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Dupuytren's contraction : its etiology and treatment

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DUPUYTREN'S CONTRACTION,
ITS ETIOLOGY AND TREATMENT

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Senior Thesis

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PREFACE

I wish to extend my sincere thanks and appreciation to my advisor, Dr. C. W. M. Poynter, for his generous expenditure of time and most helpful and constructive interest, advice and suggestions in the composition of this thesis.

I also wish to thank Miss Alice Meredith for her assistance in typing this work.

Patrick Leonard
March 8, 1943.

INTRODUCTION

The purpose of this paper is to explore and evaluate the literature on Dupuytren's contraction with emphasis on its etiology and treatment. No claim of originality is made for such an approach--one of the fundamental facts applicable to all branches of medicine is that the conception of the etiology of a disease at any one period influences the mode of treatment at that period. This, in a measure, has been true in Dupuytren's contraction; however, the ancient medical rule of treating symptomatically when the etiology is obscure also has been generously applied here.

Dupuytren's contraction, briefly defined, is a permanent acquired flexion deformity of the hand. It occurs chiefly in men of middle age and past. Reports on incidence are much in variance, but it is probable that this condition is more common than generally considered.

Dupuytren was preceded in recognition of this disease by several other men, but it took his reputation and excellent description of the disease, which he called "Permanent Retraction of the Fingers", to command the attention of the other great surgeons of the time. It is thought by some that Felix Platter recognized this condition in 1641. Henry Clive in 1808 described the pro-

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cess to his students; Sir Astley Cooper about 1822 had done the same and had developed a surgical procedure for its treatment. Boyer also was aware of this particular kind of contraction before Dupuytren described it and called the disease crispatura tendinum.

The exact time or person who first called it Dupuytren's contraction was not discovered in the literature except that Adams, 1879, states: "Since Dupuytren made this important contribution to our knowledge of this affection in the year 1832, it has sometimes been spoken of as Dupuytren's finger-contraction, a title as useful, as it is also a just compliment to the great surgeon, distinguishing it from all other forms of finger-contraction".

Events in the history of this condition are a reflection of medical thought of the period. Before aseptic surgery, the surgical treatment was designed to agree with the surgical concepts of that time. Here is where the subcutaneous surgery of Adams, etc., had its basis, as you will see when you read the treatment section. The same was true of the concept of its etiology and pathology. The general trends in medical thought exerted their influence and were applied to this specific disease,

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(foci of infection theory). History, as one can see, is best taken up in the sections on Etiology, Pathology and Treatment, where progress can be best correlated and followed; that is the way it will be dealt with in this thesis.

NORMAL ANATOMY

The knowledge concerning the anatomy of the hand, has like the knowledge of other parts of the body, been laboriously accumulated through the years, and errors have been perpetrated in many instances rather than going to the trouble of a systematic investigation. It has been incomplete as well as often inaccurate up until about the last half-century. The contribution of Kanaval in 1921 has become a classic in the field of the anatomy of the hand. His work has been repeated, corroborated and modified by other men; Grodinsky and Holyoke of our University of Nebraska are among these workers. In this discussion of anatomy, no attempt is made to review completely the voluminous literature on the subject. The opinions of present day authorities on the fascia of the hand as it applies to Dupuytren's contracture is presented here so that the pathology in this condition can be more readily understood.

The palmar aponeurosis, the main structure thought to be involved in Dupuytren's contraction, is described in Cunningham's Anatomy as follows:

"The palmar aponeurosis is a thick triangular membrane, the apex of which joins the distal edge of the transverse carpal ligament, and more superficially receives the insertion of the tendon of the palmaris longus

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muscle. The fascia separates below into four slips, one for each finger, connected by transverse fibers, and forming beneath the webs of the fingers the superficial transverse metacarpal ligament. Beyond this each slip separates into two parts, to be connected to the sides of the metacarpophalangeal joints and the first phalanx of the medial four digits. The lateral borders of the triangular central portion of the palmar aponeurosis are continuous with thin layers of deep fascia which cover and envelop the muscles of the thenar and hypothenar eminences."

Harper, 1935, from his work in the dissecting room concludes there is also a digital insertion of the aponeurosis to the thumb in most cases, (3).

The palmar aponeurosis is only a part of the fascia enclosing the hand, and its relationship must be understood before discussing this problem. For descriptive purposes, the fascia of the hand may be divided into four parts: (1), the volar interosseus fascia; (2), the superficial palmar fascia, (palmar aponeurosis); (3), the dorsal fascia; and (4), the digital fascia. Together with various septa, fibers, and ligaments which unite these parts, these fascias serve to help give shape and compactness to the hand. Some of these

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mentioned have been given particular descriptive names, (7).

The palmar aponeurosis originates proximally from the palmaris longus muscle, or in the absence of the latter, from the antibrachial fascia; it being triangular in shape, glistening and shiny, with prominent longitudinal fibers which become divided into distinct bands. The number and arrangement of these pretendinous bands vary considerably, five being the usual number, (3). Fibers extend into the deep surface of the skin along the whole length of the bands and are especially well developed over the region of the metacarpophalangeal joints, (4). This is of particular interest as dimpling of the skin here is often the first symptom of the disease. A thin fascial extension covers the interdigital spaces where the digital nerves and vessels become more superficial. Fibers from the under side of the pretendinous bands merge with the sheaths of the flexor tendons and are traceable for a variable distance along the superficial layer of the sheath as far distad as the middle phalanx, (5). The pretendinous bands continue to the fingers as digital slips, one for each finger. In the fingers each slip divides into a median and two lateral portions; the two lateral processes surround the

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tendon sheaths and are inserted into the dorsal surface of the first and second phalanges; the medial portion passes along the anterior surface of the finger and is inserted into the skin as far down as the pulp of the last phalanx, (6).

The volar interosseous fascia, the digital fascia, and the septa which unite them are important surgically. The volar interosseous fascia covers the cup of the palm left after the flexor tendons, palmar arches and digital nerves are removed. It is identical with the pronator quadratus fascia and covers the volar interossei and attaches to the metacarpal and carpal bones. It forms the transverse metacarpal ligament and distad blends with the sheath of the flexor tendons. Three longitudinal septa unite the superficial palmar and the volar interosseous fascia and separate the hand into four palmar compartments, (7, 8, 9).

The digital nerves and arteries and the lumbrical muscles lie in the wedge or V-shaped spaces left by the diverging flexor tendons. Each lumbrical muscle is covered with a thin fascial sheath continuous with the fascial covering of the flexor tendons which they accompany higher in the palm; therefore, there is a potential space extending from the fascial spaces of the palm along

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the lumbrical muscles to the dorsum of the fingers, where they insert into the extensor tendon on the dorsal part of the proximal phalanges, (8).

The flexor tendons diverge as they continue to the fingers. The fascia which forms the lateral walls of their tunnels increases in thickness distally and attaches to the transverse metacarpal ligament, the lateral aspects of the proximal phalanges and to the capsule of the metacarpophalangeal joints. This fascia also sends oblique fibers which run superficially to the flexor tendon sheaths and attaches to the opposite side of the proximal phalanges, and thus helps to form the deep layer of the digital fascia. Through contraction of these fibers the digital nerves and blood vessels may be displaced from one side of the finger to the other, thus causing fingers not primarily involved in the contraction to be flexed and partly rotated from their normal position, (7, 10).

The digital fascia is of two layers; the superficial which is a continuation of the palmar aponeurosis, and the deep, which is a continuation of the fascia about the tendons. The digital arteries, veins and nerves lie between these layers. The dorsal fascia of the fingers, not of importance in this condition, blends with the

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superficial layer of the digital fascia, (9, 7).

"The flexion deformity of the fingers which develops as the contraction of the palmar fascia progresses is due chiefly to the continuity of the superficial layer of the digital fascia with the longitudinal fibers of the palmar aponeurosis. The not infrequent deviation of the digital nerves and vessels from one side of the finger to the other results from the fact that they lie in a fibrous tunnel whose contracting walls are continually drawn proximally toward the site of the primary involvement". (7).

The normal histology of the fascia and skin of the palm is essential to be reviewed in order that a comparison can be made with the pathological findings recorded in another part of this paper. In the normal hands of adult specimens, the palmar fascia consists of bundles of dense poorly cellular connective tissue, arranged longitudinally in some areas and transversely in others. The collagenous fibers, with a few elastic fibers, are compacted into parallel bundles and have elongated and flattened nuclei with a few vascular channels. Horwitz reports these features were demonstrable in all the twenty-seven specimens of apparently normal hands of cadavers of various ages he examined. In the later

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decades of life the fascial layer becomes thicker and more dense and the bundles of collagenous fibers more compact. There is little difference, especially in the older people, between the palmar aponeurosis and tendon tissue. The progressive thickening and condensation of the normal palmar fascia with the advancing age may be of significance in the causation of Dupuytren's contracture, since this condition is most frequent in the later decades of life, (5). The relation to the skin of the palm to the palmar aponeurosis is important. Histologically, the skin of the palm is much like that elsewhere on the body, except that it contains no hair follicles or sebaceous glands. The papillae of the corium are abundant here; the deeper part of the corium consists of interlacing bundles of connective tissue fibers which form a rather loose network. Below this, strands or fascicles of connective tissue run from the skin to the palmar aponeurosis. Between these strands is adipose tissue forming the fatty pad of the hand, (11).

