Ergonomics considerations in hand and wrist tendinitis

The objectives of this article are to present (1) a historical perspective on hand and wrist tendinitis in workers, (2) new data that demonstrate a relationship between the repetitiveness and forcefulness of manual work and the prevalence of tendinitis, (3) possible biomechanical factors in tendinitis, and (4) possible job modifications for the prevention of tendinitis. Numerous studies during the last 100 years show that tendinitis is a major cause of worker suffering and workers' compensation in intensive hand work. Epidemiologic data show that the risk of hand and wrist tendinitis in persons who perform highly repetitive and forceful jobs is 29 times greater than in persons who perform jobs that are low in repetitiveness and force. A possible factor in this relationship is viscous deformation of the tendons and adjacent tissues. Although these data suggest that the risk of tendinitis among workers can be reduced by reduction of the repetitiveness and the forcefulness of the work, this hypothesis has not yet been fully tested. (J HAND SURG 1987;12A[2 Pt 2]:830-7.)

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Chronic tendon and nerve disorders, such as carpal tunnel syndrome and tendinitis, are major causes of lost work and workers' compensation in some industries. Chronic nerve injuries, such as carpal tunnel syndrome, are perhaps the most commonly cited problems in both the scientific literature and the public media. This may be because peripheral nerve injuries are particularly disabling and because there are more objective diagnostic tests for nerve impairments than for tendon disorders. As a result, there is often a tendency to overlook chronic tendon disorders in the workplace. This is unfortunate, because disorders of the tendons and the tendon accessory tissues are major occupational health problems in their own right and because they might lead to chronic nerve disorders, such as carpal tunnel syndrome.

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The literature about chronic musculoskeletal disorders goes back at least 200 years to Ramazzini[1] who in 1717 described tham as ". . . harvest of diseases . . ." ascribed to ". . . certain violent and irregular motions of the body . . ."[1]

Later studies began to focus on specific pathologic conditions, pathogenesis, morbidity, diagnosis, and treatment. In 1893, Gray's *Anatomy* read: "The tendons of the extensor muscles of the thumb are liable to become strained and their sheaths inflamed after excessive exercise, producing a sausage-shaped swelling along the course of the tendon and giving a peculiar creaking sensation to the finger when the muscle acts. In consequence of its often being caused by such movements as wringing clothes, it is known as "washerwoman's sprain."[2-4] de Quervain described this condition in 1895, and it now bears his name.[5]

As early as 1927, Zollinger[6] reviewed the records from a large Swiss insurance company and found 929 cases of crepitant tenosynovitis; most of the cases were associated with repeated trauma. Conn[7] reported that in 1930, a year interrupted by layoffs and spasmodic activity, tenosynovitis accounted for 0.99% of the total days lost from all causes. In 1942 Pozner[8] reported 20 cases among 70 soldiers in which the radial extensors of the wrist were affected during harvesting. In the same year, Flowerdew and Bode[9] described 16 cases among

52 office workers doing farm work in which the extensors of the wrist and fingers were affected. In 1943 Reed and Harcourt[10] reported 70 cases that accounted for 0.54% of all visits at an industrial clinic. Thompson and associates[11] reported that 40 cases occurred annually, with an average of 21 lost work days per case, in a Vauxhall Motors factory that employed 12,000 workers.

Hymovich and Lindholm[12] reported 62 chronic or cumulative trauma disorders that occurred during 6 years in an electronics firm that had, on the average, a work force of 160 people. This resulted in an incidence rate of 6.6 cases per 200,000 work hours and a severity rate of 41 lost work days per 200,000 work hours. Two of these disorders were classified as permanent disabilities. Wisseman and Badger[13] reported 104 Occupational Safety and Health Administration (OSHA) reportable disorders in a film products plant of 3300 workers; these disorders included 84 cases of tendinitis, ten cases of ganglionic cysts, two cases of epicondylitis, four cases of bursitis, one case of myositis, and one case of thoracic outlet syndrome. Eighty-four percent of these cases occurred in two departments in which 250 people were employed; this resulted in an incidence rate of 7.0 cases per 200,000 work hours. Jensen and colleagues[14] analyzed 1979 workers' compensation claims from six states and found that more than 6% of the claims were related to inflammation or irritation of the joints, tendons, or muscles, diseases of the peripheral nerves, or ganglia. These injuries were all related to repetitive pressure, voluntary motions, overexertion, lifting, pulling, or throwing; in 1979 the average cost per claim was $618.

