Dupuytren's Disease

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In the early 19th century, Guillaume Dupuytren described a flexion contracture in the palm and its surgical release. Since then, advances in understanding of the etiology, pathophysiology, anatomy, and surgical technique have refined the ability to restore function to those affected. By fully comprehending the pathoanatomy involved, careful surgical excision can lead to satisfactory results. Complication and recurrence rates, however, require that time be spent with patients preoperatively to educate them and adequately explain treatment goals.

On December 5, 1831, Baron Guillaume Dupuytren gave his famous lecture describing palmar fasciotomy for the treatment of a subcutaneous contracture.[1] Until that time he had subscribed to the common teaching of the day that theorized that this contracture was caused by a drying and shortening of the flexor tendons termed *crispatura tendinum*. It was Dupuytren's realization that the contracture was caused by the palmar fascia and his description of the surgical release that has attached his name to palmar fibromatosis.[1] In London, however, Sir Astley Cooper and Henry Cline Sr[2] had described the disease process and a therapeutic fasciotomy as early as 1777. Cline noted the need to dissect free the arteries and nerves, as well as the need for postoperative extension splinting.

Dupuytren's belief that the disease was limited to the palmar aponeurosis did not explain the extension of the disease into the fingers and subsequent proximal-interphalangeal (PIP) joint contractures. In 1833, Jean-Gaspard Blaise Goyrand presented dissections to support his theory that a new tissue was responsible for the contracture. He described this tissue to be deep to the palmar aponeurosis and superficial to the flexor tendons. This tissue extended into the tendons and was responsible for the PIP flexion contracture. He termed this tissue the *predigital bands* and did not feel that the skin itself was involved in the process. As a result, he advocated longitudinal incisions with elevation of skin flaps before cord transection.[1]

For the next 20 years the surgical care of this disease spread throughout Europe. In the 1840s, with the advent of anesthesia, more meticulous dissection could be undertaken. With that advance, Fergusson[3] was able to perform the first complete fasciectomy for Dupuytren's contracture.

From the mid-1850s until the present day, advances in the understanding of antisepsis, pathophysiology, surgical technique, and postoperative rehabilitation only have been refinements of the treatments and theories proposed in the mid-19th century.

Pathophysiology

The 2 cells responsible for Dupuytren's disease are the fibroblast and the myofibroblast. The morphologic characteristics of the disease process were

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1531-0914/03/0301-0006$35.00/0
doi:10.1053/jssh.2003.50005

first described in studies examining the ultrastructure of inflammatory tissues in the nodules of fascia affected by Dupuytren's.[4,5] The 3 stages described by Chiu and McFarlane[6] implicated the myofibroblast as the cell responsible for contracture formation. The early stage shows nodules with proliferative perivascular hyperplastic fibroblasts. The active stage has nodular thickening and joint contracture with a predominance of myofibroblasts. The advanced stage has fibrotic thickening with less numerous myofibroblasts and fibroblasts in mature collagen fiber stroma.[6] These stages correlate with Luck's[7] described proliferative, involutional, and residual stages. Myofibroblasts appear during the proliferative phase of the disease and come to compromise almost all of the cells present in the nodules. The involutional phase nodules are highly cellular, but individual myofibroblasts are more slender and smaller and begin to line up along lines of stress. Finally, in the residual phase, the composition of the nodules shows hypocellular populations of cells that are slender and aligned with thick bands of collagen, giving them a tendon-like appearance. This appearance and disappearance of myofibroblasts is similar to what occurs in wound healing as shown by Darby et al.[8]

Ultrastructural, immunohistochemical, and biochemical studies have shown that the fibroblast in Dupuytren's disease is identical to fibroblasts in normal tissues. Recently, however, a difference in androgen receptor expression in Dupuytren's myofibroblasts has been noted that may explain the predominance of Dupuytren's disease in men.[9] Dupuytren's fibroblasts are found in a higher concentration, they surround the microvasculature,[10] and they contain a higher concentrations of type III collagen. The increased cellularity in Dupuytren's patients may be caused by local ischemia at a microvascular level.[11] This local ischemia stimulates increased production of fibroblasts that organize themselves along lines of mechanical stress, creating the cord-like structures found in the palmar fascia. The mechanism by which ischemia stimulates fibroblast production has been tied to the production of oxygen free radicals that occur in hypoxic tissues.[5,10,11] Increased free fatty acids and short-chain fatty acids in Dupuytren's contracture are suggestive of the local hypoxia that exists.[5]

Transforming growth factor β1 plays an important role in regulating the transformation of fibroblasts to myofibroblasts and in the production of collagen.[12,13] Platelet-derived growth factor, as well as basic fibroblast growth factor, have been shown to stimulate proliferation.[14-16] Transforming growth factor β1 can promote the expression of smooth muscle actin in cultured Dupuytren's myofibroblasts and normal palmar fascia fibroblasts.[12] The presence of transforming growth factor β1 may play a significant role in the promotion of the contraction of myofibroblasts in the palmar fascia, thus producing the pathologic contracture of Dupuytren's disease.[5]

The diseased tissue of Dupuytren's presents as nodules and cords, each of which has a distinctive histologic makeup. Nodules are a dense collection of myofibroblasts undergoing high metabolic cellular activity.[17] Dupuytren's cords by contrast contain no myofibroblasts but are highly organized collections of collagen similar to tendon. Electron microscopic studies have shown that contracture of the myofibroblasts in the nodules is responsible for the digital flexion contracture. Nodules pull on cords that have extended pass adjacent joints—this contracture across joints produces the pathologic flexion contractures of Dupuytren's disease.[17]

Etiology

Many factors have been considered to predispose an individual to Dupuytren's disease. Alcohol consumption, smoking, human immunodeficiency virus, diabetes, epilepsy, genetics, and manual labor all have been implicated as potential etiologic factors, however, there is little conclusive evidence regarding these risk factors.