The essential points of the anatomy of the palmar aponeurosis, both gross and microscopic, are that it is formed of dense connective tissue made up of collagenous fibers in parallel bundles and poorly cellular. It is inserted into the anterior and lateral aspects of the

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head of the proximal phalanges and has continuity with the superficial layer of the digital fascia covering the fingers. From the aponeurosis there are septa extending from its under surface which divide the hand into four spaces. Superficially there are numerous fibrous fascicules which insert into the skin of the palm.

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Signs and Symptoms

The first sign of this disease is usually a small nodule or slight induration of the skin on the palmar surface of the hand at the base of the third or fourth finger. Later, a minute dimpling or funnel-like depression occurs in the palm just distal to the indurated noduel. These signs usually occur before the contraction of the fingers begins, and usually indicate which fingers will be affected first, (12, 34).

There are commonly no subjective symptoms noted; an occasional patient will complain of slight pain or neuralgia, sometimes a tingling sensation in the palm of the hand and extending down into the fingers, (13). There may be an occasional cramping sensation and occasional numbness. Rarely, there is intermittent tenderness, (12). The majority of the patients develop the condition with no pain or irritation at all, (7).

The usual course of development is slow and insidious; months and years even may pass before the patient becomes aware of a contraction. Blackfield, however, states a crippling contracture may develop within a few weeks, (14). After the initial symptoms mentioned above, there is a developing band of contracted fascia extending from the concavity of the palm as far

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as the first or second interphalangeal joint of the fingers, (usually the ring and little finger, see table III in Etiology).

After a time, the length of which varies, the process may spread to other fingers, and as the disease progresses other contracting bands progressively develop, one for each finger involved. The bands, raised and taut, stand out like the strings of a bow and are closely adherent to the skin. The skin of the palm becomes more and more indurated and creased, and other nodules and funnel-like depressions appear. The subcutaneous fat gradually thins out and disappears, (34). The contraction may progress so that the tips of the involved fingers press tightly into the palm, sometimes to the extent that they become macerated and become a hygienic problem, (15). The fingers sometimes are rotated at their metacarpophalangeal joints because of an unequal degree of contraction of the fascia on either side of the fingers, (7).

There may be individual variations in the form and location of the contracture. These are easily explainable if one recalls the relationship of the insertions of the palmar aponeurosis to the digital fascia,

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(see chapter on Anatomy). The main flexion deformity is in the metacarpophalangeal joint, as described by Dupuytren and agreed on almost universally. Hutchinson, 1917, thought that the proximal interphalangeal joint was the more often involved and for that reason raised the question as to whether it was the prolongations of the palmar aponeurosis that caused the contraction. He must not have been familiar with Adam's work, 1879, who discussed and explained the variances of deformity very adequately, (38, 91, 34).

The disease may be limited to the palmar portions only, or to the digital prolongations of the fascia, or a combination of both. The most common involvement is in both the digital and palmar parts combined, and next most common in the fascia of the palm alone, (6, 7, 13, 56). *

Differential Diagnosis

The diagnosis of this disease from other affections of the hand is usually not difficult, if a little time is taken to examine the hand thoroughly.

*NOTE: Because of the wide variation in deformities, Meyerding, of the Mayo Clinic, has developed a system of classification ranging from "0" to "4" in increasing order of involvement. Although this idea has not been taken up by other recent writers, his classification, I believe, would serve excellently for statistical purposes. (The reader is referred to Reference #15 for further information.)

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Contractions from tendon lesions, whatever be their nature, do not exhibit the characteristic skin changes seen in fascia contraction, and in these cases the fingers cannot usually be flexed or extended, while in Dupuytren's contraction motion of the finger is free within the limits of the contraction. In scar tissue contractions, there is the history of injury and the absence of the typical Dupuytren's contracture picture. Deformities associated with various diseases of the joints may cause some difficulty, but other symptoms of the joint disease should be of aid. However, one must remember that the arthritides are common in the same age group. I have seen one case of this combination occurring bilaterally in a man of 87 at the Clarkson Hospital.

The diagnosis of the contractions limited to the digital fascia is more difficult, as in the absence of skin changes the deformity may be very similar to certain arthritic changes, (13). Roentgenograms are helpful here. Some cases diagnosed as hammer finger may have been Dupuytren's contracture involving only the digital fascia. History, therefore, is of first importance in differentiating congenital deformities. This is of medico-legal importance also because Dupuytren's contracture has been proved a compensable injury in courts several times.

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In spastic conditions the metacarpophalangeal joints are extended; this is in direct contrast to Dupuytren's contraction, in which the characteristic deformity is flexion at this joint, (6).

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Gross

The gross pathological changes occurring in Dupuytren's contraction so closely follow the clinical course that to repeat them here would be only repetition, (recorded in signs and symptoms under section on diagnosis). However, it is well to review the mechanism by which these signs and symptoms are brought about. Essentially, the pathological processes are a thickening and shortening of the palmar aponeurosis and its distal insertions. Since the time of Dupuytren, these facts have been recognized almost universally. However, Geyrand, 1833, (quoted by Reid in 1836 and Boyer in 1847), thought the deformity was due to a new growth of a fibrous chord extending from the palmar aponeurosis to be inserted into the sheathes of the flexor tendons, (16, 17). This opinion, I believe, can be recorded as an erroneous conclusion made from a correct observation. Preceding Dupuytren, Henry Clive, 1808, Sir Astley Cooper and Boyer, all recorded this disease. Cooper ascribed the pathology to "contraction of the flexor tendon and thecae", and Boyer called the contracted cords *crispature tendinum*, (17, 32, 33, 51). It is probable, then, that Dupuytren was the first man to accurately interpret the real pathology of this disease; Boyer later agreed with him

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in 1847, (17). However, the question was one of debate for a considerable period: Adams in 1879 went to great lengths to prove that the contracture was not due to a change in the flexor tendons, (34). Strange as it may seem, there has been reported opinion to the contrary in the literature as late as 1927, when Rey recommended section of the flexor tendons for this condition, (7).

It is appropriate to quote here an account of the dissection Dupuytren performed on a hand with this contraction: "The arm was given to him, and he made a careful dissection. The skin having been removed from the whole extent of the palm of the hand, and the palmar face of the fingers, the fold or the puckering entirely disappeared; it was therefore very evident that the arrangement which presented during the disease did not depend on this cause, but was communicated to it; but how and by what? The dissection was continued; the professor discovered the palm, or aponeurosis, extended, retracted, diminished in length, its inferior part divided into cords which pass themselves on the sides of the affected finger. In extending the finger he observed that the aponeurosis underwent a kind of tension, of crispation--this was a ray of light; so he considered the aponeurosis was something in the effects of the dis-

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ease. He cut the prolongations on the sides of the fingers, immediately the contraction ceased, the fingers returned to a state of demi-flexion, and by slight force, to complete extension. The tendons were natural--the sheaths were not open--the articulations, ligaments, synovial membranes, and bones were in their normal, or natural state. It is, therefore, natural to conclude, that retraction is caused by an exaggerated tension of the palmar aponeurosis, and that this is caused by a contusion of the aponeurosis, by the strong and prolonged action of hard bodies in the palm of the hand." (32).

Kanaval, Koch, and Mason in 1929, emphasize that the fascial involvement is not limited to the palmar aponeurosis. They state that thickening and contraction of the interfascial septa which unite the superficial palmar fascia and the volar interosseous fascia, sometimes the thickening of the dorsal fascia is found, (7). This dorsal fascia involvement has been mentioned by White in 1897, and more recently by Weber in 1938, (18, 19). These were described as subcuticular, pea-sized, fibrous nodules on the extensor surfaces over the proximal interphalangeal joints.

The involvement of fascia other than the aponeurosis is a very important point surgically: The in-

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volvement of a finger is not always of the same degree on both sides; thus the finger may be deviated to one side, and through the fasciculi transveri, a contraction of the finger next to the first may be involved also. The fascia often becomes so thickened and dense, that the digital arteries and nerves are compressed and displaced from their normal anatomical position. Cases have been reported in which the blood vessel and nerves have been displaced the width of a finger, (7). The understanding of this is important when treated surgically, either by total excision or fasciotomy, because these structures are likely to be damaged unless extreme care is taken, (10).

