A Clinical Report of the Effect of Mechanical Stress on Functional Results after Fasciectomy for Dupuytren's Contracture

Roslyn B. Evans, OTR/L, CHT

Indian River Hand and Upper Extremity Rehabilitation, Inc. Vero Beach, Florida

Paul C. Dell, MD

Department of Orthopedics University of Florida Gainesville, Florida

Paul Fiolkowski, PhD, ATC

Department of Orthopedics University of Florida Gainesville, Florida

ABSTRACT: Early postoperative treatment after Dupuytren's fasciectomy traditionally has included the application of mechanical stress to digital extension with splints and exercise. This study examines the effect of mechanical stress, which may compromise nutrient delivery to the tissues, on inflammation, flare, hypertrophic scar, digital range of motion (ROM), and therapy visits. The authors compared functional outcomes in operated digits treated postoperatively with tension applied (TA) and no tension applied (NTA), retrospectively from 1983 to 1993 (TA only) and prospectively from 1993 to 1999 (TA and NTA). The charts of 268 patients who underwent Dupuytren's fasciectomy were reviewed and divided into 2 groups (TA and NTA). Each case was analyzed with respect to age, sex, number of digits operated, postoperative management technique, therapy visits, metacarpophalangeal joint, and proximal interphalangeal joint ROM, degree of flare, and scar. There were significant differences in ROM, scar formation, flare, and treatment time in favor of the NTA technique. The results indicate that postoperative management that prevents applied mechanical tension in the early phases of wound healing decreases complications after this surgery and that no digital motion is lost to extension with the NTA technique.

J HAND THER. 2002;15:331-339.

Postoperative complications after release of Dupuytren's contracture include sympathetic flare, hypertrophic scar, joint stiffness, recurrence, and disease extension.^{1–6} This disease has been studied extensively over the past 150 years in terms of anatomy,⁷ cause,^{8,9} pathophysiology,¹⁰ associated ailments,¹¹ genetics,^{12,13} demographics,^{9 14} and surgical technique.^{15–17} Dupuytren's contracture is better understood now, with much new research on biochemical and cellular aspects; however, the true cause of the disease remains unknown,^{18–20} and there are few available data about the response of these cells to various agonists.²¹

Although postoperative complications and outcomes have been studied with regard to surgical technique,²² little has been written about postoperative management as it applies to complications and outcomes other than recommendations for splinting and therapy as an adjunct to surgery^{23–26} and one study concerning continuous passive motion used postoperatively.²⁷

The effect of mechanical stress on Dupuytren's palmar fascia and cellularity,^{21,28,29} occlusion of capillary endothelium,^{30,31} tissue anoxia, and oxygen free radicals^{30,32-37} and growth factor release has received increased attention from investigators.^{38–42} In an attempt to understand the pathogenesis of this condition, studies have defined the role of the myofibroblast in force transmission to the extracellular collagen and matrix in Dupuytren's disease.^{43,44} Research into therapeutic regimens has examined the effect of continuous stress on the collagen fibers of Dupuytren's tissue applied before surgery, with results indicating that stress to these tissues preoperatively can trigger the release of enzymes that weaken collagen.^{45–49}

These studies, which address the biochemical and biomechanical response of these tissues to mechanical agonists, may raise questions regarding some popular rehabilitation techniques that apply tension to vessel, nerve, and incision lines with splinting and exercise techniques that often apply stress beyond the physiologic limits of accommodation in this operated tissue. Tissue nutrition in the early postoperative phase after Dupuytren's fasciectomy has not received enough attention by hand therapists, and mechanical stress applied by the therapist or patient may be a crucial variable with regards to complication rate and outcomes that has been overlooked.

The authors' hypothesis, based on clinical observation and literature review, was that splinting patients with Dupuytren's fasciectomy under tension postop-

Correspondence and reprint requests to Roslyn B. Evans, OTR/L, CHT, Indian River Hand and Upper Extremity Rehabilitation, Inc, Suite E110, 787 37th Street, Vero Beach, FL 32960. E-mail: rose-vans@ gate.net.

TABLE 1. Descriptive Data for Patients in the Study

	No Tension Applied	Tension Applied
No.	165	103
Male	128	76
Female	37	27
Mean age (SD)	69.33 (6.78)	67.15 (8.91)

eratively may contribute to negative alterations in neurovascular function and altered tissue nutrition and facilitate hypertrophy of scar in lines of tension and that stressful exercise technique that contributes to edema or inflammation contributes to localized hypoxia. This theory is supported by basic science studies on the effect of tissue anoxia, free radical release, cellular alteration, and basic wound healing principles. The purposes of this article are to examine the effects of mechanically applied stress on flare and hypertrophic scar with regard to splint position and to raise awareness, on the part of surgeons and therapists, that improperly applied stresses postoperatively may have a negative influence on biochemical and biomechanical events in this tissue.