The exact role that alcoholism plays in the development of Dupuytren's contracture is not clear. Noble et al[18] showed a higher incidence of the disease in alcoholic patients and proposed that this was related to the liver disease present in this group. There are no studies that show the quantity of alcohol that leads to Dupuytren's disease or what the cumulative affect is of alcoholism on Dupuytren's. Smoking has been suggested as a causative factor and there is some evidence that the increased incidence of smoking in alcoholic patients actually is responsible for the rate of Dupuytren's disease in this group.[19]

There has long been an association between epilepsy and Dupuytren's disease. The reported incidence in the literature ranges form 8% to 57% and has

prompted questions regarding the possible role of antiepileptic medication as a possible causative agent.[20] Although there is some evidence that the rate of the disease is higher in epileptic patients, drug therapy has not been implicated clearly as the cause of this increased incidence.[21]

The association between Dupuytren's contracture and manual labor has its foundation in Dupuytren's original statement that, ". . .most individuals who are affected with this condition have been obliged to use the palms of their hands constantly and to handle hard bodies."[1] There have been numerous investigations trying to elucidate the relationship between the development of Dupuytren's contracture and manual labor or repetitive trauma. There has been little conclusive evidence to support this as the cause for most cases of Dupuytren's disease. Because of the medicolegal considerations that surround the treatment of Dupuytren's disease after trauma, McFarlane[20] defined 5 criteria to help establish a causal relationship. They are: (1) appearance of the disease in men aged 40 years or less or women aged 50 or less, unless they exhibit signs of a strong diathesis; (2) in bilateral disease, the uninjured hand should not develop disease in men until after the age of 40 and in women until after the age of 50; (3) there is objective evidence of injury to the hand; (4) the disease develops in the area of injury; and (5) the disease appears within 2 years of injury.

The prevalence of the disorder among those of Northern European descent, combined with its prevalence in Australia (largely settled by this Northern European population), provided early evidence for a genetic etiology or predisposition. It is significantly less common in Mediterranean populations and in Asia. Inheritance appears to follow an autosomal-dominant model with variable penetrance.[21,22] Investigation into histocompatibility antigens and their involvement in Dupuytren's disease has been performed. Although the human leukocyte antigen is found in many patients with Dupuytren's disease, its exact association remains unclear and more research is needed to elucidate its role.

Anatomy and Pathoanatomy

To understand the pathologic process responsible for contractures in Dupuytren's disease, it is necessary to be familiar with the normal anatomy of the fascial layers of the palm and fingers (Fig 1). The normal bands and ligaments lead to a predictable pattern of the formation of pathologic cords and nodules.

The palmar aponeurosis is a triangular facial layer with its apex overlying the palmaris longus.[25,26] This fascial sheet divides distally into the pretendinous bands that come to the distal edge of the metacarpals. At this level, the pretendinous band splits into 3 layers.[27] The superficial layer attaches to the skin at the palmar digital crease. The central layer gives rise to 2 spiral bands. These travel distally and dorsally to form the web space coalescence. As they progress distally, they rotate 90° and pass from the coronal plane into the sagittal plane. The deepest layer of the pretendinous band dives dorsally around the metacarpophalangeal (MCP) joint to insert on the interosseous muscle fascia and the deep transverse metacarpal ligament. These sagittal extensions, termed the *septa of Legue and Javara*, create 7 compartments that contain the flexor tendons and the neurovascular structures. These septa join the spiral bands and natatory ligaments in the web space coalescence.[25]

The superficial transverse palmar ligament runs perpendicular and deep to the pretendinous bands, proximal to the division of the spiral bands. Distal to this division, the natatory ligaments run transversely. Although the superficial transverse palmar ligaments essentially remain in the coronal plane, the natatory ligaments give rise to deep fibers that join the web space coalescence and fibers that attach to the flexor tendon sheath at the level of the MCP joint.

The web space coalescence gives rise to a sagittally oriented fascial layer in the finger termed the *lateral digital sheet*. These sheets are lateral to the neurovascular bundle. The lateral digital sheets give off coronal fibers both volar and dorsal to the neurovascular bundle that insert into the phalangeal periosteum and flexor tendon sheath. The volar structure is termed *Grayson's ligament* and the dorsal structure is termed *Cleland's ligament*.