Microscopic

The early writers were interested mainly with the gross pathology, and only sketchily, if at all, described the microscopic. Richer, 1797, found the diseased fascia identical in structure with that of normal palmar fascia. The fibrous parts were more numerous, thick and dense in the contracting part. He saw no signs of inflammation and the lesions were similar to those of chronic arthritis, (20). Chevrot, 1882, described the subcuticular fascia of the palm as dense, fibrous and

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sclerotic. He found no connective-tissue cells or elastic fibers. The normal fat layer was absent and the skin and fascia showed no line of demarcation. The layers of the skin showed changes similar to those of disuse, (21).

Anderson described the condition in 1897 as an inflammatory hyperplasia or neoplastic growth which began in the skin and subcutaneous tissues of the palm and by a process of nuclear proliferation spread along the course of the blood vessels, replacing the adipose tissue and, secondarily, involving the palmar fascia, (35).

Nichols, 1899, reported three cases in two different articles. He observed the thickened skin attached to the fascia and the absence of the fatpad of the palm. He noted a variation of vascularity in different parts of the same specimen. One of the cases presented the tissues of the palm as one dense fibrous mass. From these three cases he concluded the process was a connective tissue hypertrophy which passed through definite stages; first it was vascular and highly cellular, then it reached a stationary stage and became more and more dense and fibrous, (13, 22).

Janssen in 1902 reported a close resemblance

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microscopically between the cellular fibrous tissue and fibrosarcoma. He concluded, however, that the process was neither inflammatory or neoplastic, but was a hypertrophic process, (23).

McWilliams, 1904, reported that the essential pathologic condition responsible for the contraction of the palmar fascia is a chronic hyperplastic inflammation with subsequent formation of scar tissue in the fascia and the adjacent connective and fatty subcutaneous tissues. He correlated clinical symptoms with microscopic findings. He believed the inflammation did not involve these tissues in their entirety at first, but only certain portions of them at any one time, thus giving the stage by stage clinical picture. He concluded the process was a continuous one with various stages of connective tissue growth in different parts of the band at the same time. This would explain some of the conflicting microscopic findings of other writers. He further states the joints may be finally ankylosed from loss of movement, (43).

Hutchinson, 1917, harks back to the opinion of Geyraud, 1833, who thought the contracting bands in the fingers were "new growths" and not hypertrophy of existing structures. As to microscopic arrangement he states:

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".....the overgrowth of fascia, (and sometimes the skin), becomes almost a caricature of the normal arrangement". (38).

Ledderhose in 1920, in a report on palmar fasciculitis described the process as a chronic proliferative process without inflammation, (37).

Kanaval, Koch and Mason, 1929, add nothing new to the picture except to describe the fibrotic changes in the skin. They found the corneal layers of the epidermis thickened, and the deep epidermal layers flattened. The papillae of corium were replaced by fibrous tissue and the fat gone from about the blood vessels, (7).

Davis and Finesilver, 1932, in investigating their cases found that recent contractures had a less mature type of connective tissue than long standing contractures. This is to say the tissue was more vascular and cellular. They noticed a profusion of cells along the blood vessels, and a greater abundance in the adventitia, (6). This is practically the same view as Nichols' in 1899 and others.

The recent report of Meyerding, Black and Broders, 1941, gives much more attention than the early writers to the pathological changes in the skin and subcutaneous fat. They examined thoroughly tissue removed

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from fifty-seven patients. They could not definitely decide where the initial process began: "It is probable that the disease begins in the interstitial connective tissue and then usually spreads to involve all the tissues of the palm down to the deep structures, tendon sheaths, nerves, and blood vessels." They noticed the same skin changes as Kanaval, Koch and Mason, and reasoned that this accounted for the early loss of skin elasticity. The line of demarcation between the skin and subcutaneous tissue was very indefinite and sweat glands rare. The connective tissue bands which separated the lobules of fat were increased in number and size at the expense of the fat. The process seemed to be one of increased number of capillaries with lymphocytes surrounding them, followed by increasing fibrosis and decreasing fat, (18). Fatty tissue normally separates the nerves and blood vessels from the aponeurosis, but as the process progresses, it is easily seen how the nerves and vessels become imbedded in the contracted fascia. This is an important point to consider in the surgical treatment, as previously emphasized. The description of the palmar fascia itself was as described by other authors. These authors assumed that the degree of cellularity was an index to proliferative activity. They found no relation-

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ship between connective tissue maturity and degree of contraction, the latter being more dependent upon the duration of the disease. However, the maturity of the connective tissue examined did show that the tendency to recurrences was greater in those cases which showed a less mature, (more cellular), type of tissue. They concluded that the pathological picture was best explained on the basis of a chronic inflammatory process, (18). Horwitz, 1942, who has published an article recently, disagrees with Meyerding, et al., in their view that the tendency to recurrence was greater in the more cellular specimens, nor does he believe the degree of contraction related to its duration. He could find no evidence to support the contention that this process was a chronic inflammatory one. He noted a close histologic resemblance here to that of keloids, fascial desmoid and fibromas, and concluded this was a benign fibroplasia, (5). The tendency for keloids to recur is well known; Dr. Poynter described a case he once operated. He also found the tissue not unlike a keloid.

If one views the opinions of the various men on the morbid histology here, it is seen that there is in the pathology as in the etiology of this disease, much variance in interpretation. The two latest articles re-

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ported are in almost direct opposition to each other. It is my opinion that correct interpretation will be determined when the etiology is definitely proved. It is significant, I believe, when such men as Kavanal, Koch and Mason describe what they see, but do not append any particular name to the process.

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This affection, like so many other diseases has been attributed to a great variety of factors and conditions. A good proportion of the entire literature on the subject has been devoted to the discussion of cause. The trend of thought through the years has been a parallel to that concerning diabetes; treatment has changed as the conception of etiology has been changed. The last word on Dupuytren's contraction has not yet been written, and the controversy of etiology among contemporary writers on this subject is as lively as it ever was. For the sake of avoiding undue confusion in recording, the findings of various authors will be arranged into four groups.

Hereditary or Constitutional Predisposition.

The number who believe in this factor is large. This theory has been considered, evidently, since before Dupuytren described the disease in 1831, because he states: "It has been made successively to depend upon a rheumatismal, a gouty affection, on a morbidic cause induced by metastasis" (32). I was not able to find in the literature whose work he referred to. Adams in 1879, Anderson in 1897, Keen 1881, have held that this is a manifestation of a constitutional condition, such as

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gout or rheumatism. Keen said such a history could always be found and reported an incidence of 48% in his series. Adams and Anderson are in accord with Keen. The former agrees that the disease always depends on a constitutional rather than on a local cause. The latter thinks the earlier workers exaggerated the importance of irritation and trauma. He believed it was less common in the laboring occupations, (See Table I), than in the less rigorous callings, (33, 34, 35). Gill, (1938), believes it is akin to gout and rheumatism and associated with aging, but he believes a broader view should be taken: "It, (Dupuytren's Contracture), probably is not an entity itself; it is probably due to one or more constitutional conditions". (36).

Ledderhose, (1920), associates arthritis deformans and Dupuytren's contraction, and believes the contraction is part of the symptomatology of the arthritis, usually being present in some degree. He states that both conditions often begin in youth and never progress to any great extent, (37).

Hutchinson, (1917), reported three cases in which there were family histories of the condition. In one case, the father and grandfather of the patient were both affected; in another patient, an uncle and a brother

TABLE I
Laborers and Nonlaborers

<u>Author</u>	<u>Cases</u>	<u>Laborers</u>	<u>Nonlaborers</u>
Black	131	63 - 48%	68 - 52%
Meyerding	273	123 - 45%	150 - 55%
Horwitz	35	18 - 51%	17 - 45%
Davis & Finesilver	40	20 - 50%	20 - 50%
Kanaval, Koch, Mason	29	10 - 35%	19 - 65%
Keen	123	49 - 39%	74 - 61%
Byford	<u>38</u>	<u>24 - 63%</u>	<u>14 - 37%</u>
	669	307 - 45.8%	362 - 54.2%

TABLE II
Incidence in Male and Female

<u>Author</u>	<u>Cases</u>	<u>Male</u>	<u>Female</u>
Meyerding	273	241 - 88%	32 - 12%
Davis & Finesilver	40	35 - 80%	5 - 11%
Kanaval, Koch, Mason	29	27 - 93%	2 - 7%
Byford	38	35 - 91%	3 - 9%
Black	240	221 - 92%	19 - 8%
Anderson	39	25 - 64%	14 - 36%
Keen	227	187 - 82%	40 - 18%
Horwitz	<u>35</u>	<u>33 - 94%</u>	<u>2 - 6%</u>
	921	804 - 87.7%	117 - 12.3%

These tables and the succeeding tables were compiled by the author in order that the reader could better compare the findings of the various authors and more easily obtain a comprehensive view of the disease statistically, especially the etiology.

