thickening of the tendon and even lead to its locking ("trigger finger"). Insertion tendinitis means inflammation at the insertion point of the tendon to the bone. Tenosynovitis may develop into stenosing tenosynovitis, in which the tendon sheath is narrowed, e.g., de Quervain's disease, which is stenosing tenosynovitis of the long and short abductor muscles of the thumb.

Especially with regard to older publications, it is sometimes impossible to de-

¹ Institute of Occupational Health, Helsinki, Finland.

² Institute of Clinical Sciences, University of Tampere, and the Tampere Central Hospital, Tampere, Finland.

Fig. 1. The muscle tendon unit.

cide whether the article concerns cases of tenosynovitis or peritendinitis or both entities. In our present review, we have used the term muscle-tendon syndrome to comprise both variants.

TENDON ANATOMY AND LOCATION OF TENOSYNOVITIS AND PERITENDINITIS

The anatomy of the tendon and the location of tenosynovitis and peritendinitis are presented in fig. 1. These diseases may occur in all significant muscle-tendon units of the extremities. The usual sites are described in fig. 2.

Tenosynovitis and tendinitis may affect the long head of the biceps tendon in the intertubercular groove around the shoulder joint. Tenosynovitis is located more commonly in the wrist and ankle regions, where tendons cross tight ligaments and each other. Tendon sheaths of tendons from finger and wrist extensors and anterior tibial and peroneal muscles are the most commonly afflicted (10, 22, 24) (table 1).

TENOSYNOVITIS PERITENDINITIS

Fig. 2. The most common areas of tenosynovitis and peritendinitis affliction. (1. biceps muscle of the arm, 2. ulnar extensor muscle of the wrist, 3. long abductor muscle of the thumb, 4. extensor muscles of the fingers, 5. extensor muscles of the thumb, 6. radial extensor muscles of the wrist, 7. extensor muscle of the toes, 8. peroneal muscles, 9. anterior tibial muscle, 10. long extensor muscle of the great toe, 11. supraspinous muscle, 12. long radial extensor muscle of the wrist, 13. short radial extensor muscle of the wrist, 14. extensor muscles of the fingers, 15. ulnar extensor muscle of the wrist, 16. long abductor muscle of the thumb, 17. extensor muscles of the thigh, 18. quadriceps muscle of the thigh, 19. patellar ligament, 20. anterior tibial muscle, 21. long extensor muscle of the toes, 22. triceps muscle of the calf, 23. peroneal muscles)

Table 1. Location of peritendinitis according to Rais (13).

Location	Right	Left	Bilateral
Short extensor and long abductor muscles of the thumb	77	31	5
Radial extensors of the thumb and the wrist	12	2	—
Radial extensors of the wrist	3	5	—
Extensors of the fingers	10	12	3
Achillis tendon	10	13	2
Anterior tibial muscle	7	10	—
Extensors of the toes	8	4	—
Other sites	5	2	2
Total	132	79	12

Two-thirds of the cases of peritendinitis have been reported to occur in the upper extremities (7, 13, 22), usually in the arm (table 2). Peritendinitis of the patellar tendon is relatively common among runners, basketball players, and high jumpers. The Achilles tendon of athletes may contract tendinitis, peritendinitis, insertion tendinitis, or bursitis near its insertion (24), but not tenosynovitis because it has no tendon sheath.

Peritendinitis seems to be a much more commonly occurring variant than tenosynovitis. Thompson and others (22) reported on a series of 544 patients, 419 of whom were diagnosed as having peritendinitis and the remaining 125 patients being afflicted by tenosynovitis. In a statistical summary of a Swiss insurance company, peritendinitis comprised 70–80 % of all the cases in the muscle-tendon syndrome group (18). Work-related exertion was considered to be the most important etiologic factor.

ETIOLOGY AND OCCURRENCE

The most common causes of peritendinitis and tenosynovitis are thought to be overexertion, sprain, or local blunt trauma.