The pathologic changes seen in Dupuytren's disease usually commence with nodule formation in the distal palm over the pretendinous bands (Fig 2). Pits are formed between the distal palmar crease and the MCP crease owing to contracture of the superficial layer of the pretendinous band that inserts on the skin. As involvement progresses to the main portion of the pretendinous band, the most common cord, the pre-

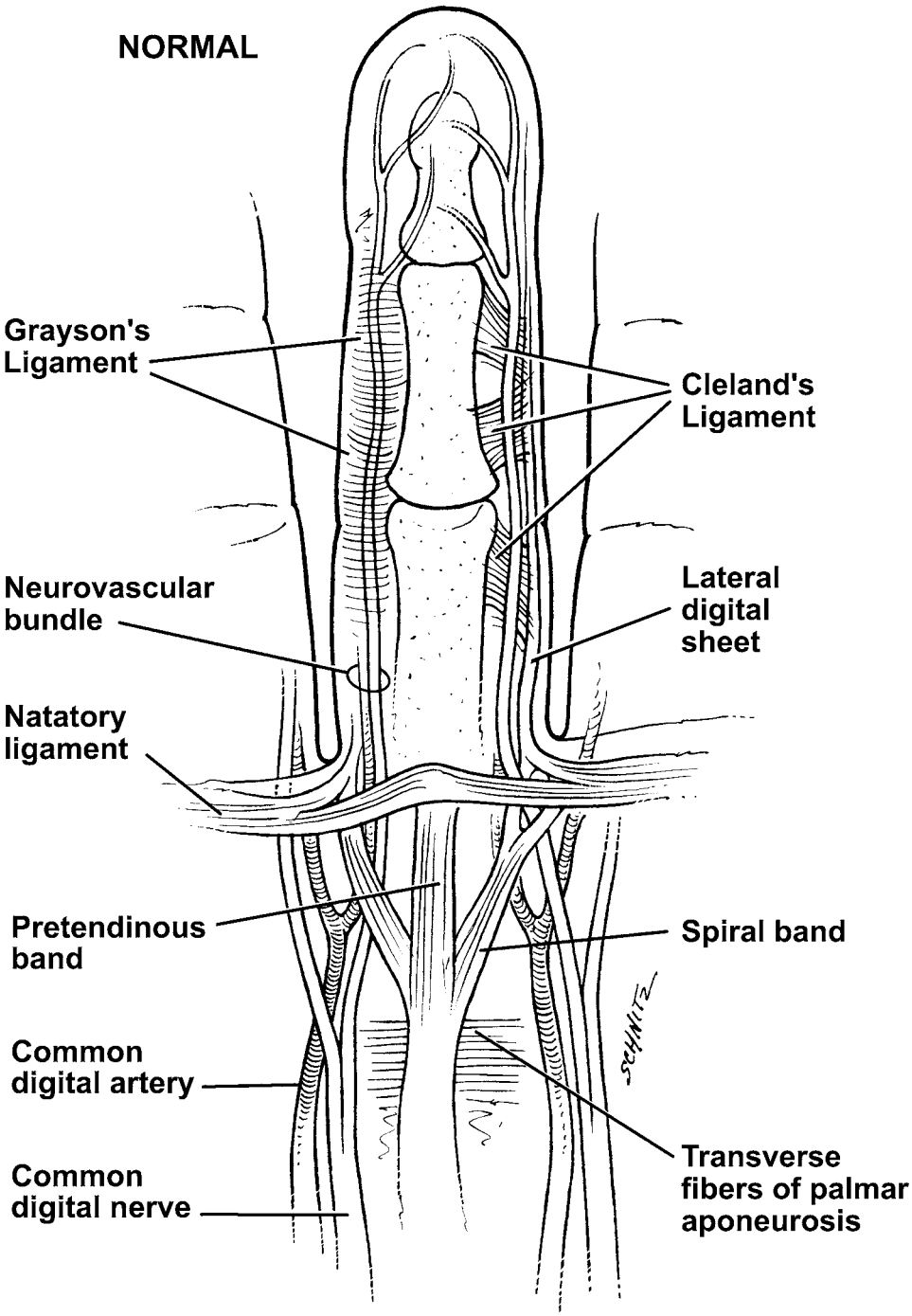

FIGURE 1. Normal anatomy palm and finger.

tendinous cord, is formed (Fig 3).[28] As the spiral bands are affected, they form, in conjunction with the lateral digital sheath and Grayson's ligament, the spiral cords (Fig 4). These cords lead to the MCP flexion contractures seen. Natatory cord contraction leads to PIP joint contraction and limits abduction via its attachments to the web space coalescence and the lateral digital sheath. Formation of a central cord occurs without the presence of a normal fascial precursor. This cord overlies the proximal phalanx and arises at the level of the bifurcation of the pretendinous band into the spiral bands. Finally, the lateral digital sheath can be affected, forming a lateral digital cord distal to its involvement in the spiral cord.

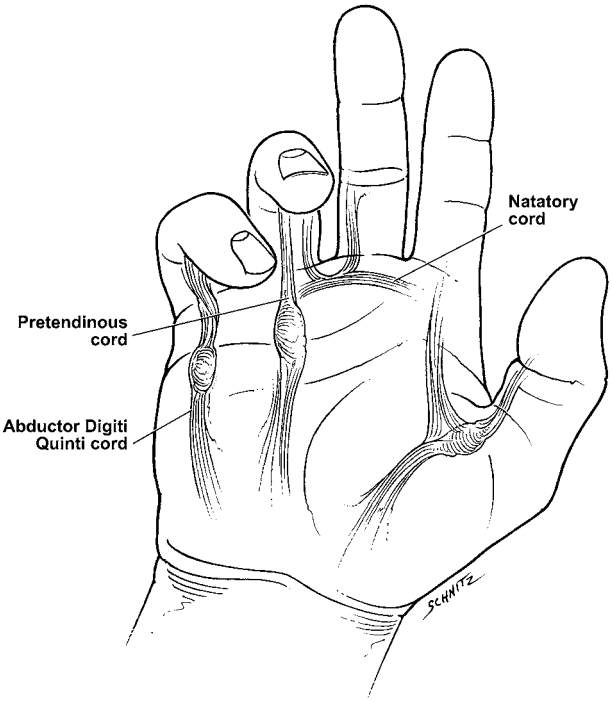

FIGURE 2. Anatomic locations of Dupuytren's nodules and cords.