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were affected; and in the third patient, the mother was also afflicted, (38). Apert, (1925), reports observations of a family with contracture in four generations. It affected only the male members, (See Table II), and appeared earlier with each generation, (39). Lowry, 1923, reported a decreasing age with each generation also, and cited a family with five contractures in three generations, (68).

Sprogus, (1926), discovered seventeen of fifty-three members of a family affected in three generations. He agrees with Krogius, (1920), that this is a developmental disease due to disorders in growth in the superficial palmar muscles, (40, 41). Manson, (1931), reports a family showing the atavistic tendency in this disease; the mother, father and three sons had contractions, but the daughters did not. The third generation, who are now adults, show no signs of contracture, (42).

McWilliams in 1904, found only two cases in twenty-four with a family history of the disease, (43). Davis and Finesilver report only five cases in a series of forty who gave a family history. One of the two men who gave a family history of the condition was a doctor, and both his grandfather and father, who also were doctors, had Dupuytren's contraction. Three of the cases were

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women; one had a grandmother and great-grandmother afflicted; one had a brother and father; and the third had a mother with Dupuytren's contraction. The fifth patient, a male, had a father and a younger sister with a similar contraction, (6). It is interesting to note that the family having several physicians in it gave the best family history; perhaps this is because a physician would be more on the alert in observing such a condition, especially if it was of slight degree. Couch, (1938), reported two identical twins with identical contractures; the accompanying pictures of the hands of the two patients were so near alike as to appear as duplications. No information was given as to the time interval between the onset in the two patients nor any family history, (44).

Powers in 1934, expressed his view that this disease is a trophic disturbance caused by factors which arise within the body. He cited a multitude of diseases which, in some way or another, caused nervous irritation and thence this contraction of the fascia in the hand. He does not believe that Dupuytren's contraction is an isolated condition or a clinical entity, but usually an effect of past or present visceral disease producing irritation of the sympathetic system. He regards the hereditary factor on the basis of an inherited sympathetic un-

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balance. According to him this disease should be classified with scleroderma, hypertrophic osteoarthropathy, multiple chondromata, fibrositis penis plastica, et cetera, and proposes a term "dystrophilia" to cover all of these, (45). The literature of late has had a number of foreign articles associating Dupuytren's contracture with some of these diseases, especially scleroderma and Peyronie's disease, and a contraction of the plantar fascia. Of those written in English, Kaplan, 1942, quoting Volvasek's work, (46), reports an incidence of 4.6% of Dupuytren's contraction in a series of one hundre ninety-eight cases of Peyronie's disease. Horwitz, 1942, noticed the striking pathological similarity to Peyronie's disease, plantar contraction and Dupuytren's contraction: "There is a striking histologic resemblance, (of Dupuytren's contraction), to other localized fibroplasias like both keloid and fascia desmoid, are prone to recur despite extensive surgical excision".(5).

Hereditary or Constitutional
Predisposition plus Trauma.

McWilliams, 1904, thought that a constitutional predisposition to the disease must be present and that, in a certain group of patients, local insults acted not

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only as exciting causes, but also as continuing influences; as an example, as people grow older they begin to use a cane, or after being in an occupation such as shoemaking--which requires the use of an awl over a long period of years--this oft repeated trauma would be of importance in etiology in persons already constitutionally predisposed, (43).

Shubert, 1923, observed that the disease most frequently involves the fourth and fifth fingers, (See Table III), the distribution of the ulnar nerve, and stated that the disease occurs in every kind of condition in which the ulnar nerve is involved, such as tabes, neurities, syringomyelia, trauma to the nerve and others. Because the contraction does not develop every time the nerve is involved indicates the predisposition factor, which is the constitutional connective tissue weakness, must also be present. This idea is not new; Abbe, 1888 described its clinical course. According to his theory, there is first traumatism to the palm, often forgotten, which produces a spinal "impression" of the irritation, then occurs a reflex influence to the affected part producing nutritional disturbances of the tissue, hyperplasia of connective tissue of the fascia with contraction. In some cases a reflex also is set up which causes the

TABLE III

Fingers Affected in Several Series

<u>Author</u>	<u>Cases</u>	<u>Thumb</u>	<u>Index</u>	<u>Middle</u>	<u>Ring</u>	<u>Little</u>
Anderson	39	4	3	22	39	28
Byford	38	4	1	10	35	18
Kanavel, Koch, Mason)	29	4	3	9	31	27
Davis and Finesilver)	40	3	6	14	40	43
Keen	214	11	24	73	199	165
Scholle	54	1	6	10	42	30
Horwitz	50 (hands)	1	1	7	50	50

TABLE IV

Age of Onset in Various Series of Cases

<u>Author</u>	<u>Under 30</u>	<u>30-39</u>	<u>40-49</u>	<u>50-59</u>	<u>60-69</u>	<u>70-79</u>
Davis and Finesilver)	14.0%	25.5%	34.0%	17.5%	9.0%	---
Horwitz	---	10.0%	33.0%	41.0%	16.0%	---
Scholle	15.0%	16.5%	22.0%	28.0%	18.5%	---
Kanavel, Koch) and Mason)	14.0%	20.0%	37.0%	17.0%	12.0%	---
Anderson	2.5%	---	(---51.0%--)	(---46.5%--)		
Keen	27.7%	20.0%	(-----47.7%-----)			
Nichols	2.2%	16.0%	13.3%	35.5%	26.4%	6.6%
Meyerding	-----Average age 54 years-----					

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same thing to happen in the other hand and perhaps in other parts of the body, (Peyronie's disease), (48). Shubert states that Dupuytren's contraction is seen infrequently in mental workers, (47). This is not born out by other authors; Meyerding found 55% of his 273 patients were non-laborers, (15), (See Table I). Grieg reported one case of a congenital bilateral contraction; from his case description, the deformity was very similar to that of Dupuytren's contracture. Grieg concluded that it was the result of a predisposition factor and probably some aggravated force or process in utero, (49). In my opinion, he has no grounds for such conclusions.

Weber, 1938, and Telecky, 1939, are convinced that trauma is a factor but constitutional predisposition and heredity are of the predominate importance. The latter approached the problem as a heredity-environment study, and reasons that everyone has a certain amount of the heredity factor. Those that have only a slight predisposition who develop the contracture must, therefore, be subjected to a great amount of trauma or repeated trauma over a long period of time. He explains the patients with this contracture in non-workers who have had no trauma, as having such an overwhelming abundance of heredity factor that they develop the contracture with-

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out any traumatization. He reports a 1% incidence of this disease among office workers, (19,3150). It seems to me that Telecky has done a good deal of theorizing here, although his ideas are somewhat consistent with McWilliams, 1904, and Weber, 1938.

Weber used the constitutional factor to explain the cases where onset was at an early age, (See Table IV). It is significant to note that he, (Weber), considers advancing age a constitutional factor in itself. In the treatment section of this these the use of thyroid extract will be considered; perhaps this idea expressed by Weber and the observed fact of lower temperatures and lower metabolism in the aged all fit together. However, as far as I know, this combination of facts has not been reported on in the literature of Dupuytren's contracture. Weber also noticed the frequency in which the plantar fascia was also involved in this disease. In spite of the evidence he got together on the heredity factor and constitutional predisposition, he could not alienate himself from the idea that trauma was an important influence, (19).

Davis and Finesilver, 1932, have not yet made up their minds and dispose of etiology as follows:

"There is no single constitutional disease with which it

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is exclusively associated or of which it is a manifestation. In the majority of the cases it does not appear to be specifically caused by trauma or local irritation, though the factor, as well as local or constitutional pathologic-conditions, seems at times to have some exciting or contributing influence." (6).

Trauma to the Palm

That Dupuytren's contraction was due to trauma to the palm is the first opinion that I could discover in the literature as regards to etiology. Sir Astley Cooper, in 1822, expressed this view: "The fingers are sometimes contractedby a chronic inflammation of the thecae and aponeurosis of the palm of the hand, from excessive motion of the hand in the use of the hammer, the oar, plowing, etc." (51). Dupuytren was of the same opinion and his two classic cases which he cited in his original lecture are familiar; "the wine-merchant and the coachman, whose cases we will report, had the habit, one to pierce large barrels with a piercer--the other to exert incessantly his whip on the backs of his jaded horses". (32).

The group of men who have believed in trauma as the only exciting cause is very small. The litera-

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ture records the swing away from this conception of etiology. From the period in which Dupuytren lived until about the 1870's very little was written on this condition. Then, at about this time there was a revival of interest. Ferguson, 1875, begins his discussion of etiology as if it were traumatic and ends up saying he thinks it is a constitutional disease. Paget, 1875, does the same thing, (52, 53). Adams, 1879, and Keen in 1882, who each wrote a monograph on the subject, are definitely opposed to this theory. Nichols, 1899, gives a good summary regarding trauma, which is also the opinion of present day writers: "On the whole, so far as the histories go, the facts observed in this series of cases are decidedly adverse to the supposition that the disease is essentially due to local traumatic causes. There are other considerations opposed to the theory of the traumatic origin and in favor of the contrary view of the idiopathic origin of the disease. The favorite age of onset is after middle life, (See Table IV), years after the period of active labor begins; the left hand is as frequently affected as the more used right, and the ring and little fingers much more frequently than the radial half of the hand, which bears the brunt of labor equally, if not to a greater degree, (Tables III

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and V), " (13). As in all theories, after they are abandoned by most, there are some who still cling to them; Girdwood in 1916, reported a case he attributed to trauma, (54).