Although morbidity studies that were based on plant medical visits and compensation reports indicate a problem of epidemic proportions, the magnitude is probably underestimated. Fine et al.[15] found from seven to 31 more cumulative trauma disorders through an analysis of personal benefit records than from plant medical and compensation records.

Luopajarvi et al.[16] surveyed 163 female assembly line packers in a food production factory and 143 female shop assistants. Symptoms of tenosynovitis or peritendinitis were found in the wrists or forearms of 56% of the assembly line packers and 14% of the packers.

These studies show that tendinitis, peritendinitis, tenosynovitis and related hand and wrist disorders are common among workers who perform intensive work with their hands. Many factors, both occupational and nonoccupational, have been reported to cause, precipitate, or aggravate these conditions. As a result, some controversy has arisen among investigators searching for the causes of hand and wrist disorders. It is almost always possible to find cases to argue for one factor or set of factors over another. Perhaps one of the greatest controversies pertains to the relative contribution of occupational and nonoccupational factors. Examples of occupational factors include repetitive exertions, forceful exertions, mechanical insult, awkward postures, and vibrations. Examples of nonoccupational factors include congenital defects, acute injuries (occupational or nonoccupational), chronic diseases, aging, gender, and recreational factors. This article considers the industrial type of population and discusses the biomechanical basis of excess morbidity of wrist tendon disorders and carpal tunnel syndrome.

Methods

A cross-sectional study was undertaken to evaluate the relationship between repetitiveness, forcefulness, and selected cumulative trauma disorders of the hand and wrist. The details of this study are described by Armstrong and associates[17] and Silverstein and colleagues.[18, 19] A total of 652 workers were selected from each of four combinations of force and repetitiveness at seven work sites. Work sites included electronics, sewing, appliance, bearing fabrication, bearing assembly, and investment molding plants. High-repetitive jobs were defined as those with a cycle time of less than 30 seconds or with more than 50% of the cycle time involved in performing the same motion pattern. Low-repetitive jobs were those with a cycle time of more than 30 seconds and with less than 50% of the cycle time involved in performing the same kind of motion pattern. High-force jobs were those with estimated average hand force requirements of more than 40N and low-force jobs were those with estimated average hand force requirements below 10N. Peak hand forces were estimated from the weight of tools and materials and then verified by means of surface electromyography.[20] Numerous schemes were considered for characterizing hand force: peak force, average force, root mean square force, etc. (See Armstrong and associates[17] and Silverstein and colleagues.[18, 19]) An adjusted force, cycle variance/cycle mean + cycle mean, was found to provide the best dispersion among jobs.

The assessments of worker health were focused mainly on de Quervain's disease, trigger finger, tendinitis, and tenosynovitis. Standardized interviews and noninvasive physical examinations are described by Fine and Silverstein.[21] Diagnostic criteria for these conditions were as follows:

1. de Quervain's disease.[22, 24] The interview indicated pain in the anatomic snuffbox that might radiate

Table I. Prevalence of hand and wrist tendinitis based on interview and physical examination by job repetitiveness and force category (n = 652)

Jobs	*LOF.LOR*	*HIF.LOR*	*LOF.HIR*	*HIF.HIR*	*Total*
Number of workers	157	195	143	157	652
Number of cases	1	6	5	17	29
% affected	0.6	3.1	3.5	10.8	4.4
Odds ratio[29]	1.0	6.1	3.3	29.4*	

*$p < 0.001$.

up the forearm; no history of fracture or radial wrist fracture; the symptoms lasted more than 1 week or occurred more than 20 times in the last year. The physical examination ruled out radial nerve entrapment. There was a positive Finkelstein test with a pain score of 4 or greater (pain scored 1 to 8).

2. Trigger finger.[25] The interview showed that the subject's finger locked in extension or flexion and required assistance in locking; a nodule was present on the tendon. The physical examination revealed a palpable nodule at the base of the digit and locking of digit in flexion or extension.