Knowledge of the course of the neurovascular bundles in Dupuytren's disease is crucial to safe surgical excision. In the palm, the digital nerve and artery lie deep to the palmar aponeurosis. As the spiral bands travel dorsally and laterally the neurovascular structures come to lie superficial to, and to the midline of, the fascia. As the fascia thickens and contracts it becomes straighter, causing the neurovascular bundle to coil around the spiral cord. The greater the contracture of the cord the more superficial and more midline the neurovascular structures are displaced.[25,28]

Isolated digital cords have been well described and originate and insert entirely distal to the MCP joint. They are most common in the small finger (Fig 5). They originate from the base of the proximal phalanx or from the adductor digiti minimi tendon and form a cord that appears very similar to the spiral cord. This cord displaces the nerve and artery toward the midline (Fig 6).[29]

Cords formed in the thumb come from 3 fascial precursors: the natatory ligament, a rare thumb pretendinous band, and the superficial transverse ligament. These structures cause thumb adduction and MCP flexion but they do not disturb the position of the neurovascular bundle because all 3 lie superficial to it.

Diagnosis

The typical patient presentation is that of a man in his fifth decade of life who has progressive involvement of Dupuytren's disease in both hands that is asymmetric. The differential diagnosis of the disease includes hyperkeratosis, callous formation (caused by excessive use in laborers), masses such as giant-cell tumors and inclusion cysts, and flexor tendon pulley ruptures in rheumatoid arthritis patients.[30,31]

The earliest sign of Dupuytren's disease is the nodule that appears between the distal palmar crease over the metacarpal head; however, it is not uncommon for patients to present much later in the disease process. Frequently cords and contractures already have formed and this may cause the patient to seek treatment. After the nodule development, skin changes are observed. These include thickening of the skin, fibrosis of the subcutaneous fat, and pitting of the skin.

The formation of cords occurs as the nodules regress, although they may both be present simultaneously. The cords may remain in the palm or progress into the fingers. The ring finger is the most commonly affected digit followed in order of frequency by the small finger, thumb, middle finger, and index finger. Pretendinous cords form in the palm, and, as they mature and contract, they cause flexion contractures of the MCP joints. Digital cords, if present, appear next and cause PIP flexion contractures.

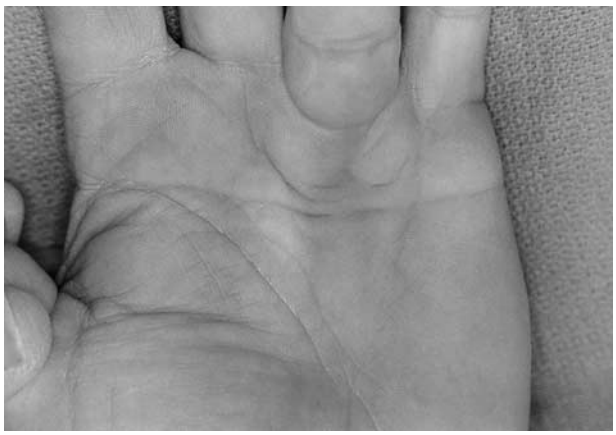

FIGURE 3. Pretendinous cord with palmar nodule ring finger.