Miscellaneous Causes

There is yet another group of theories on etiology that must be considered here for completeness. For want of a better term, let this group be called Miscellaneous Causes, and here will be considered the more prominent theories not dealt with earlier in the paper. One of the most unusual theories is that of a group of French investigators who believe that Dupuytren's contraction is a symptom of lead poisoning. They obtained histories of working with lead in 68% of their cases, (6). This work has not been corroborated by any other workers as far as known. Wainwright, 1926, thought that this disease was a symptom of thyroid deficiency, and Tubby, 1923, intimated that it was a local manifestation of some endocrine disturbance, (55). Powers, 1934, whose theories have been recorded elsewhere reported an improvement in several cases with the use of parathyroid extract; evidently he was thinking along the lines of endocrine disturbance, but did not go into the

TABLE V

Hand Affected in Several Series of Cases

<u>Author</u>	<u>Cases</u>	<u>Right</u>	<u>Left</u>	<u>Bilateral</u>
Anderson	39	10 -- 25.5%	5 - 13%	24 - 61.5%
Black	240	89 - 37%	47 - 15%	104 - 48%
Byford	38	9 - 24%	4 - 11%	25 - 65%
Kanavel, Koch) and Mason)	29	4 - 14%	8 - 28%	17 - 56%
Keen	184	58 - 31.5%	23 - 12.5%	103 - 53%
Scholle	54	28 - 52%	8 - 15%	18 - 33%
Davis and) Finesilver)	40	8 - 20%	6 - 15%	26 - 65%
Meyerding	273	69 - 25%	29 - 11%	175 - 64%
Horwitz	<u>35</u>	<u>12 - 34.5%</u>	<u>4 - 11.5%</u>	<u>19 - 54%</u>
	732	287 - 30.8%	134 - 14.3%	511 - 54.9%

TABLE VI

Incidence Dupuytren's Contracture

<u>Author</u>	<u>Cases Examined</u>	<u>Av. Age Group</u>	<u>Percentage</u>
Nichols - 1899	1000	53 yrs.	4%
Noble Smith- 1884	700	Elderly	10%
Anderson - 1897	2600 800	Middle Age Under 16	1.7% 0.0%
Black - 1915	270 men 168 women	----- -----	2.1% 1.8%
Byford - 1921	1000 men 106 women	----- -----	3.4% 2.8%
Kanaval) Koch) Mason)	- 1929 83800 Hosp. Adm.	----- (Est.)	12%
Leonard - 1938-42) Univ. Hosp. Adm.)	15200 (4)	48-73 yrs.	.0003%

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idea in discussion, (45). It is my opinion that little weight should be placed on information gained or conclusions drawn from such empirical procedures.

Tubby 1923, Byford 1921, and Ely 1926, believe in a foci of infection theory. Byford came to this conclusion by observing that 60% to 84% of his cases with Dupuytren's contraction had an associated rheumatism, and most of them also had infected tonsils or teeth. Ely came to his conclusions in the same way, and in his lectures to his students advised the removal of all foci of infection before any surgery was attempted. Tubby states this is the only theory that explains the occurrence in both laborers and non-laborers, (56, 57, 55).

In considering the focal infection as an etiology, one must remember that most old people have bad teeth and in most cases have not entirely perfect health. I believe that, if the diseases often referred to as resulting from a foci of infection occur with Dupuytren's contraction, it is only by coincidence. Anderson, 1897, proposed a theory of local infection by which micro-organisms gained access to the subcutaneous tissues by accidental lesions. This theory is not logical because pathological findings do not bear it out nor does it explain bilateral lesions, or the usual age of onset, (35).

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As one would expect, the etiology has been explained on an anatomical basis. Noble Smith, 1884, thought that the palmaris longus muscle which keeps the palmar aponeurosis tense normally, was to blame for the contraction. He theorized that the constant movement of the muscle caused it to be irritated and thus contract more. This in turn would make the palmar aponeurosis more tense and likely to be irritated. In other words, it was a vicious circle starting with the palmaris muscle. He thought that an early severing of the palmaris tendon would arrest the condition. The results of this operation were not reported, (58). This theory does not explain the contractures in people with a congenital absence of the palmaris longus muscle.

After examining the findings of the various men who have studied patients with this disease, one begins to form an opinion. Dupuytren's contraction has been reported occurring with disease processes in other parts of the body; it has been most frequently associated, according to the literature, with diseases having a similar micropathological picture--contraction of the palmar aponeurosis, fibrositis of the penis, and so forth. It often has been reported occurring in various members of the same family through several generations. These are

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observations difficult to explain other than that they are due to some factor arising within the patient himself. Local trauma as an etiological factor is untenable, in my opinion, because this disease, according to statistics I have acquired, does not occur in people accustomed to using their hands roughly as often as in people who suffer little trauma to their hands. It occurs usually in a characteristic age group; it begins first in the lateral part of the hand which is the least used, and occurs rarely in the thumb, which is the most used digit. As to the combination of the two and the miscellaneous causes, they are, in my opinion, groping in the dark.

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There has been a great variety in the forms of treatment used in this condition. Down through the years almost every type of therapy physicians could devise-- ointments, injection of connective tissue ferments, physiotherapy, internal drugs, mechanical apparatus, surgery, and even hypnotism. For purposes of organization, treatment will be considered under two headings: non-operative and operative therapy.

Non-operative Treatment.

It is now well recognized that the treatment for Dupuytren's contraction is essentially surgical, but it is interesting to mention the more prominent nonsurgical methods that have been used.

Dupuytren himself tried all sorts of non-operative treatment--vaporized fumigations, resolvent ointments and mechanical traction--to no avail, and in the present day men are still seeking a non-operative cure.

Powers, 1934, reported several cases much improved by taking parathyroid by mouth, (45). Thyroid extract was used with excellent results by Wainwright in four cases. The dosage was one-half grain by mouth at first and gradually increased, changing the dose to suit the individual patient. The patient's hands be-

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came flexible and usable, but there was still increased density of the fascia present, (24).

Humanol was used by Stahnke with reported excellent results. The preparation was made from lipomas and omental fat obtained at operations. This substance was sterilized and injected into and around the diseased aponeurosis, the idea being that the human fat would soften it. The injections were very painful and had to be done under anesthesia. A puncture with a heavy needle was made on either side of the web of the fingers next to the flexor tendon sheath. The needle was inserted under the skin until it reached the transverse fold of the palm, and one-sixth of an ounce of warm humanol was injected on either side. Small amounts were injected into the region of the proximal and medial phalanges if there were atrophic conditions in that region, and the whole hand became swollen. Physio-therapy was instituted two days later. Two to three weeks later a second injection was made. Two injections sometimes caused a marked improvement, and sometimes five or six injections were necessary. No complications or failures were reported, except that the results were not permanent and had to be repeated at variable intervals; one case went six years before requiring further treatment, (25).

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Radiation therapy, with the use of both X-ray and radium, have been reported by Blackfield with good results in most cases, (14). Doctor L. B. Morrison of Boston gave a two-thirds erythema dose over the hand using an aluminum filter and repeated the treatment every two or three months for three treatments. He has had moderate, but significant, success in most cases. There is a danger, as Davis and Finesilver warn, that surgical treatment may be needed later, and the roentgen treatment interferes with the healing process. It should, then, be used only by expert X-ray therapists, (6). A combination of X-ray and surgery was mentioned, but not discussed, by one author, (14). Beatty used X-ray and reported improvement in seven of ten cases; however, only six of these case histories sounded like true Dupuytren's contraction, and only three of these showed improvement, (26).

Operative Methods

I. Subcutaneous Division of the Fascial Bands. (Subcutaneous Fasciotomy), is the method Dupuytren used and is described in his original paper thus: "The operation was performed on June 12, (1831),: The hand being firmly held, he commenced by making a trans-

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verse incision, ten lines in length, opposite the metacarpophalangean articulation of the annular finger; the bistoury divided the skin, and the palmar aponeurosis, with a crackling noise, sensible to the ear. The incision being performed, the ring-finger straightened itself, and was as easily extended as in the natural state. Wishing to avoid the infliction of pain, M. Dupuytren prolonged the section of the aponeurosis, by gliding the bistoury, transversely and deeply, under the skin, on the cubital side of the hand, to accomplish the disengagement of the little finger, but this was in vain. He was obliged to dilate the incision of the aponeurosis, and made another transverse incision opposite the articulation of the first and second phalanges of the little finger, and thus detached its extremity from the palm of the hand, but the rest of the finger remained permanently fixed towards this part. Another incision was therefore necessary opposite the corresponding metacarpo-phalangean articulation, which, when accomplished, was incomplete. Finally, a third incision was practised across, opposite the middle of the first phalanx, and then the little finger was extended with the greatest facility, and announced that this last division affected the aponeurotic digitation. Very little blood was lost by these incisions.