3. Tendinitis and tenosynovitis.[11, 26, 28] The interview indicated localized pain and/or swelling over muscle-tendon structure that lasted more than 1 week. The physical examination revealed pain increased by resisted motions, possibly fine crepitus on passive range of motion (ROM), no pain on passive range of motion; pronounced asymmetrical grip strength, more than 4 kg.

Results

There were 45 (6.9% subjects; 2.5% of the males and 12.2% of the females) who fulfilled the criteria for tendinitis in the interview and 29 (4.4%) subjects with hand-wrist tendinitis disclosed by the physical examination and the interview. For the purposes of this discussion, only subjects with positive findings in the interview and on physical examination will be considered. The prevalence of hand-wrist tendinitis by force and repetitiveness category shown in Table I ranged from 0.6% in the low-force–low-repetitive jobs to 10.8% in the high-force–high-repetitive jobs (Table I). Similar associations between force, repetitiveness, and prevalence were observed for both males and females, although the overall prevalence was significantly higher in females (7.8%) than in males (1.7%) resulting in a job-adjusted odds ratio[29] of 4.3 for females as compared with males. There were no significant associations with personal factors (use of birth control pills, hysterectomy, oophorectomy, recreational activities) or other work factors (exposure to vibrations and work posture). A significant interaction between sex, age, and years on the job suggested that the risk of hand-wrist tendinitis might actually decrease with an increased number of years on the job, but this effect was too small to merit further discussion.

Differences in posture were examined by comparing the percentage of time spent in various postures between jobs in which there were workers with tendinitis and those in which there were no workers with tendinitis. No significant differences ($p < 0.05$) were found for the percentage of work time spent in wrist flexion, ulnar deviation, flexion and ulnar deviation, pinching, and flexion with pinching. Significant differences in posture were observed between males and females and may, in part, account for the significant gender factor.

Discussion

This study demonstrates a highly significant association between recognized signs and symptoms of hand-wrist tendinitis and the repetitiveness and forcefulness of manual work. The plant-adjusted odds ratio for the high-force, high-repetitive group was 29.4 compared with the low-force, low-repetitive group ($p < 0.001$, Table I). Hand and wrist posture and vibration exposure were not significant factors. With the exception of gender, none of the nonoccupational factors was significant.

The conclusion that women are at greater risk for the development of hand and wrist tendinitis has been suggested by other studies, as well as this one. In many cases, this conclusion was made on the basis of clinical experience in which more female than male patients were seen by a particular clinician or in a particular clinic.[23, 30-34] The study by Faithfull and Lamb,[32] in which 70 of 80 patients with de Quervain's disease who were seen in the Hand Clinic at the Royal Infirmary in Edinburgh were female, is typical. A noteworthy exception is the study by Thompson and associates,[11] who reported on 544 patients from the Vauxhall Motors Ltd and Lutton and Dunstable Hospital with peri-tendinitis and tenosynovitis; 490 of these patients were males. Aitken[35] said: "Actually there is no adequate explanation of why the condition does not occur more commonly in males." A large proportion of women in a clinical series may reflect more the social and reporting differences between males and females than an underlying difference in risk.[15]

Some investigators have used biomechanics to argue that women are exposed to higher stresses than men and, as a result, are at higher risk of a chronic tendon

injury.[29, 36-39] Biomechanical aspects of tendon trauma are discussed below. It can be argued that women may be exposed to higher stresses because the average dimensions of the female hand are less than those of the male hand. Other investigators have used physiologic considerations to argue that women are at greater risk than males.[38, 40] These arguments are generally based on the observation that women over the age of 50 years predominate in a given clinical series. None of the obvious physiologic female factors, such as the use of birth control pills, hysterectomy, or oophorectomy, were significant in this study.

Finally, it is important to put the contribution of gender into proper perspective with the occupational factors. The risk of hand and wrist tendinitis in this study was 4.3 times greater among females than males, while the risk associated with repetitiveness and forcefulness of work was between 29 and 37. This finding is very similar to that of Wisseman and Badger[13] and findings discussed by Armstrong.[41] The population in the departments under study was 49% female while the population of the diseased workers was 73% females. A standardized odds ratio shows that the risk of a repetitive trauma disorder developing in females was 4.6 times that of males. Thus, the risk to females is slightly greater than to males but substantially less than the risk associated with certain types of work.