METHODS AND MATERIALS

Subjects

Data collection for this project was accomplished by a review of 268 patient medical records from 312 available cases of Dupuytren's fasciectomy (Table 1). There were 44 cases excluded for incomplete data or follow-up. The patients were treated in a single hand therapy practice from 1983 through 1999. The patients were operated on by 49 surgeons, with no controls on surgical technique.

The first group (tension applied [TA], n = 103) consisted of 76 men and 27 women with a mean age of 67.15 years (± 8.91) and average number of digits operated of 1.96 (± 0.71). These cases were evaluated retrospectively from 1983 to 1993 and prospectively from 1993 to 1999. The primary author (R.B.E.) treated the cases from 1983 to 1993 with extension splinting immediately postoperatively as standard accepted protocol. Cases included in this group from 1993 to 1999 were those sent to the primary author at 2 weeks or greater postoperatively wearing physician-applied splints. A limited number (n = 13) were seen as follow-up cases after being treated first at other therapy sites.

The second group (no tension applied [NTA], n = 165) consisted of 128 men and 37 women with a mean age of 69.33 years (\pm 6.78) and an average number of digits operated of 1.6 (\pm 0.7). These patients all were studied prospectively from 1993 to 1999 and treated with the no-tension protocol following the clinical

observation by the primary author that after Dupuytren's fasciectomy many patients treated with tension applied developed flare.

All patients treated prospectively and their referring physicians were informed that the NTA protocol, although used by some clinicians, differed from the TA protocol used by many clinicians. Patients were not asked to sign a consent for medical record review, and this was not an issue in a private practice setting. Data collection identified each patient by number and not name. It was thought that the data collected involving dates of referral, time in therapy, range of motion (ROM), and complications was non-sensitive material that would not violate privacy issues.

Treatment Protocols

Patients in the TA cohort were splinted with tension applied to the operated palmar and digital regions with efforts to obtain extension immediately after surgery (from 20° to 0° metacarpophalangeal [MCP] and proximal interphalangeal [PIP] extension) (Figure 1). Extension splints were worn intermittently during the day (with self-exercise sessions prescribed 4 to 6 times per day) and overnight. Exercise efforts were directed toward regaining digital extension immediately postoperatively according to standard postoperative protocol. No attempt was made to apply aggressive tension for the sake of this study, and the technique of applying tension was not used by the primary author after 1993.

NTA cases were splinted with no wound tension to the operated palmar or digital regions by blocking the last 40° to 45° of MCP extension with a static dorsal blocking splint that supports the wrist at 0° extension, MCP joints at 40° to 45° flexion, and PIP joints in neutral position allowing controlled flexion but no digital extension beyond these parameters (Figure 2). The dorsal blocking splint was prescribed for 24-hour wearing time, with the patients advised to work with gentle active flexion exercise within the splint every 2 hours. Careful attention to gentle exercise technique and no repetitive forces prevented wound tension from being applied to these tissues with exercise. At 7 to 10 days postoperatively, the operated digits were fitted with volar digital extension splints to improve PIP extension (to be worn intermittently during the day and at night), and by 2.5 to 3 weeks postoperatively, the blocking splint was discontinued in the daytime and replaced by a static volar MCP and PIP extension splint for nighttime, usually hand based.

Both groups were treated with flexion exercise for each digital joint with composite motion and individual joint blocking exercise. Distal joint motion was encouraged to stretch the often tight oblique retinacular ligaments and to encourage tendon gliding for the flexor digitorum profundus tendon.



FIGURE 1. Volar static extension splints used immediately postoperatively for TA patients. This splint applied extension forces to MCP and PIP joints as a hand-based extension splint (A) or with the wrist included (B).

Data Analysis

Each case was analyzed with respect to age, sex, surgeon, number of digits operated, therapy technique (splint attitude and exercise), days from start of therapy to discharge, number of therapy visits required for rehabilitation, MCP and PIP ROM, degree of flare, and degree of scar. No attempt was made to match the effects of diathesis, osteoarthritis, or surgical technique. All comparisons were made based on the TA versus NTA rehabilitation technique.

All recordings of physical characteristics were made by the principal investigator. Flare was graded using the criteria defined in Table 2. Flare complications for each patient were assigned a number (0, 1, 2)based on symptoms and required treatment. Similarly, scar was graded and was assigned a number (0, 1, 2) by using the criteria defined in Table 3.

For comparison of ROM, individual *t*-tests with a Bonferroni correction were used to compare the ROM at the PIP and MCP joints for each of the 4 fingers. The independent variable was tension, or the lack thereof, with the dependent measure being ROM (in °). A separate 3×2 analysis of variance (ANOVA) was run using tension, age, and number of days between surgery and start of treatment as the independent variables, whereas days to discontinuation of treatment and number of visits were the dependent variables. Duncan's post-hoc test was used to test the individual effects of independent variables, should any differences be indicated by the test results.