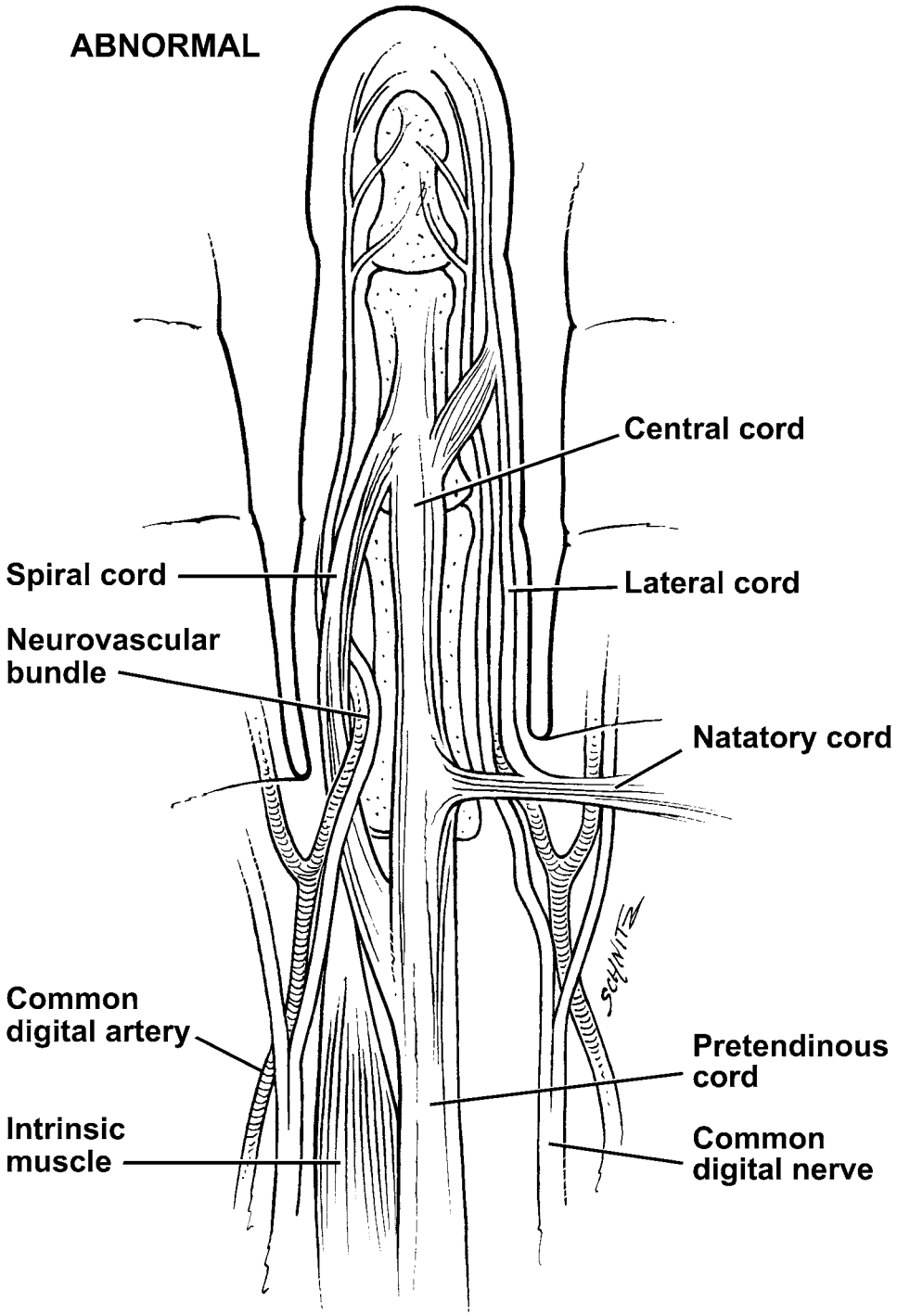

FIGURE 4. Abnormal palmar and digital anatomy.

Dupuytren's disease is systemic and sites other than the palm and fingers can be involved. Regionally, the dorsal PIP joints may show subcutaneous fibrosis termed *knuckle pads* or *Garrods nodes*. These are found in up to 54% of patients and they are predictive of bilateral involvement.[31] Distal sites of involvement include the plantar surfaces of the feet (Lederhose disease) and the penis (Pyeronie disease).

The term *Dupuytren's diathesis* refers to a patient with severe disease. These patients are more likely to be younger, have bilateral involvement, have distal sites involved, and have more rapid progression of the

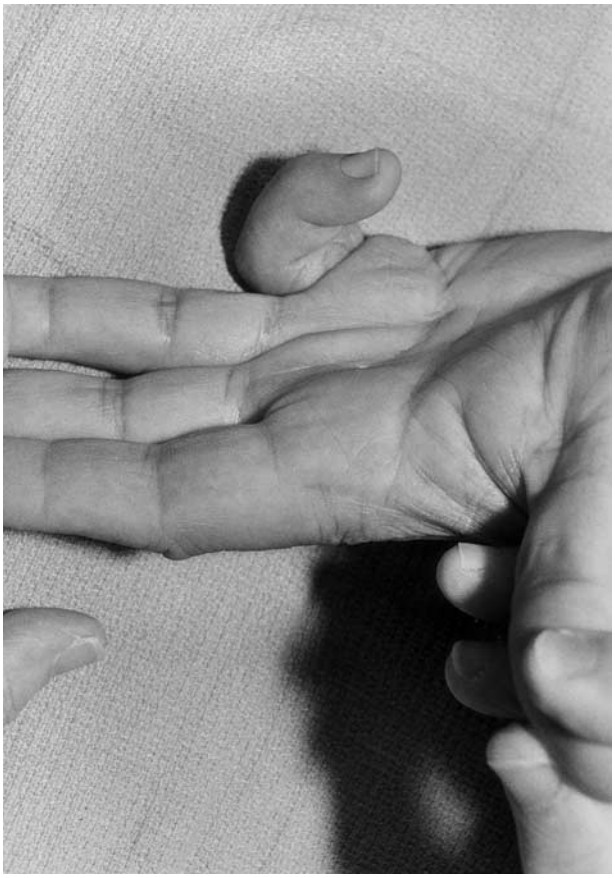

FIGURE 5. Flexion contracture small finger caused by Dupuytren's.

disease. Another atypical presentation is of the isolated digital cord. These patients may never have nodule formation in the digit with the cord, but they almost always have other signs of Dupuytren's disease in the remainder of the hand.

Nonsurgical Treatment

Initially, some patients presenting with Dupuytren's disease may be managed nonsurgically. Frequently, a patient presents with a newly diagnosed nodule prepared to have it surgically excised. The first steps in treatment are to teach the patient about the disease and its likely course. Only when the patient presents with advanced disease should surgery be recommended. If patients present early in the disease process, they should be instructed in the table-top test. They are instructed to make certain that, at regular intervals, they can place their hand flat on the table. If they lose this ability, they are encouraged to follow-up so that joint contracture may be reassessed.