The small risk associated with gender in the workplace could be more a matter of the design of the workplace than one of physiology. Since the stature of the average female is less than that of the average male, women often have to work in a different posture than that of their male counterparts and may be exposed to greater stress than males.[42, 43] Unfortunately, it was not possible to analyze enough postural data in the present study to compare within-job postural differences between males and females.

No significant associations were found between the prevalence of hand and wrist tendinitis and age in the present study. This finding is contrary to the commonly held idea that tissue vitality decreases with age and the risk of tendinitis increases.[38] This finding is in agreement, however, with the findings of Wilson and Wilson,[44] who reported that the age and gender distribution of 88 patients with tenosynovitis from the Stanton Ironworks Company, Ltd. closely corresponded to that of the plant. Similarly, Wisseman and Badger[13] reported that the median age of workers with chronic hand and wrist injuries in their study was 23 years, while the median age of the unaffected workers in the same departments was 24 years.

Without question, repetitiveness and forcefulness of work are the most important factors of hand and wrist tendinitis in this study population of 652 workers. This finding coincides with studies of other occupational groups. In 1927, in 189 cases of tenosynovitis of the upper extremities among a group of 700 packers in a tea factory, it was calculated that the packers performed 50 to 60 hand movements during 1 minute and 7600 to 12,000 per shift.[45, 46] Hammer[47] concluded that human tendons will not tolerate more than 1500 to 2000 manipulations per hour. Kuorinka and Koskinen[48] found that the number of symptoms of muscle-tendon disorders increased as the number of parts handled per year increased from less than 200,000 to more than 300,000. Luopajarvi et al.[16] found that the prevalence of muscle-tendon syndromes in the hands of assembly line packers was 56%, while the prevalence in shop assistants was only 14%. The assembly line work required that the fingers and hands be used constantly at the pace of the machine, up to 25,000 cycles per work day. Armstrong et al.[49] investigated jobs in a poultry-processing plant in which the incidence of upper extremity musculoskeletal complaints was 17.4 cases per 200,000 work hours; 14,120 cuts were required to bone as many as 3780 turkeys per shift. In 1974 Maeda[50] attributed a 20-year growth in the incidence of neck-shoulder-arm disorders to "rationalization" work in Japan that had increased the repetitiveness of work. Perhaps the ultimate example of this is the keyboard data entry device, such as the computer terminal and word processor that have been the subjects of much controversy.[51, 52]

The relationship between signs and symptoms of hand and wrist tendinitis and related disorders is well established. The question then becomes: what is the mechanism? Unfortunately, there have been relatively few controlled studies of how tendons respond to chronic loads. Although the complete pathogenesis is far from known, it probably involves both biomechanical and physiological elements.

Pathogenesis

Tendons function as mechanical links that transmit forces, stabilize movements, and move the kinematic chains of the extremities. As a result, tendons are subjected to tensile stresses by the muscle and to compressive and shearing stresses from adjacent bones and ligaments. Tensile stresses are related to the contractile force of the muscle and the cross-sectional area of the tendon. The compressive stress is related to the force, the radius of curvature, and the area of contact of the tendon.[39] The shearing force is related to the tendon force and velocity, the area of contact, and the friction between the tendon and the adjacent surface. Tendons respond mechanically to these stresses by becoming deformed.