Chi-square tests were used to examine relationships between the application of tension and the development of scar and flare. Chi-square tests also were used to examine the association of gender with these developments. Significance was set *a priori* at 0.05.

RESULTS

The results of the ANOVA using age, gender, and tension as independent variables indicated that neither gender nor age showed an effect on the time spent in rehabilitation. Also, there was no interaction between the variables of age, gender, and treatment on the number of visits required or the days until discharge. The only significant difference in these measures was seen when comparing TA and NTA. To achieve similar results, the TA cohort required 20 therapy visits compared with 13 for NTA (p < 0.01) (Figure 3). Similarly, TA patients were in therapy for an average of 67.73 (± 47.21) days, whereas NTA patients were in therapy 36.49 (± 22.83) days from the initial visit until discharge (p < 0.01) (Figure 3). Average time between surgery and therapy for the TA group was 5 days and for the NT group was 2 days.

Final ROM in flexion for the MCP and PIP joints was significantly improved in the NTA group compared with the TA group ROM only at the PIP level in

Grade	Description	Treatment Required
Grade 0	No inflammation beyond normal wound healing	Edema control
Grade 1	Inflammation limited to operated fingers, with redness, stiffness, edema lasting beyond 2–3 weeks	Edema control, high volt, cold, anti-inflammatories
Grade 2	Sympathetic symptoms extending beyond operated digits	TENS, stellate blocks, gabapentin (Neurontin), steroids, stress loading

TABLE 2. Grading System Used to Evaluate Flare

ABBREVIATION: TENS, transcutaneous electrical nerve stimulator.



FIGURE 2. Dorsal static protective splints used immediately postoperatively for NTA patients. This dorsal splint design allows flexion but not MCP joint extension in a controlled range preventing neurovascular and wound tension the first 2.5 weeks postoperatively.

the long, ring, and small fingers; these differences were 4.03°, 3.83°, and 4.04° (p < 0.05) (Figure 4). Similarly the 2 treatments resulted in a significant difference in the extensor deficit at the PIP joint level only. These differences were 5.39°, 6.91°, and 6.32° in the long, ring, and small fingers (p < 0.05) (Figure 5). Analysis of the subjective grading for flare and scar revealed differences between the groups. There was a significant difference between the 2 groups with regard to flare development (p < 0.01) (Table 4) and degree of scar complication (p < 0.01) (Table 5) in favor of the NTA group.

Splinting the MCP joints in flexion for the first 2.5 weeks of wound healing to relieve tension did not result in any loss of ROM in digital extension, but it did result in fewer therapy visits, less time to discharge (less expense for treatment was incurred), and decreased complications of flare and scar. Final ROM was clinically similar for both groups; however, the TA group required more therapy to reach similar measurements obtained by the NTA group.

DISCUSSION

Patients in the NTA group had fewer scar complications, developed less flare response, and required less therapy. The final ROM at time of discharge was statistically significant in favor of the NTA protocol, but for practical purposes (differences of 4° to 9° per digit) was not clinically significant. No motion was lost to extension with this protocol. Clinical experience shows that postoperative splinting among hand surgeons and therapists varies from relaxed extension, aggressive extension, tension relieved, to no splinting.^{10,19,23–26,50,51} Thirty years of clinical experience by the primary investigator have led to the observation that large variations in force application exist among therapists and with patient self-exercise. Many patients tend to overexercise with repetition and stress that creates local inflammation. Therapy "aides," such as exercise putty, hand grippers, and sponge balls, often used postoperatively for these cases elevate pressures at the A1 pulley region⁵² and within the carpal tunnel⁵³⁻⁵⁶ and can contribute to problems of carpal tunnel symptoms and triggering of the unoperated digits. Delaying PIP joint extension splinting by 7 to 10 days does not result in loss of motion gained from surgical release, and similarly delaying MCP joint extension splinting by 2.5 to 3 weeks after MCP joint release does not result in loss of motion.

The NTA technique described in this study may decrease adverse tissue response and complication rate because it may facilitate improved tissue nutrition, decrease inflammation, and decrease incision line tension. The rationale for this theory is supported in studies on tissue anoxia, inflammation, and mechanical stress. The studies validating these concepts are reviewed subsequently.