A variety of nonsurgical treatments for Dupuytren's disease have been attempted in the past with limited success. Before the development of surgical techniques of treatment, traumatic rupture, therapeutic or accidental, was the only method of cure. Continuous elongation techniques using external fixation is a more modern approach to this method. However, high recurrence rates have been shown with this procedure if it is not followed-up with fasciectomy.[33] Other proposed modalities include radiation therapy, dimethyl sulfoxide, physical therapy, and steroid injections. Although some still support these techniques, there is little objective evidence that supports them as a long-term solution in Dupuytren's disease. Injection of nodules with triamcinolone acetonide has been shown to have softening effect on nodules, but there is a 50% recurrence rate at 1 to 3 years.[34]

Recently, direct cord injection with collagenase has been investigated. In a phase II clinical trial 29 patients with 34 MCP contractures and 9 PIP contractures were treated. Eighty-two percent of MCP contractures and 44% of PIP contractures were corrected to full extension within 2 weeks. Seventy-eight percent of PIP contractures were corrected to within 15° of full extension.[35] Randomized, blinded clinical trials have yet to be performed, but this may prove to become a reasonable alternative to surgical intervention.

Surgical Treatment

Indications for surgical intervention include loss of function, rapid progression, and flexion contracture. Frequently cited indications include MCP joint contractures greater than 30° and PIP contractures of any amount (Fig 7). This indication for early PIP release stems from the resistance of the PIP joint to full extension after Dupuytren's excision. There is no evidence that early PIP release provides better outcomes and some have shown that mild PIP flexion deformities actually may be made worse with early release.[36]

Although joint contracture is one factor that plays a part in the surgical decision-making process, these values are only a guide to treatment. Patients must understand the surgery they are undertaking and the risks and benefits of this intervention. They must be

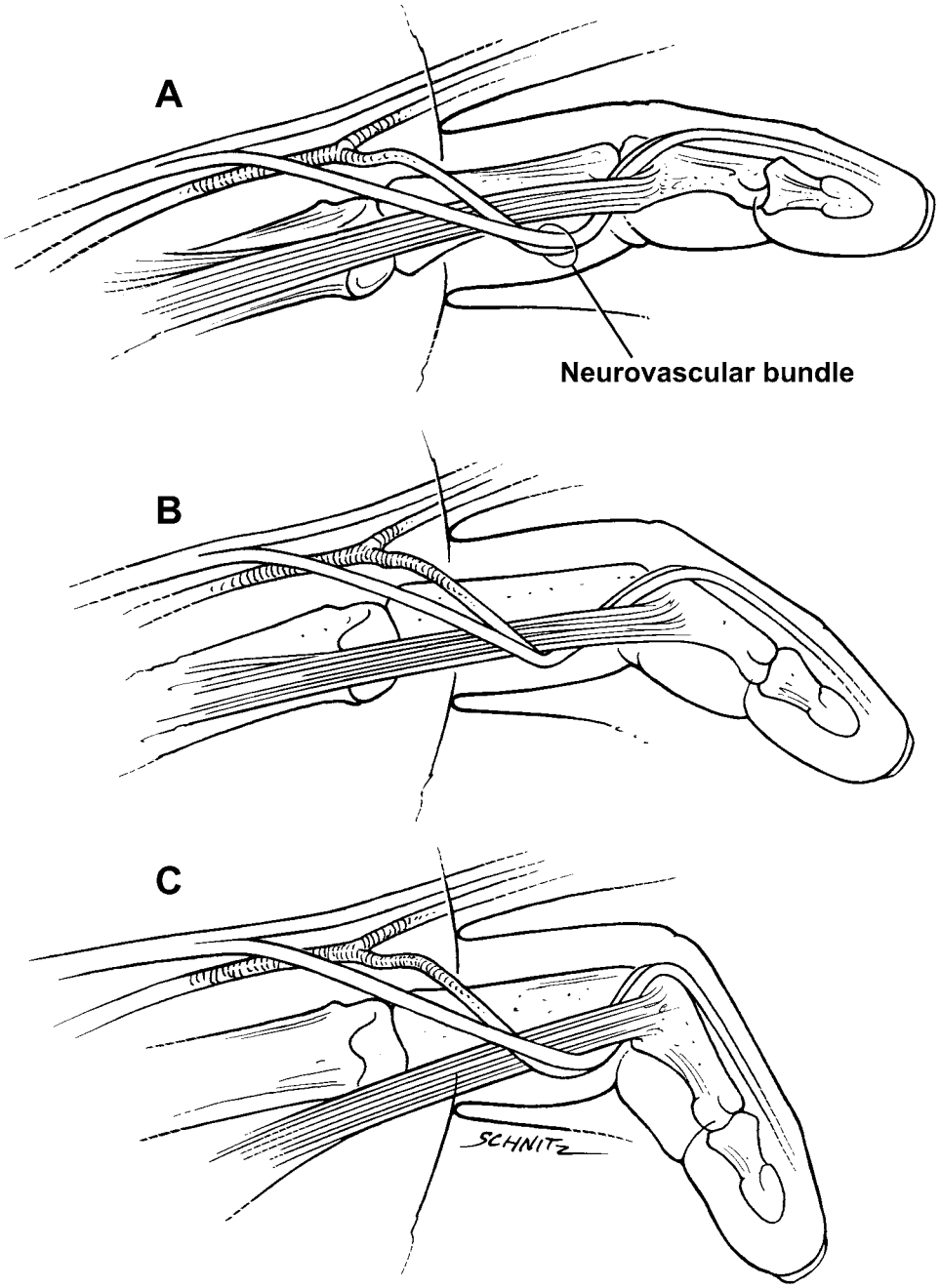

FIGURE 6. Neurovascular displacement by a spiral cord.