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Dry charpie was applied, and the ring and little fingers extended, and then fixed by a machine, placed over the back of the hand." (32).

Adams, 1879, who was one of the outstanding authorities of his time, and the greatest of all proponents of the subcutaneous operation, advised multiple subcutaneous division of the palmar fascia. In his procedure he used a small tenotomy knife, inserting it through a small opening under the skin and cutting from above downward. The finger was immediately extended and bandaged to a splint, which was not removed until the fourth day. Another splint was then worn night and day for three weeks, and after that at night for a month longer. He believed that this method was applicable in the most severe cases. Sir Astley Cooper, Fergusson, Hawkins, and other early nineteenth century surgeons also used this method, (34).

Keen, 1907, did not believe this operation of Adams' was adequate, stating it was no longer a rational procedure since the advent of aseptic surgery, (63).

A. A. Davies, 1932, believes this a practical operation for the common man because it gives relief and does not subject the patient to major surgery, even though recurrences are common and complete cure not always

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possible, (62). Roth, 1919, also recommended this treatment, (61).

This operation was improved upon and changed somewhat, and the form used today is one in which the multiple sections of the fascia is made through a wider exposure than Adams advocated, (7).

II. Complete Excision of the Palmar Fascia with Closure of the Skin, is now held by the best surgical opinion to be the method of choice. This method was first reported in 1887 by Kocher, and various adaptations are now commonly used. The principle behind it is good: the palmar aponeurosis is diseased, therefore it should be removed. This method is simply dissecting the aponeurosis from the underlying structures through an appropriate incision, and excising it. The anesthesia used is local median and ulnar nerve block or a general anesthesia. The skin is then closed with sutures. The procedure has been refined with time, and the succeeding surgeons have added their touches; such as devising various skin incisions to reach the digital prolongations, splinting, passive motion and massage, the use of Esmarch bandage to gain a clear field, etc., (64).

Hutchinson, 1917, was another surgeon who advocated the open operation, with excision of the palmar

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fascia. His method was original and ingenious. He realized, too, that many hands were still functionless because the fingers could not be extended and concluded that this was probably due to the fact that the glenoid ligament in front of the first interphalangeal joint, as well as the lateral ligaments, had become shortened and incapable of extension because of lack of motion in the joints. He insisted that the only way to overcome this was to excise the head of the first phalanx. His method was: -1- To excise the bands of palmar fascia, including the prolongations over the first phalanx, and then suture the wound on the palm. -2- On the dorsal surface he made a semilunar incision over the first interphalangeal joint, divided the extensor tendon, and removed the head of the first phalanx. -3- The extensor tendon was then slightly shortened and sutured with fine Kangaroo tendon. The finger was now somewhat shorter than normal, but straight and without tension. -4- A dorsal splint was worn, and gentle, active passive movement started within a few days. He was emphatic that no joint should be allowed to stiffen, and vehemently denounced long splinting with a challenge to his colleagues to wear a splint they recommended and decide for themselves what effect it would have on their surgical ability, (38).

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Davis and Finesilver recommend a modification of Hutchinson's operation: If the joint surfaces are intact, they advise loosening the glenoid and lateral ligaments from the head of the proximal phalanx and allowing them to slide forward as the finger is extended; they do not remove the head. They believe the extensor tendon should not be shortened because it will contract of its own accord anyway, the danger of interfering with the tendon's gliding ability or the formation of adhesions is less, (6). I believe this is a very common-sense modification.

Abbott, 1929, devised another ingenious reconstruction operation. He removed the aponeurosis and its ramifications, and then replaced it with a piece of fascia lata from the thigh; he reported perfect results. He emphasized that the following points be observed: use as fine a piece of fascia as possible; make it larger than defect so as to allow for shrinkage; merely tack in place so as not to strangulate any part of it by too tight a suture; make no tension; maintain complete hemostasis; complete rest of the hand for three to six months, (39).

Gill, 1919, excises the contracted palmar fascia

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by open operation and adds a free fat transplant from the thigh to the hollow of the hand before closing the skin flaps. This helps prevent the skin from becoming bound down to the deep structures, gives a more normal anatomic relationship and helps to control incipient bleeding and oozing after the wound is closed. With the patient under general anesthesia and without a tourniquet, a transverse incision is made along the distal palmar crease, and through this incision alone the entire palmar fascia is removed. If contracted fascia is present on the palmar aspect of the proximal phalanges, it may be excised through transverse incisions along the crease at the base of each finger involved. A small free fat transplant from the thigh is then inserted smoothly beneath the palmar skin without sutures, and the incision is closed with a sparse number of interrupted catgut sutures, (27).

Byford, 1921, stated that the treatment for Dupuytren's contracture was purely surgical, and the open operation with dissection of the palmar fascia from the skin and underlying structures was his usual method. He followed this by use of splints for one month constantly, then intermittently for another month, and then at night for six to eight months. He also made the follow-

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ing important observation: The operator should always make sure contraction is stationary. To prevent a recurrence, a thorough general examination should also be made, and all sources of infection removed, (see his views on etiology). Repeated examination of the operated hand for signs of recurrence should be made. If the process is stationary for six months, the chances for recurrence are rare. Another author, Ely, emphasized this point, too, and began treatment of his patients by clearing up sources of infection, such as diseased teeth and tonsils, before attempting surgery, (56, 57).

Meyerding believes in hopelessly involved cases such as seen in recurrences, or with concomitant arthritis, etc., amputation is considered the procedure of choice, (28).

III. Excision of Skin and Palmar Fascia with Skin Grafting or flap shifting to fill the defect is the procedure recommended for best results by Lexor of Germany. This radical procedure is undoubtedly necessary in cases where the skin is hopelessly fibrotic, or in contractures of long duration where the skin has retracted so that it is no longer big enough to cover the palm when the fingers are extended. However, the consensus of present day opinion is to excise the skin only when it is

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hopelessly involved, and to use grafts only when the exposed area cannot be covered with healthy skin and closed without tension, (6, 65, 10).

Kanaval, Koch and Mason summarize very well modern surgical opinion: ". the essential factors in treatment are as complete an excision of the palmar fascia as can be accomplished through the operative incision most suitable for the case in question, the excision of hopelessly affected skin, and the primary closure of the wound without undue tension. In some cases it may be necessary to use a free full thickness graft of skin to replace the excised covering tissue". (7).

When an operative procedure is undertaken, a thorough surgical preparation of the hand is of utmost importance, (the methods used vary with the surgeons), and every effort should be made to maintain asepsis until healing is complete. Meyerding of the Mayo Clinic, for example, uses the following preoperative preparation: A thorough scrubbing of the hands with soap and water and an alcohol dressing applied the night before. At the time of operation the hand is rubbed with benzine to remove the oil, dried with ether, and then painted with a tincture of merthiolate or iodine diluted half with

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alcohol. He has no infections, (65).

Anesthesia is usually novocaine, ($\frac{1}{2}\%$), locally infiltrated or an ulnar and median nerve block. Some surgeons prefer a general anesthesia, because of the danger of lowering the tissue vitality in an area where the blood supply may not be good to start with, (6).

A bloodless field is obtained by the use of a sphygmomanometer cuff. The arm is first elevated to drain off blood, then the cuff is applied. At intervals the pressure is released and bleeders are clamped and tied. This is important, as complete hemostasis is necessary for best results, (7, 10, 14, 15, 29, 30).

Davis and Finesilver take exception to this and do not use a tourniquet because of the possible damage from interfered circulation, especially in the older age group and diabetic patients, (6).