Mibble 0. Gludnig bystelli Osed to Evaluate Stat				
Grade	Description	Treatment Required		
Grade 0	Soft, pliable, non-painful flat scar	Light massage, longitudinal paper tape at 2 weeks		
Grade 1	Thick, widened scar with no joint limitation, tender, hypersensitive	Silicone gel sheets, ultrasound, splinting, massage		
Grade 2	Hypertrophic, inflexible scar with joint limitation, hyperemic, itching, pain	Silicone gel sheets, ultrasound, iontophoresis, splinting, serial casts, massage		

TABLE 3. Grading System Used to Evaluate Scar

Anoxia and Cellular Activity

Many of the epidemiologic associations of Dupuytren's disease are associated with poor vascularity and implicate a relationship between tissue nutrition and the pathophysiology of Dupuytren's disease.14,37,57 An epidemiologic association of Dupuytren's disease has been noted with regard to age, race, and diabetes, which are situations involving microvessel narrowing.³⁷ Similarly, age, race, diabetes, alcohol consumption, human immunodeficiency virus, infection, cigarette smoking, and trauma are associated with increased free radical production, which also has epidemiologic ties to Dupuytren's disease.9,37 The deleterious effects of poor oxygenation resulting in release of oxygen free radicals in response to altered oxygen economy have been studied^{32,58} and have been associated with the development of Dupuytren's contracture.8,34,37

Capillary occlusion from endothelial fibroblast infiltration in Dupuytren's nodules and cords also contributes to decreased local tissue oxygenation.^{30,31} These microvascular changes may be a common pathway to the development of fibrotic lesions^{8,31,33,37} and may have a relationship to the development of hypertrophic scars.⁵⁹

At a cellular level, the major phenomenon in Dupuytren's contracture is an increase in proliferating fibroblasts.³⁶ Hypoxia is known to stimulate fibroblasts in culture.³³ The myofibroblast, a specialized fibroblast cell, has contractile properties⁶⁰ and is thought to have a major role in the development of Dupuytren's disease.^{40,44} There is speculation that this specialized fibroblast cell could be the agent that regulates the palmar fascia⁶¹ and that myofibroblast activity is increased with wound-edge tension.⁶²

Murrell et al^{8,34} studied the effect of tissue anoxia and oxygen free radicals in Dupuytren's tissue. They noted that the potential for hypoxanthine-xanthine oxidase free radical formation is greater in Dupuytren's tissue than in control tissue and that the production of free radicals may be a factor in the pathogenesis of Dupuytren's disease.³⁴ Oxygen free radicals that may be released from narrowed or occluded microvessels (and from the fibroblasts that infiltrate this tissue) may stimulate increased fibroblast proliferation, contributing to further microvessel ischemia.^{31,36,37}

Externally applied stress to the microvessels of patients with Dupuytren's contracture increases the number of stress fibers in the endothelium of post-capillary venules.^{47,48} Vascular endothelial cells respond to changes in hemodynamic forces, such as fluid shear stress.⁶³

Local hypoxia from capillary occlusion or microvessel narrowing could be increased further as mechanical tension is applied to this vasculature with postoperative extension splinting. The normal elasticity of neurovascular structures may be compromised from chronic MCP and PIP joint flexion contractures, and surgical correction of joint deformity followed by stretch into extension may result in digital artery spasm.⁵

Mechanical Stress, Inflammation, and Cellular Activity

Clinically, we have noted the correlation between prolonged postoperative inflammation and edema

80 70 60 50 40 30 20 10 0 days to D/C number of visits

FIGURE 3. Comparison between NTA and TA of number of therapy visits and days from initial therapy visit to discharge (p < 0.01).



FIGURE 4. Comparison between NTA and TA final ROM with respect to MCP and PIP joint flexion.

from forceful extension splinting, aggressive manual therapy, or repetitious exercise and complications of flare. In the laboratory, this correlation has been supported by several studies. Local edema, such as that produced from too-vigorous therapy, decreases vascularization. It does so by altering hydrostatic capillary pressure, leading to decreased tissue oxygenation necessary for healthy tissue.^{64–66}

Wiseman et al⁶⁷ suggested that there is a dosedependent relationship between local inflammation, the number of macrophages, and the number of fibroblasts that are active in a wound. Macrophagemediated growth factors may provide the initial stimulus for the progression of Dupuytren's disease, and a correlation has been made between macrophage numbers and the presence of myofibroblasts in the palmar fascia of patients with Dupuytren's disease.³¹

The effects of mechanical stress on the palmar fascia and cellularity in Dupuytren's disease has been studied.^{21 28 29} Palmar fascia responds to mechanical stress,²¹ and studies suggest that transforming growth factor-TGF-B combined with mechanical stress can promote differentiation of fibroblasts into myofibroblasts.²⁹ An increase in cellular proliferation and platelet-derived growth factor-PDGf-A expression in cultured cells occurred in response to 12 hours of mechanical stress.²⁸

Mechanical stress may play an important role in myofibroblast differentiation.^{68,69} The myofibroblast is thought to be the cell responsible for contracture in Dupuytren's disease.⁴³ Andrew et al³¹ proposed that a relationship exists between the events of macrophage migration (possibly related to trauma) with growth factor release and local proliferation of fibroblasts, which leads to microvascular occlusion. They theorized that the problem is exacerbated further by the high metabolic demand created locally by the proliferating cells and hypoxia, which further stimulates fibroblast proliferation.³¹ These concepts are supported by the work of Murrel et al³⁵ and studies noted previously concerning tissue anoxia, free radical release, growth factor release, and cellular proliferation.