Overexertion

The speed and force of muscle effort are often regarded as decisive for the develop-

Table 2. Location of traumatic tenosynovitis according to Thompson et al. (21).

Tendon sheath	Patients
Extensors of the fingers	66
Anterior tibial muscle	21
Flexors of the wrist and fingers	15
Peroneal tendons	10
Extensors of the toes	7
Other sites	6
Total	125

ment of the muscle-tendon syndrome. During a period of eight months, 189 cases of "tenosynovitis" of the upper extremities were recorded among a group of 700 packers in a tea factory (10). It was calculated that packers performed 50 to 60 hand movements during 1 min and 7,600 to 12,000 hand movements during a workday.

The inexpediency of the movements and the use of muscles would seem to be important in the development of the disease. New beginners and workers who resume work after an absence due to vacation or a long sick leave have been reported to contract the disease more often than others. Morbidity has been observed to double after summer holidays (1, 7, 10, 22). Morbidity is not restricted to new beginners or to workers returning from holidays, however. Thompson and others (22) reported that unaccustomed work or return to work after a long absence was the cause of the disease in only half of the cases (table 3).

Numerous new cases have been diagnosed among soldiers and town residents

Table 3. Etiology of peritendinitis and tenosynovitis according to Thompson et al. (22).

Etiologic factor	Patients	Percentage
Unaccustomed work or resumption of work after absence	258	47
Local "strain" ^a	79	14
Direct local trauma	76	14
Repetitive movement ^b	53	11
Cause unknown	78	14
Total	544	100

^a "Strain" signifies unusually heavy manual work requiring strength, dexterity, and speed.

^b For example, filing, hammering, assembly work. In addition, simple repetitive movement was regarded as an associated factor in 32 % of all cases.

commanded to farm work during war time (4, 12, 21). Sudden increases in training may bring about the disease in athletes (2). Bicipital tendinitis has been reported to be mechanical (17), and it has been found as an isolated entity or as a disorder secondary to lesions of the rotator cuff (11, 19).

Blunt trauma and sprain

Half of the 78 patients studied by Howard (7) had a history of blunt trauma of the hand, arm or leg before their contraction of peritendinitis. They had continued working for 1 to 14 d (mean 3 d) before the start of symptoms. The relation of a blunt trauma or sprain to the occurrence of peritendinitis and tenosynovitis has been reported also in other studies (9, 14, 22).

Other causes

Supraspinous tendinitis is generally regarded as being associated with hypovascular degeneration of the tendon with age, and the role of mechanical factors has been thought to be secondary (23). Tenosynovitis may be rheumatic, tuberculous, luetic, or gonorrheal in origin. Wounds extending to the tendon sheath may cause a purulent inflammation. Gout has been suggested to be a cause of tenosynovitis (22), as well as mechanical irritation

caused by an anomalous short extensor muscle of the toe (15). Although morbidity of tenosynovitis among patients with rheumatic arthritis is high (6), all the mentioned causes combined are thought to represent a minor etiologic role among the patients.

PATHOGENESIS

According to Lipscomb (9), microscopic and macroscopic changes vary in degree and depend upon the duration rather than the location of the disease.

In traumatic tenosynovitis, exudation flows into the tendon sheath and leads to a deposition of fibrin which in turn may organize and result in the formation of adhesions (9, 16).

Lipscomb (9) found that the involved sheath of the tendon was greatly thickened and had lost its normal pearly luster. In some of the 15 cases studied, the synovial lining was proliferated, but in most cases the synovial layer was almost or totally absent. In all cases the fibrous tissue had increased. No changes in the tendon tissue were observed. Other authors have found thickening or constriction (14) or fraying (15) of the tendon.