The choice of incision is variable. Incisions should never cross flexion creases, if possible, or lie in the mid line of the finger, (29); and should be chosen to fit the particular case. Koch recommends an L-shaped incision which is suitable in most cases; one arm extends along the distal flexion crease of the hand and the other runs parallel to the fifth metacarpal bone. If it is necessary to carry the dissection into the fingers,

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anterolateral incisions are made along the fingers, (10). The skin over the palm is dissected carefully from the underlying aponeurosis, and every effort is made not to traumatize the skin unnecessarily. The skin is then reflected and held with a blunt retractor. At the proximal end of the aponeurosis the palmaris longus is dissected and separated, and the aponeurosis is grasped with a small hemostat and lifted away from the palm, as the fasciulae which bind it to the deep structures are widely excised. As the dissection is carried into the regions at the bases of the fingers the technique must be even more careful. Due to the contraction hypertrophy of the connective tissue of the palm, the digital arteries and nerves may be directly incorporated into the fibrotic mass. Each artery and nerve must be carefully located and dissected free before the connective tissue is excised. Mason has shown that these structures may be displaced as much as a finger-breadth from their usual anatomical position. If the pathological process extends into the fingers, the dissection is carried on so that every bit possible of the diseased tissue is removed. This may call for accessory excisions along the anterolateral aspect of the finger, as mentioned previously. All skin of questionable vitality is excised and the

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incision closed without tension with a fine needle and horsehair, silk or fine catgut sutures. If the skin margins cannot be approximated, a full-thickness skin graft is used or a tubed flap graft is made. Meyerding has brought the skin around from a disarticulated functionless finger to cover a denuded area in the palm, (65). Gill believes is never necessary, (15). Recently Skinner has suggested the use of tunnel grafts inserted directly under the involved the skin. Among the advantages he mentions for this procedure is that they give the surgeons fewer headaches. No antiseptic solution is put into the wound because of irritation to the tissues. If washing is necessary, warm saline is used, (65).

Strictest aseptic technique must be practised throughout the entire procedure and during the post-operative period until incisions are healed. Even mild infection in an extensive wound of the palm is disastrous. Meyerding dresses the hand in a posterior aluminum splint so as to hold the fingers in extension. The splint should be well padded to protect the skin over joints from excessive pressure. If there is difficulty in obtaining full extension of the fingers, he allows them to remain flexed slightly and straighten them gradually. This is done by placing an adhesive band about the tip of the

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finger and pulling it toward the splint. The splint is extended upward to about mid forearm and is molded to fit the arm. The gauze over the wound is covered with sea sponges so as to exert gentle pressure and prevent oozing, (6). Dressings are not changed for about a week. If the gauze at this time is dry and adherent, it is allowed to remain in position; the fingers are inspected for pressure or circulatory symptoms and the splint re-applied. The duration of splinting varies with the deformity; no motion is allowed until the skin incision is entirely healed. The splint is then worn at night until free motion is obtained. One of the most important factors in recurrence is splinting for an insufficient length of time, (65).

The postoperative care is very important as far as end results are concerned. The postoperative treatment commonly used by Meyerding is as follows: The stitches are removed in ten days but otherwise the wound is untouched and the fibrotic scabs allowed to fall off by themselves. For the next week radiant heat is applied for thirty a day to the palmar surface followed by passive movement of the fingers for five minutes. The movement is urged but not to the degree that drainage appears because this invites infection.

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When the skin incision is well healed, the hand and forearm are treated in a whirlpool bath. This bath is an oval tub containing whirling, aerated water at a temperature of 110° F. It produces hyperemia of the superficial tissues and exerts a general massage effect which relaxes the musculature and has a sedative effect on the peripheral nerves. This is continued for thirty minutes daily. At this stage active assistive exercises are instituted--the patient makes an active effort to move the affected fingers, and is assisted in completion of the movements for about five minutes daily. The active phase of this exercise is gradually increased and the passive decreased until full motion is obtained. The continuous splint is now discontinued unless there is a tendency toward flexion. In most cases, the splint is worn at night for about another month and then completely discarded. By this time danger to the dissected nerves by friction is past and the patient is instructed in gentle massage of the hand and in the use of a heat lamp to be used at home. The patient is cautioned about subjecting the hand to severe or repeated trauma, (65). Meyerding has treated three hundred fifty patients with Dupuytren's contraction, and has subjected one hundred

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of them to routine treatments similar to that just described with a satisfactory result in ninety percent of the cases.

In the treatment of this condition, the complete excision of the palmar fascia has been emphasized; this, as stated before, is considered the best treatment by most present day authors. However, in selected cases, these same men believe that subcutaneous fasciotomy procedure is the operation of choice. In patients who cannot or will not enter the hospital, those who are poor surgical risks because of some other disease, and in certain age individuals, the more conservative operations give a satisfactory result. It should be looked upon more as a palliative procedure than as a permanent cure. The same statements are also applicable to the nonoperative procedures, such as the use of thyroid, radiation and humanol injections. Davis and Finesilver cite an interesting case in which a bilateral contraction was present, and which gives a comparison of subcutaneous fasciotomy and a wide excision operation. Both of the patient's hands were operated upon using the two operative procedures. The hand on which the subcutaneous fasciotomy was done showed a recurrence in eighteen months, while the other hand was entirely cured, (6).

CONCLUSIONS

Baron Dupuytren, after whom this disease was named, was the first man to attempt an accurate description of the gross pathology of this condition, so far as I was able to determine.

The micropathology of Dupuytren's contraction has yet to be proved with certainty.

The etiology of Dupuytren's contraction cannot be definitely stated at this time.

No one method of treatment is ideal for all patients; case reports seem to indicate the greatest number of satisfactory results, other factors being equal, are gained from wide excision of the palmar fascia and overlying skin when involved, with careful skin grafting followed by physiotherapy.

SELECTED BIBLIOGRAPHY

1. Adams, H. V. D.; Dupuytren's Contracture. ~~Surg. Clin. of North America~~; 22:899-906, June 1942. S. Clin. North America
2. Cunningham, D. J.; Textbook of Anatomy, 5th Ed. W. Wood & Co., New York; P. 386, 1923.
3. Harper, W. F.; The Distribution of the Palmar Aponeurosis in Relation to Dupuytren's Contracture of the Thumb. ~~Jour. of Anat.~~; 69:193-195, Jan. 1935. J. ANAT.
4. Kaplan, E. B.; The Palmar Fascia in Connection with Dupuytren's Contracture. Surgery; 4:415-422, ~~Sept.~~ OK 1938.
5. Horwitz, T.; Dupuytren's Contracture; A Consideration of the Anatomy of the Fibrous Structures of the Hand in Relation to this Condition, with an Interpretation of Histology. Arch. Surg.; ~~44:687-706,~~ OK April 1942.
6. Davis, J. S., and Finesilver, E. M.; Dupuytren's Contraction, with Note on Incidence of Contraction in Diabetes. Arch. Surg.; 24:933-989, ~~June~~ OK 1932.
7. Kanaval, A. B., Koch, S. L., and Mason, M. L.; Description of Palmar Fascia; Review of the Literature and Report of 29 Surgically Treated Cases. Surg., Gyn., & Obst.; 48:145-190, Febr. 1929. Surg. Gynec & Obst.
8. Spalding, J. E.; The Fascial Spaces of the Palm: A Contribution to Their Surgical Anatomy. Guy's Hospital Reports; 88:432-439, Oct. 1938. Guy's Hosp. Rep.
9. Grodinsky, M., and Holyoke, E. A.; Fasciae and Fascial Spaces of the Palm. Anat. Record; 79:435-451, April 25, 1941. ANAT REC.
10. Koch, S. L.; Dupuytren's Contracture. J. A. M. A.; ~~100:878,~~ OK 1933.
11. Meyerdig, H. W., Black, J. R., and Broders, A. C.; Etiology and Pathology of Dupuytren's Contraction. Surg., Gyn., & Obs.; 72:582-590, March 1941. Surg. Gynec & Obst.

SELECTED BIBLIOGRAPHY

12. Meyerding, H. W., and Overton, L. W.; Bilateral Dupuytren's Contracture. Proc. ~~of~~ Staff Meet. Mayo Clin~~ic~~; 10:801-803, Dec. 18, 1935. ←
13. Nichols, J. B.; A Clinical Study of Dupuytren's Contraction of Palmar and Digital Fascia. Am. J. M. Sc.; 117:285, 1899. ^{ok}
14. Blackfield, H. M.; Dupuytren's Contracture, Treatment by Radical Excision of Palmar Fascia. Medico-Surgical Tributes to Harold Brunn; Berkeley and Las Angeles, Calif., University of California Press, 1942.
15. Meyerding, H. W.; Dupuytren's Contracture. Arch. ~~of~~ Surg.; 32:320-333, Febr. 1936. ←
16. Reid, J.; Permanent Flexion of the Fingers from Shortening and Thickening of the Palmar Aponeurosis. Edinburgh M. ~~Mag.~~ J.; 46:74, 1836. ←
17. Boyer, A.; Traite des Maladies Chirurgicales Tome Quatrienne, Cirrquienne Ed. Vol. 4, Paris, Bechet jeune, 1828.
18. White, W. H.; On Pads on the Finger Joints. ^{Quart J. Med.} Quart-~~erly~~ J. Med.; 1:479, 1907-1908. ←
19. Weber, F. P.; A Note on Dupuytren's Contraction, Comptodactylia and Knuckle Pads. ^{BRIT. J DERM} Br. Journ. ~~of Derm. & Syphilis.~~ 50:26-31, Jan. 1938. ←
20. Richer, P.; Retractione de l'Aponeurose Palmaire. Bull. Soc. de la Anat. de Par.; S.4, 2, 111 ^{ok}. 124-129, 1877. (Quote. #13).
21. Chevrot, F.; Recherches sur la Retraction de l'Aponeurose Palmaire. Thesis de Paris, 1882. ^{ok} (Quoted from Nichols, #13).
22. Nichols, J. B.; The Histology of Dupuytren's Contracture of the Palmar Fascia: Report of Microscopic Examination in ~~Two~~ Additional Cases. M. News; 75:491, 1899. ^{ok}