Inflammation also can be increased by stretch injury to the digital nerves and may be induced by compression, irritation, or stretching of digital



FIGURE 5. Comparison between NTA and TA final extension deficit in MCP and PIP joints.

TABLE 4. Percentages	of Patients	in	Each	Group
Exhibiting Flare				

	No Tension	Tension
No flare	85	48
Mild flare	13	42
Severe flare	2	10

nerves.⁷⁰ After joint release, stretch to the digital nerves that may have lost their normal excursion secondary to joint contracture may lead to neurapraxia. Microstretching, epineural edema, and impaired neural oxygenation may contribute to nerve fiber dysfunction and flare reaction.⁷⁰ Pain syndromes and digital nerve neurapraxia are serious complications of Dupuytren's fasciectomy.^{71–73}

Mechanical Stress and Scar Formation

Hypertrophic painful scars are a postoperative complication after Dupuytren's release and may limit patients' ability to tolerate frictional forces on the palmar surface. Mechanical tension and chronic inflammation have been associated with hypertrophic scarring,⁷⁴ and scarring specifically is greater on the flexor surface of the extremities exposed to high dynamic skin tension.⁷⁵

Wound site tension may reduce the rate of repair, compromise tensile strength, and increase the final width of the scar.⁷⁶ Laboratory studies have shown that mechanical tension increases fibroblast proliferation77; myofibroblast activity has been associated with wound site tension⁶²; and repetitive mechanical stress has been shown to promote collagen synthesis and deposition, resulting in hypertrophic scarring.⁷⁸ In addition to these biochemical activities, it has been suggested that excessive tension at the incision site may cause flap necrosis by jeopardizing local blood supply.79,80 The numerous published results documenting the deleterious effects of tension applied to a wound site seem to indicate a need to rethink current treatment protocols for this condition. Relieving tension is a relatively simple process. Tension on an incision line may be relieved with splints that limit motion and stress and micropore paper tape placed longitudinal with the incision from the second postoperative week.81,82

Limitations of Study

A valid shortcoming in this study is the possibility of a gender bias (Table 1) and the influence of the time of referral to rehabilitation. These factors could have affected the number of visits required to obtain a satisfactory result, treatment time to discharge, and final ROM. The information regarding rate of flare and scar complications should be considered soft data because the instrumentation has been neither TABLE 5. Percentages of Patients in Each Group with Scarring

8		
	No Tension	Tension
No scar	78	22
Mild scar	17	56
Severe scar	5	22

investigated nor determined to be reliable⁸³ (evaluation by the principal investigator based on symptoms and required treatment) and because the collected data are descriptive.⁸⁴ This portion of the study was limited by imaging costs. Objective flare rate may be measured by magnetic resonance imaging, bone scintigraphy, Doppler sonography, and thermography.^{85,86} Scar may be evaluated objectively by B-scan ultrasound to measure intradermal height and width of scar, laser-Doppler flowmetry to evaluate metabolic activity, and color measurements with a chromometer to establish scar maturity.⁸¹ Such methods are not readily available to most clinicians, however.

CONCLUSION

Although this study is limited by methodologic flaws^{83,84} relating to the soft methods of evaluating flare and scar, the data support the conclusion that patients treated with no tension and therapeutic exercise with low load and repetition during the early phase of wound healing have better outcomes than patients treated with tension applied. There is no motion lost to extension of the MCP or PIP joints or composite joint flexion with the NTA rehabilitation technique. The large number of cases presented support the clinical observation, and results of chart review show that the NTA technique, which has as its focus tissue nutrition instead of joint motion during the early wound healing phases, yields decreased complications of flare and scar. The significant differences in time required in therapy with reduced expense lend support to the NTA technique. The results also support early referral to therapy.

This is a clinical study with practical application. The authors found no prior attempt to characterize objectively the measures that have been recorded with respect to the effect of therapist-applied mechanical tension applied to these cases postoperatively in the literature. The results of this clinical study suggest that applied mechanical tension may be an important variable in complication rates after this surgery.

REFERENCES

- 1. Zemel NP. Dupuytren's contracture in women. Hand Clin. 1991;7:707-13.
- 2. Zemel NP, Balcomb TV, Stark HH, et al. Dupuytren's disease in women: evaluation of long-term results after operation. J Hand Surg Am. 1987;12:1012–6.