prepared for the extensive therapy required after surgery and the possible complications that they may encounter. They also must have reasonable expectations regarding the outcome of their surgery. The patient's history also plays a role. Patients who note rapid progression of the disease or other signs of Dupuytren's diathesis should be encouraged to consider earlier surgical intervention. Those that complain of severe loss of function are better surgical candidates than those that only recently noted changes in their skin.

Surgical techniques available include dermofasciectomy, radical fasciectomy, partial fasciectomy, and fasciotomy. Management of the palm includes primary closure, skin grafting, and the McCash open palm technique. Incision choices in the fingers include Brunner incisions, longitudinal incisions with multiple Z-plasties, and V-Y plasties.[37]

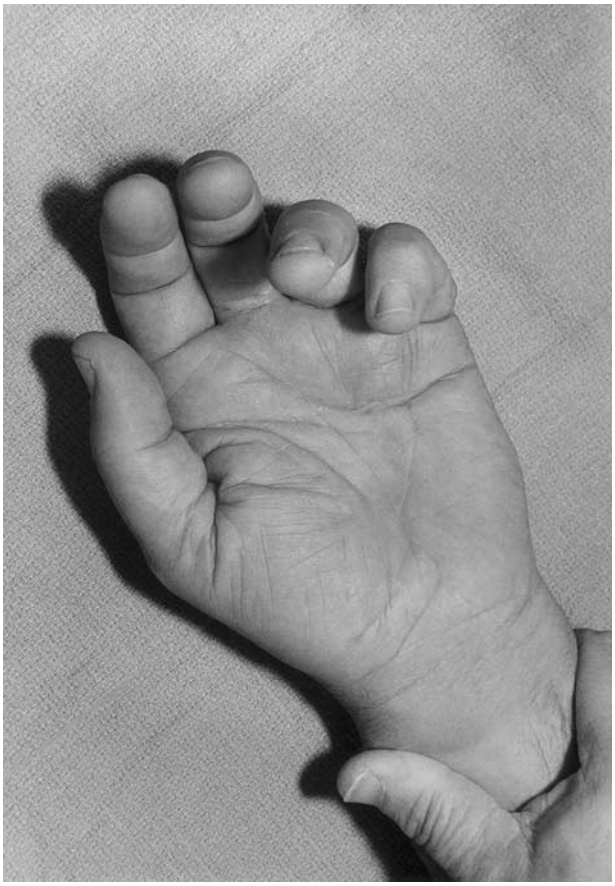

FIGURE 7. Preoperative flexion contractures of ring and small finger with surgical indications.

Dermatofasciectomy involves removal of the fascia as well as the overlying skin. This excised skin is replaced with full-thickness skin grafts. Proponents of this method report excellent results. Critics, however, cite poor graft sensibility and contour, as well as increased stiffness from prolonged immobilization as possible complications. Although recurrence rarely is seen at the site of the graft, it remains a problem because it recurs at the edges of the graft as well as in ungrafted areas. These factors combined with donor site morbidity make dermatofasciectomy a less popular procedure.

Radical fasciectomy initially was thought to decrease recurrence rates by removing all of the tissue involved in the disease process. There were high complication rates and disease recurrence remained a problem. As a result, this method rarely is recommended.

Partial fasciectomy is the most commonly used technique for the management of Dupuytren's disease. Through any of the earlier-listed incisions, fascia that has formed cords or nodules is excised. Normal-appearing fascia is left behind. It is accepted that some small amounts of diseased fascia may be left behind. For most patients this method provides the best combination of reasonable outcomes with a more limited risk for complications.

Single or multiple fasciotomy may be a good option in patients with limited disease, in elderly patients with multiple surgical risk factors, and as a preliminary treatment for very severe disease. The results after fasciotomy usually are not acceptable for other groups of patients. The procedure can be performed quickly and safely, limiting anesthetic time for the elderly. In patients with severe contractures that cause macerated skin, fasciotomy can be a definitive procedure in a nursing home patient, or it can be the first step in a staged procedure that allows the skin to heal and be cleaned before formal fasciectomy.

Management of the palmar skin may be handled in several ways. Primary closure is performed frequently and allows for early motion, limited patient inconvenience, and good skin sensibility. It has a higher rate of hematoma formation, skin necrosis, and more complicated flap design is required to allow closure in some cases. Skin grafting of open areas is recommended by some. As mentioned before, this method requires longer immobilization, has poor sensibility and contour, and does not eliminate recurrence. The McCash open palm technique was described in 1964 and involves a transverse incision at the midpalmar crease. Additional incisions are made to gain access to the fingers. Although the remainder of the incisions are closed primarily, the transverse incision is left open to heal by secondary intention. This method decreases hematoma formation and skin graft morbidity, as well as increases patient postoperative comfort. There is no increase in infection rates and there is an increase in active range of motion.[38] The largest disadvantage of this technique is the wound care that the patient must perform on the open incision for 3 to 5 weeks after the surgery.