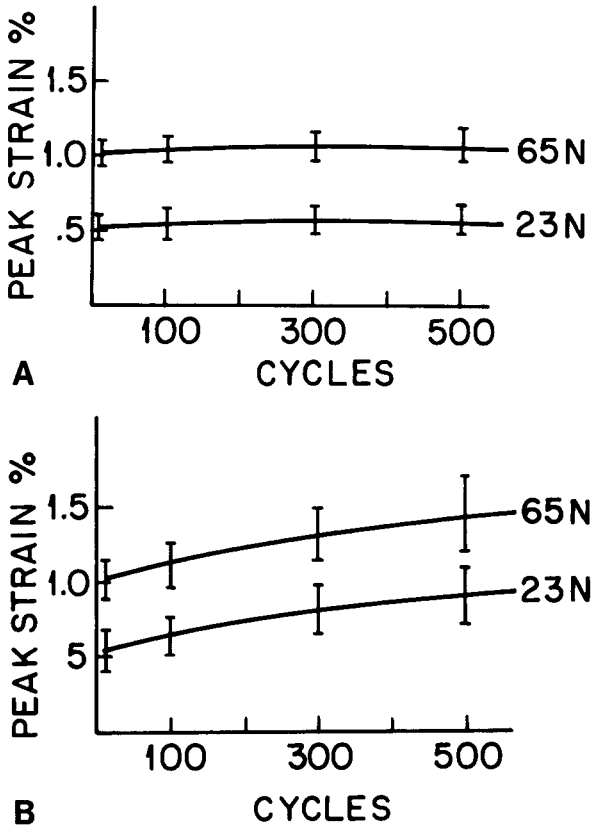

Fig. 1. A, Strain responses for profundus tendons subjected to 23N and 65N loads for 1 second at 10-second intervals show very little viscous deformation. **B,** Strain responses for tendons subjected to loads for 9 seconds at 10-second intervals show significant viscous deformation.

The deformation of tendons subjected to tensile loads has been studied extensively. The deformation that occurs immediately on the application of or the removal of stress is referred to as elastic strain; strain that occurs after the application of load is referred to as viscous deformation or *creep*.[53]

$$\text{Total strain} = \text{Strain}_{\text{elastic}} + \text{Strain}_{\text{viscous}}$$

Viscous deformation or creep has been explained by some investigators as cumulative microdamage or as cumulative microfailure of the molecular links between the tissue matrix and the filler material.[54] Some investigators refer to the viscous deformation that occurs in the first 100 msec after the stress is applied as transition creep and attribute it to shearing of the gel.[55, 56] Deformation that occurs in the time after 100 msec is referred to as fibrillar creep. Similarly, strain recovery after cessation of the load is also dependent on time. The total strain at any time can be determined by integrating the viscous component and adding the elastic component.

A study of tendon strain in situ was undertaken to determine how much strain might occur in submaximal exertions in light work activities. The details of the study are described in Goldstein et al.[56] The profundus tendons of 12 postmortem hand specimens from four women and three men between the ages of 55 and 72 were exposed proximal and distal to the carpal tunnel. Strain gauges and pneumatic cylinders were attached to the exposed tendons. The tendons were then subjected to various regimens of force and frequency to determine their elastic and viscous stress-strain functions. Data for two load levels, 23N and 65N loads, at two load regimens of 1-second loads and 9-second recoveries and 8-second loads and 2-second recoveries are shown in Fig. 1. The data show that if the time between successive loads is not sufficient, there will be significant creep in tendons subjected to physiologic loads. The 9-second recovery time following the 1-second load time is sufficient for nearly complete viscous recovery, so that after 500 work-rest cycles there is no significant creep. When the work time is increased to 8 seconds and the recovery time is decreased to 2 seconds, the time is insufficient for complete recovery, and there is significant creep. After 500 cycles, the creep is equivalent to the elastic strain produced by an 80% increase in load. Viscoelastic tendon models may be used in the future to compare the trade-off between the magnitude of exertion, rest time, and total work time in the occupational setting.

In addition to mechanical responses, tendons also respond physiologically. Physiologic responses include metabolic, circulatory, and adaptive changes. These responses may be greatest in the accessory tissues, synovial and mesenteric membranes, and adjacent ligaments. Gray[2] and de Quervain[5] were among the earliest modern investigators to describe these changes. Hauck[57] reported "serous edema, with congestion of the peritendinous areolar tissue" but without naked-eye changes in the tendons themselves. Howard[58] reported "jelly-like" changes in the tendon-muscle junction, even when the tendons and sheaths appeared normal to the naked eye. Typical responses include thickening, proliferation of fibrocytes and fibrous connective tissue, destruction of synovial membranes, and adhesions.[23, 24, 40, 59, 60] Armstrong et al.[60] reported that proliferation of fibrous connective tissue was greatest on the palmar and dorsal sides of the finger flexor tendons where the compressive forces are greatest.