- Tubiana R, Fahrer M, McCullough CJ. Recurrence and other complications in surgery of Dupuytren's contracture. Clin Plast Surg. 1981;8:45–50.
- 4. Hueston JT. Regression of Dupuytren's contracture. J Hand Surg Br. 1992;17:453–7.
- 5. Boyer MI, Gelberman RH. Complications of the operative treatment of Dupuytren's disease. Hand Clin. 1999;15:161-6.
- Watson H, Fong D. Dystrophy, recurrence, and salvage proceedures in Dupuytren's contracture. Hand Clin. 7:745–56.
- 7. McGrouther DA. Anatomy, descriptive and surgical. J Hand Surg Br. 1991;16:240–2.
- Murrell GA, Hueston JT. Aetiology of Dupuytren's contracture. Aust N Z J Surg. 1990;60:247–52.
- 9. Ross DC. Epidemiology of Dupuytren's disease. Hand Clin. 1999;15:53-62.
- Lubahn JD. Open-palm technique and soft-tissue coverage in Dupuytren's disease. Hand Clin. 1999;15:127–36.
- 11. Sok Y, Johnson G. Moneim M. Etiology of Dupuytren's disease. Hand Clin. 1999;15:43–52.
- 12. Burge P. Genetics of Dupuytren's disease. Hand Clin. 1999;15:63-71.
- Whaley DC, Elliot D. Dupuytren's disease: a legacy of the north? J Hand Surg Br. 1993;18:363–7.
- 14. Wilbrand S, Ekbom A, Gerdin B. The sex ratio and rate of reoperation for Dupuytren's contracture in men and women. J Hand Surg Br. 1999;24:456–9.
- Jabaley ME. Surgical treatment of Dupuytren's disease. Hand Clin. 1999;15:109–26.
- Hall PN, Fitzgerald A, Sterne GD, Logan AM. Skin replacement in Dupuytren's disease. J Hand Surg Br. 1997;22:193–7.
- 17. Chick LR, Lister GD. Surgical alternatives in Dupuytren's contracture. Hand Clin. 1991;7:715–22.
- Fitzgerald AM, Kirkpatrick JJ, Naylor IL. Dupuytren's disease: the way forward? J Hand Surg Br. 1999;24:395–9.
- 19. McFarlane R. Dupuytren's disease. J Hand Ther. 1997;10:8-13.
- Yi S, Johnson G, Moneim MS. Etiology of Dupuytren's disease. Hand Clin. 1999;15:43–51.
- 21. Gupta R, Allen F, Tan V, Bozentka D, Bora F, Osterman AL. The effect of shear stress on fibroblasts derived from Dupuytren's tissue and normal palmar fascia. J Hand Surg Am. 1998;23:945–50.
- Shaw D, Wise D, Holms W. Dupuytren's disease treated by palmar fasciectomy and an open palm technique. J Hand Surg Br. 1996;21:484–5.
- 23. Mackin EJ. Prevention of complications in hand therapy. Hand Clin. 1986;2:429-47.
- 24. Mullins PA. Postsurgical rehabilitation of Dupuytren's disease. Hand Clin. 1999;15:167–74.
- Prosser R, Conolly WB. Complications following surgical treatment for Dupuytren's contracture. J Hand Ther. 1996;9:344–8.
- 26. Rives K, Gelberman R, Smith B, Carney K. Severe contractures of the proximal interphalangeal joint in Dupuytren's disease: results of a prospective trial of operative correction and dynamic extension splinting. J Hand Surg Am. 1992;17:1153–9.
- Sampson SP, Badalamente MA, Hurst LC, et al. The use of a passive motion machine in the postoperative rehabilitation of Dupuytren's disease. J Hand Surg Am. 1992;17:333–8.
- Alman BA, Greel DA, Ruby LK, Goldberg MJ, Wolfe HJ. Regulation of proliferation and platelet-derived growth factor expression in palmar fibromatosis (Dupuytren contracture) by mechanical strain. J Orthop Res. 1996;14:722–8.
- 29. Tomasek JJ, Halliday NL, Updike DL, et al. Gelatinase A activation is regulated by the organization of the polymerized actin cytoskeleton. J Biol Chem. 1997;272:7482–7.
- Kischer CW, Speer DP. Microvascular changes in Dupuytren's contracture. J Hand Surg Am. 1984;9:58–62.
- 31. Andrew JG, Andrew SM, Ash A, Turner B. An investigation into the role of inflammatory cells in Dupuytren's disease. J Hand Surg Br. 1991;16:267–71.
- Horton AA, Fairhurst S. Lipid peroxidation and mechanisms of toxicity. Crit Rev Toxicol. 1987;18:27–79.