In peritendinitis, the pathological changes seem to be localized in the paratenon and muscle tissue, especially at the muscle-tendon junction. No changes have usually been observed in tendons or in tendon sheaths (7, 13, 22). However, local

degeneration and disappearance of collagen of the tendon have been described in connection with peritendinitis of the patellar tendon of athletes (24). Both animal experiments (10, 13) and human biopsies have indicated the existence of edema and the accumulation of fibrin in the paratenon and muscle interstitium. Sparse fibroblastic proliferation has been observed in the paratenon after 1—5 d from the beginning of the disease, and abundant fibroblastic proliferation with collagen and new capillary formation after 11—25 d (13). Fibrinoid degeneration probably takes place in the paratenon. It becomes thicker and adhesions appear between it and the tendon (20).

In the biopsy specimens of peritendinitis patients, Howard (7) found edema of the peritendinous areolar tissue and muscle (particularly at the muscle-tendon junction) thrombosis of the venulae and hemorrhages in the muscle interstitium, glycogen depletion, lactic acid retention, and degenerative changes in the muscle. Based on these biopsies and his clinical findings, Howard concluded that peritendinitis is the result of the exhaustion of particular muscle groups by unaccustomed and unremitting toil, or by continued, routine, accustomed labor following direct trauma.

Rais (13) studied both rabbits and biopsy specimens from peritendinitis patients. His findings lend support to Howard's opinion. Rais concluded that, as a result of muscular fatigue or direct violence to muscles, circulatory disturbances and edema of the muscles and their peritenon develop. Depending on the severity of the damage, degenerative changes develop in the muscles. Fibrin is deposited in the edematous peritenon and interstitially in the muscles.

DISCUSSION

Most studies on peritendinitis or tenosynovitis have concentrated on the results of various treatment methods, and they discuss etiology only in passing. The reports that handle etiology or pathogenesis more thoroughly are scarce. There seems to be

no studies on etiology with an adequate control group or, indeed, with any control group at all. The situation is deplorable from the point of view of insurance medicine, which should be able to found its compensation practice on the proved existence of a cause-effect relationship between disease and work.

Thus, the hypothesis of the work-relatedness of peritendinitis or tenosynovitis has not been scientifically tested. It is well known that certain types of manual tasks and these diseases correlate clinically, but this correlation does not necessarily imply the existence of a causal relationship. Whatever the cause of the disease might be, it is conceivable that manual workers experience more nuisance from the symptoms than sedentary employees. Therefore, manual workers are probably more prone to contact a doctor when afflicted, and the probability of contact increases the more the nature of the job demands dexterity and the use of the diseased extremity. Thus the clustering of workers from certain types of jobs in the statistics or in physicians' offices does not prove much as far as the etiology of peritendinitis or tenosynovitis is concerned.

It has also been claimed that there is a preponderance of women among the patients. Evidence to support this opinion is anecdotal or nonexistent in a scientific sense. Selection may play an important role in uncontrolled studies.

There are no controlled studies which could give us information concerning possible sex differences. Nor is the claim that the peak incidence is found in the age group around 40 years based on proper occurrence studies.

A number of uncontrolled studies and reports has accumulated during the past 100 years, and most of them take the position that the disease is usually caused by exertion with or without a precedent blunt trauma or sprain. There is little evidence contrary to this hypothesis. Yet, the supposedly causative task or job has seldom been analyzed with regard to its biomechanical components. A few exceptions exist, but usually the job has concisely been described as "repetitive" or "strenuous" without an attempt at a more precise ergonomic analysis.

A lot of work must be done in order to increase our knowledge about peritendinitis and tenosynovitis. At present, reliable data are rare and too incomplete to form a basis for a sound understanding of these diseases. We need prospective longitudinal studies which include proper control groups. The diagnosis should be verified by objective clinical findings and the results, possibly in favor of increased morbidity, judged as to whether they are due to a precipitative or causative nature of the job under study. An accurate ergonomic analysis of the biomechanics of the tasks is also necessary so that possible causative factors can be adequately identified and eliminated in future job designs.

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