The mechanism of these changes has not been described, but it probably involves occlusion of blood

flow and deprivation of nutrients. The nutrient pathway involves both bulk flow and diffusion.[61] Diffusion requires a concentration gradient between the tendon and surrounding synovium that is maintained by circulation. Occlusion of blood flow caused by compression of the tendon against adjacent surfaces, thickening of tendon sheaths, or increased diffusion distances due to thickened sheaths would all contribute to deprivation of nutrients. The deprivation will be related to the intensity of the exertion, the duration of the exertion, and the frequency of exertion—or forcefulness and repetitiveness.

Compressive and shearing forces on tendons may be transferred to adjacent nerves. For example, it is well known that flexion of the wrist causes the finger flexor tendons to impinge on the median nerve and that the opening and closing of the fist causes the finger flexor tendons to rub on the median nerve. The tendons also may impinge on an adjacent nerve when thickening occurs as a result of tenosynovitis. In either case, the result is nerve involvement.

Some tissues, such as tendons and tendon sheaths, are highly adapted to bearing mechanical loads, while others, such as nerves and blood vessels, are not. The result is that some tissues can endure much more trauma than others; however, both have limits before the recovery becomes insufficient for the response.

The remainder of this article will focus on how forcefulness and repetitiveness contribute to hand and wrist tendinitis and how they can be controlled through job design.

Ergonomic considerations. Regardless of the mechanism of force and repetitiveness, an obvious hypothesis is that the reduction of repetitiveness will reduce the risk of hand and wrist tendinitis.

From a physiologic viewpoint, repetitiveness is best characterized as the number of times that a tendon is stretched or squeezed per unit of time. However, employers and job designers tend to think in terms of work quantities per unit of time. The number of exertions or movements can be determined through an analysis of the work methods and standards.[42, 43] The number of exertions per unit of time can be used to select a job design that minimizes repetitiveness, for placement of restricted workers, or for epidemiologic studies. Some of the methods to reduce repetitiveness are listed in Table II.

Tendon force is related to the force exerted with the hand and to the posture of the hand.[62, 63] In some cases it is possible to estimate the force of exertion from the weight of tools and materials and their friction characteristics. In other tasks, such as grinding or assembling parts, the force cannot be easily estimated, and only gross rankings may be possible. Gross estimates can also be obtained by means of surface electromyography, but specialized equipment and trained technicians are required.[20, 49] Some of the ways that the forcefulness of a manual task can be reduced are listed in Table II.

Table II. General repetitiveness and force controls for reducing ergonomic stresses in manual work

Repetitiveness
Mechanical assists
Process changes
Product changes
Work enlargements
Worker rotations
Rest allowances
Forcefulness
Decrease weight of objects held in hand
Increase friction of objects held in hand
Balance tools
Use air shutoff or external torque bars to control tool torque
Use mechanical assists for holding tools and lifting parts
Slide, do not lift parts
Use roller or powered conveyors to moving parts
Use handles that can be gripped to avoid pinching
Maintain quality control on part fit
Replace or service dull and worn tools
Avoid gloves that are excessively bulky

Although this discussion has focused on repetitiveness and forcefulness, there is ample evidence that mechanical stresses and certain postures are also important factors in hand and wrist tendinitis and should be considered as possible intervention routes.[42, 43]

It is emphasized that, although a highly significant association between repetitiveness, forcefulness, and symptoms of hand and wrist tendinitis has been demonstrated, the effectiveness of preventive job design is still to be demonstrated. For this reason, any attempt to modify the job to prevent tendinitis or similar nerve disorders must include some formal follow-up evaluation of the workers.[64]

Summary

The available data show that hand and wrist tendinitis and related disorders are long-standing causes of worker suffering and lost work in hand intensive industries. Data show a strong association between the signs and symptoms of hand and wrist tendinitis and repetitiveness and forcefulness of manual work. Although these data also suggest that women are at slightly greater risk than their male counterparts, this risk is very small in

comparison with that of forcefulness and repetitiveness. The pathogenesis of chronic tendon disorders is not yet known, but it probably involves both mechanical and physiologic responses of the tendons to mechanical stress. Although there was a highly significant association between repetitiveness, forcefulness, and symptoms of hand and wrist tendinitis, the effectiveness of preventive job design is still to be demonstrated, and it is essential that any attempt to modify the job to prevent tendinitis or similar nerve disorders include some formal follow-up evaluation of the workers.

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