- 33. Hunt TK, Banda MJ, Silver IA. Cell interactions in post-traumatic fibrosis. Ciba Found Symp. 1985;114:127–49.
- 34. Murrell GA, Francis MJ, Bromley L. Free radicals and Dupuytren's contracture. BMJ. 1987;295:1373-5.
- 35. Murrell GA, Francis MJ, Howlett CR. Dupuytren's contracture: fine structure in relation to aetiology. J Bone Joint Surg Br. 1989;71:367–73.
- Murrell GA, Francis MJ, Bromley L. Modulation of fibroblast proliferation by oxygen free radicals. Biochem J. 1990;265:659–65.
- Murrell GA. An insight into Dupuytren's contracture. Ann R Coll Surg Engl. 1992;74:156–61.
- Badalamente MA, Hurst LC. The biochemistry of Dupuytren's disease. Hand Clin. 1999;15:35–42.
- Badalamente MA, Hurst LC, Grandia SK, Sampson SP. Platelet-derived growth factor in Dupuytren's disease. J Hand Surg Am. 1992;17:317–23.
- 40. Gabbiani G, Majno G. Dupuytren's contracture: fibroblast contraction? An ultrastructural study. Am J Pathol. 1972;66:131–46.
- Kloen P, Jennings CL, Gebhardt MC, Springfield DS, Mankin H. Transforming growth factor-beta: possible roles in Dupuytren's contracture. J Hand Surg Am. 1995;20:101–8.
- 42. Terek RM, Jiranek WA, Goldberg MJ, Wolfe HJ, Alman BA. The expression of platelet-derived growth-factor gene in Dupuytren contracture. J Bone Joint Surg Am. 1995;77:1–9.
- Tomasek JJ, Schultz RJ, Haaksma CJ. Extracellular matrixcytoskeletal connections at the surface of the specialized contractile fibroblast (myofibroblast) in Dupuytren disease. J Bone Joint Surg Am. 1987;69:1400–7.
- 44. Tomasek JJ, Schultz RJ, Episalla CW, Newman SA. The cytoskeleton and extracellular matrix of the Dupuytren's disease "myofibroblast": an immunofluorescence study of a non-muscle cell type. J Hand Surg Am. 1986;11:365–71.
- 45. Brandes G, Reale E, Messina A. Microfilament system in the microvascular endothelium of the palmar fascia affected by mechanical stress applied from outside. Virchows Arch. 1996;429:165–72.
- Afoke A, Meagher PJ, Starley I, McGrouther DA, Bailey AJ, Brown RA. Biomechanical characterization of tissues in Dupuytren's disease. J Hand Surg Br. 1998;23:291–6.
- Citron N, Messina JC. The use of skeletal traction in the treatment of severe primary Dupuytren's disease. J Bone Joint Surg Br. 1998;80:126–9.
- Messina A, Messina J. The continuous elongation treatment by the TEC device for severe Dupuytren's contracture of the fingers. Plast Reconstr Surg. 1993;92:84–90.
- Bailey AJ, Tarlton JF, Van der Stappen J, Sims TJ, Messina A. The continuous elongation technique for severe Dupuytren's disease: a biochemical mechanism. J Hand Surg Br. 1994;19:522–7.
- Abbott K, Denney J, Burke FD, McGrouther DA. A review of attitudes to splintage in Dupuytren's contracture. J Hand Surg Br. 1987;12:326–8.
- Jain AS, Mitchell C, Carus DA. A simple inexpensive postoperative management regime following surgery for Dupuytren's contracture. J Hand Surg Br. 1988;13:259–61.
- 52. Azar C, Fleegler E, Culver J. Dynamic anatomy of the flexor pulley system of the fingers and thumb. Second International Meeting for International Federation of Societies for Surgery of the Hand; 1983; Boston, Mass.
- Cobb TK, An KN, Cooney WP, Berger RA. Lumbrical muscle incursion into the carpal tunnel during finger flexion. J Hand Surg Br. 1994;19:434–8.
- Cobb TK, An KN, Cooney WP. Effect of lumbrical muscle incursion within the carpal tunnel on carpal tunnel pressure: a cadaveric study. J Hand Surg Am. 1995;20:186–92.
- 55. Seradge H, Jia YC, Owens W. In vivo measurement of carpal tunnel pressure in the functioning hand. J Hand Surg Am. 1995;20:855–9.
- Siegel DB, Kuzma G, Eakins D. Anatomic investigation of the role of the lumbrical muscles in carpal tunnel syndrome. J Hand Surg Am. 1995;20:860–3.