JOURNAL

Author, Title, JOURNAL, Vol, page, date

Book Ref

Author, Title, Place, Pub, date, page

SELECTED BIBLIOGRAPHY

23. Janssen, P.: Zur Lehre von der Dupuytren'schen Fingerkontractur mit besonderer Beruecksichtigung der Operativen Beseitigung und der Pathologischen Anatomie des Leidens. Arch. f. Klin. Chir.; 65:761, 1902. (Quoted from #11.)
24. Wainwright, L.; Dupuytren's Contracture. Practitioner; 117:263, July-Dec. 1926. (Quoted #6). ^{OK}
25. Stahnke, E.; Zur Behandlung der Dupuytren'schen Fingerkontractur. Zentralbl. f. Chir.; 1927. (Quoted from # 7 & #6.) ^{instead of "2"}
26. Beatty, S. R.; Roentgen Therapy in Dupuytren's Contracture. Radiology, 30:610-612, May 1938. ^{OK}
27. Gill, A. B.; Dupuytren's ^{ure}Contraction with a Description of Method of Operation. Ann. Surg.; 70:221, 1919. ✓
28. Meyerding, H. W.; Dupuytren's Contracture. Proc. Staff Meet. Mayo Clin.; 10:694-696, Oct. 1935. ✓
29. Mason, M. L.; Plastic Surgery of the Hands. ^{Surg. Clin. North America} Clinics of North America; 19:227-248, Feb. 1939. ✓
30. Skinner, H. L.; Operative Correction of Dupuytren's Contraction by ^{use of} Tunnel ^{skin}Graft. Surgery; 10:313, Aug. 1941. ^{OK}
31. Teleky, L.; Dupuytren's Contracture as an Occupational Disease. Journ. Indus. Hygiene & Toxicology; 21:233-235, Sept. 1939. ^{J. Indust Hyg. & Toxicol} ✓
32. Dupuytren, Baron; Selections from the Clinical Lectures Delivered at Hotel-Dieu in Paris During Session of 1831-32. (Ed. or Trans. unknown). ~~London Med. & Surg. Journ.~~; 1:266, 1832. ^{London Med. & Surg. J.}
33. Keen, W. W.; The Etiology and Pathology of Dupuytren's Contraction of the Fingers. Philadelphia M. Times; 12:370, 1881. ✓

SELECTED BIBLIOGRAPHY

34. Adams, W.; Observations on Contractions of the Fingers, (Dupuytren's Contraction), and Its Successful Treatment by Subcutaneous Division of the Palmar Fascia and Immediate Extension. London: J. & A. Churchill, 1879.
35. Anderson, W.; Deformities of the Fingers and Toes. London, J. A. Churchill, 1897. (Quoted #13).
36. Gill, A. B.; Dupuytren's Contracture. Ann. Surg.; *arc* 107:122-127, Jan. 1938.
37. Ledderhose, G.; Die Aetologie der Fascietis Palmaris (Dupuytren'sche Kontraktur). ~~München, Med. Wehnschr.~~ *München, med. Wehnschr.*; 67:1254, 1920. (Quoted from #6 and #1).
38. Hutchinson, J.; Dupuytren's Contraction of the Palmar Fascia: Dupuytren's Life and Works. Lancet; 1:285, 1917. *OK*
39. Abbott, A. C.; Dupuytren's Contraction, A Review of Literature and a Report of New Technique in Surgical Treatment. ~~Can. Med. Ass. Journ.~~ *CANAD. M.A.J.*; 20:250-253, March 1929. ✓
40. Krogius, A.; Neue Gesichtspunkte zur Aetologie der Dupuytren'schen Fingerkontraktur. Zentralbl f. Chir.; 47:914, 1920. (Quoted #6).
41. Sprogis, G.; Beitrag zur Lehre von der Vererbung der Dupuytren'schen Fingerkontraktur. ~~Deutsche~~ *Deutsche* ~~Ztschr. f. Chir.~~ *Ztschr. f. Chir.*; 194:259, 1926. (Quoted from #6.). ✓
42. Manson, J. S.; Heredity and Dupuytren's Contraction. ~~Br. Med. Journ.~~ *Brit M.J.*; 2:11, July 4, 1931. ✓
43. McWilliams, C. A.; Dupuytren's Finger Contraction. New York M. J.; 80:673, 1904. *OK*
44. Couch, H. D. C.; Identical Contracture in Identical Twins. Canadian Med. Assn. Journ.; 39:225-226, Sept. 1938. *CANAD. M.A.J.* ✓
45. Powers, H.; Dupuytren's Contracture One Hundred Years after Dupuytren: Its Interpretation. ~~Jour. Nervous & Mental Disease~~ *J. Nerv. & Ment. Dis.*; 80:386-409, Oct. 1934. ✓

SELECTED BIBLIOGRAPHY

46. Volavsek, W.; Dupuytren's Contracture; the Relation to Plastic Induration of the Penis. ~~Zeitschrift für Urologie~~; 35:173-178, 1941. (Quoted by 67, also in English in J.A.M.A.).
Ztschr. f. Urol.
47. Schubert, A.; Die Aetologie der Dupuytren'schen Kontraktur. Deutsche Ztschr. f. Chir.; 177:362, 1923. (Quoted #6). OK
48. Abbe, R.; Dupuytren's Finger Contraction; Further Remarks on its Nervous Origin. ~~Med. Rec.~~; M. Rec 33:236, 1888.
49. Grieg, D. M.; A Case of Congenital Dupuytren's Contraction of the Fingers. Edinburgh M. J.; 19:384, 1917. OK
50. Bulley, F. A.; Contraction and Thickening of the Palmar Fascia; Permanent Flexion of the Fingers. ~~Med. Times & Gaz.~~; 1:479, 1864. M. TIMES & GAZ.
51. Cooper, Sir Astley; A Treatise on Dislocations and Fractures of the Joints. Lilly & Wait, and Carter & Hendee, Boston, 1832. Second American from 6th London Ed. (2nd London ed. 1822).
53. Paget, Sir James; On the Minor Signs of Gout in the Hands and Feet. ~~Br. M. Journ.~~; 1:665-666, 1875. BRIT. M. J.
54. Fergusson, W.; On the Minor Signs of Gout in the Hands and Feet. ~~Br. M. Journ.~~; 1:666, 1875. BRIT M. J.
55. Girdwood, R.; An Unusual Case of Dupuytren's Contraction. Brit. M. J.; 2:650, 1916. OK
56. Tubby, A. H.; Dupuytren's Contraction of the Palmar Fascia and Some Other Deformities. Practitioner; 110:214, 1923. ~~Dr. P.~~
57. Byford, W. H.; The Pathogenesis of Dupuytren's Contraction of the Palmar Fascia. M. Rec.; 100:487, 1921. OK
58. Ely, L. W.; Dupuytren's Contraction. ~~Surg. Clin. North America~~; 6:421, 1926. S. Clin North America
59. Smith, Noble; Seventy Cases of Dupuytren's Contraction of the Fingers. Brit. M. J.; 1:603, 1884. OK

SELECTED BIBLIOGRAPHY

60. No reference--error in numbering.
61. Roth, P. B.; Dupuytren's Contracture in a Young Man Following Injury. Proc. Roy. Soc. Med.; OK
13:227, 1919. (Quoted from #6).
62. Davis, A. A.; The Treatment of Dupuytren's Contracture--A Review of Thirty-one Cases with Assessment of Comparative Value of Different Methods of Treatment. Brit. J. Surg.; OK
19:539-547, April 1932.
63. Keen, W. W.; Surgery, Its Principles and Practice. W. B. Saunders Co., Philadelphia; 2:569, 1907.
64. Kocher, T.; Behandlung der Retraktion der Palmaraponeurose. Zentralbl. f. Chir.; 14:481-487, OK
497-502, 1887. (Quoted from #6).
65. Meyerdig, H. W.; The Treatment of Dupuytren's Contracture. Am. J. Surg.; 49:94-103, OK
July 1940.
66. Mason, Michael L.; Plastic Surgery of the Hands. ~~Surgical Clinics of North America~~; 19:238-240, February 1939. S. Clin North America
67. Kaplan, I. I.; Peyronie's Disease. Urologic & Cutaneous Review; 46:350-352, June 1942. L
Urol. & Cutan Rev.
68. Löwy, J; Ein Beitrag zur Heredität der Dupuytren'schen Kontraktur. Zentralbl. f. inn. Med.; OK
44:51, 1923.