- 57. Murrell GA. The role of the fibroblast in Dupuytren's contracture. Hand Clin. 1991;7:669–81.
- White MJ, Heckler FR. Oxygen free radicals and wound healing. Clin Plast Surg. 1990;17:473–84.
- 59. Kischer CW, Thies AC, Chvapil M. Perivascular myofibroblasts and microvascular occlusion in hypertrophic scars and keloids. Hum Pathol. 1982;13:819–24.
- 60. Tomasek JJ, Vaughan MB, Haaksma CJ. Cellular structure and biology of Dupuytren's disease. Hand Clin. 1999;15:21–34.
- Fitzgerald AM, Kirkpatrick JJ, Foo IT, Naylor IL. A picropolychrome staining technique applied to Dupuytren's tissue. J Hand Surg Br. 1995;20:519–24.
- Rudolph R, Berg JVd, Ehrlich P. Wound contraction and scar contracture. In: Cohen I, Diegelmann R, Linblad W (eds). Wound Healing: Biochemical and Clinical Aspects. Philadelphia, Penn.: W.B. Saunders; 1992:1296–314.
- Ando J, Komatsuda T, Kamiya A. Cytoplasmic calcium response to fluid shear stress in cultured vascular endothelial cells. In Vitro Cell Dev Biol. 1988;24:871–7.
- 64. Hunt TK, Knighton DR, Thakral KK, Goodson WH, Andrews WS. Studies on inflammation and wound healing: angiogenesis and collagen synthesis stimulated in vivo by resident and activated wound macrophages. Surgery 1984;96:48–54.
- LaVan FB, Hunt TK. Oxygen and wound healing. Clin Plast Surg. 1990;17:463–72.
- Knighton DR, Silver IA, Hunt TK. Regulation of wound-healing angiogenesis—effect of oxygen gradients and inspired oxygen concentration. Surgery 1981;90:262–70.
- Wiseman D, Pharm S, Rovee D, Alvarez OM. Wound dressings: design and use. In: Cohen I, Diegelmann R, Linblad W (eds). Wound Healing: Biochemical and Clinical Aspects. Philadelphia, Penn.: W.B. Saunders; 1992:562–80.
- 68. Grinnell F. Fibroblasts, myofibroblasts, and wound contraction. J Cell Biol. 1994;124:401-4.
- 69. Halliday NL, Tomasek JJ. Mechanical properties of the extracellular matrix influence fibronectin fibril assembly in vitro. Exp Cell Res. 1995;217:109–17.
- Lundborg G. Nerve compression injuries: the role of microvascular dysfunction. In: Hunter J, Schneider L, Mackin E (eds). Tendon and Nerve Surgery in the Hand. St. Louis, Mo.: Mosby, 1997:137–44.
- 71. Rodrigo JJ, Niebauer JJ, Brown RL, Doyle JR. Treatment of

Dupuytren's contracture: long-term results after fasciotomy and fascial excision. J Bone Joint Surg Am. 1976;58:380-7.

- 72. Tubiana R, Thomine JM, Brown S. Complications in surgery of Dupuytren's contracture. Plast Reconstr Surg. 1967;39:603–12.
- Bower M, Nelson M, Gazzard BG. Dupuytren's contractures in patients infected with HIV. BMJ. 1990;300:164–5.
- Su CW, Alizadeh K, Boddie A, Lee RC. The problem scar. Clin Plast Surg. 1998;25:451–65.
- Murray J, Pinnel S. Keloids and excessive dermal scarring. In: Cohen IK, Diegelmann RF, Lindblad WJ (eds). Wound Healing: Biochemical and Clinical Aspects. Philadelphia, Penn.: W.B. Saunders, 1992:500–9.
- Harris DR. Healing of the surgical wound: II. factors influencing repair and regeneration. J Am Acad Dermatol. 1979;1:208–15.
- 77. Curtis AS, Seehar GM. The control of cell division by tension or diffusion. Nature. 1978;274:52-3.
- Chvapil M, Koopmann CF Jr. Scar formation: physiology and pathological states. Otolaryngol Clin North Am. 1984;17:265–72.
- 79. Adamson J, Fleury AF. Incisions in the hand and wrist. In: Green DP (ed). Operative Hand Surgery. 2nd ed. vol 3. London: Churchill Livingstone, 1988:1785.
- Falanga V. Occlusive wound dressings: why, when, which? Arch Dermatol. 1988;124:872–7.
- Niessen FB, Spauwen PH, Robinson PH, Fidler V, Kon M. The use of silicone occlusive sheeting (Sil-K) and silicone occlusive gel (Epiderm) in the prevention of hypertrophic scar formation. Plast Reconstr Surg. 1998;102:1962–72.
- 82. Reiffel RS. Prevention of hypertrophic scars by long-term paper tape application. Plast Reconstr Surg. 1995;96:1715–8.
- Fess EE. Guidelines for evaluating assessment instruments. J Hand Ther. 1995;8:144–8.
- Szabo RM. Statistical analysis as related to hand surgery. J Hand Surg Am. 1997;22:376–85.
- Graif M, Schweitzer ME, Marks B, Matteucci T, Mandel S. Synovial effusion in reflex sympathetic dystrophy: an additional sign for diagnosis and staging. Skeletal Radiol. 1998;27:262–5.
- Holder L. Bone scintigraphy. In: Gilula L, Yin Y (eds). Imaging of the Wrist and Hand. Philadelphia, Penn.: W.B. Saunders; 1996: 319–50.