Incisions into the digits are chosen based on the particular deformity present as well as the appearance of the skin over which the incision will lie. In extreme flexion contractures, a longitudinal incision is likely to be the best option. This incision, combined with

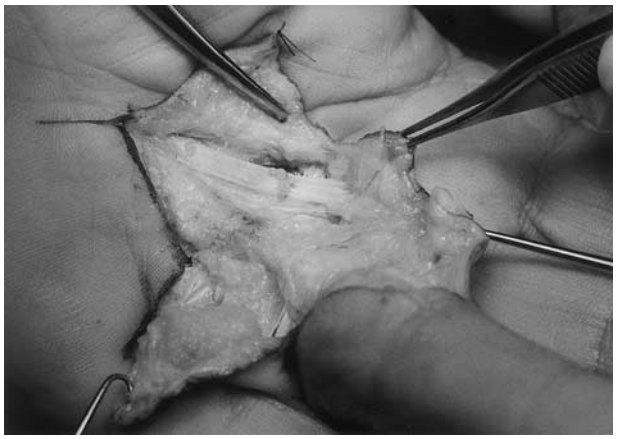

FIGURE 8. Pretendinous cord in the palm during dissection.

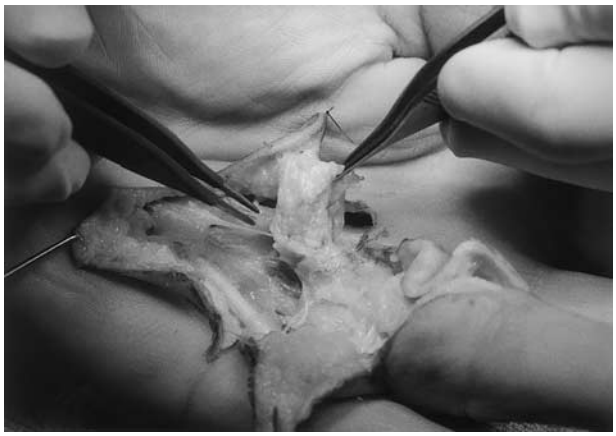

FIGURE 9. Initial palmar dissection isolating the neurovascular bundle.

multiple Z-plasties, provides increase length and technically is easier to perform when operating underneath the severely flexed finger.

Brunner incisions provide excellent exposure to the finger and should be planned so that the base of the flaps are in less diseased areas. It is expected that the tips of the flaps will be thin given the subcutaneous nature of the disease. To successfully preserve these tips, however, the flap base must not also be compromised in the dissection. Similar to the Brunner incision is the V-Y plasty–type incision. It combines the advantages of the Brunner incision with the ability to provide some additional length to the skin.

Regardless of the incision used or amount of fascia to resected, a methodic approach is required to ensure a complete and safe resection. In the palm, the neurovascular structures lie deep to the involved fascia (Fig 8). The proximal edge of the fascia is identified and the neurovascular bundle on either side is located and retracted laterally (Fig 9). A clamp is passed underneath this proximal fascial edge and it is sectioned transversely. From this point, the dissection proceeds in a proximal-to-distal fashion within the remainder of the palm (Fig 10). Although the superficial dissection does not involve the neurovascular bundles, care must be taken when the septae of Legue and Javara are resected down to the level of the flexor sheath. Because of the pull of the digital cords on the bundle in the finger, the nerve and artery can be pulled more toward the midline in the distal portion of the palm over the natatory ligament (Fig 11).

If the disease continues into the finger, a meticulous dissection of the neurovascular bundle is required. Even with the initial incision, it is important to be wary of the nerve because it can be transposed to the midline and superficially to lie directly beneath the skin. If a spiral cord is present, dissection will need to proceed both proximal-to-distal and distal-to-proximal along the course of the neurovascular bundle to safely free it from the diseased fascia (Fig 12). It is critical that the neurovascular bundle is identified before any tissue is cut (Fig 13).

Care must be taken to release all bands of fascia that are involved in the disease process. In particular, the abductor digit minimi insertion must be released in many cases of small finger involvement. Occasionally,

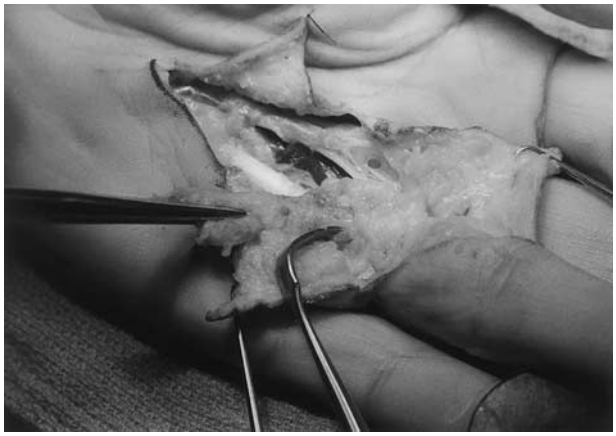

FIGURE 10. Dissection of pretendinous cord with neurovascular bundle and flexor tendons well visualized.

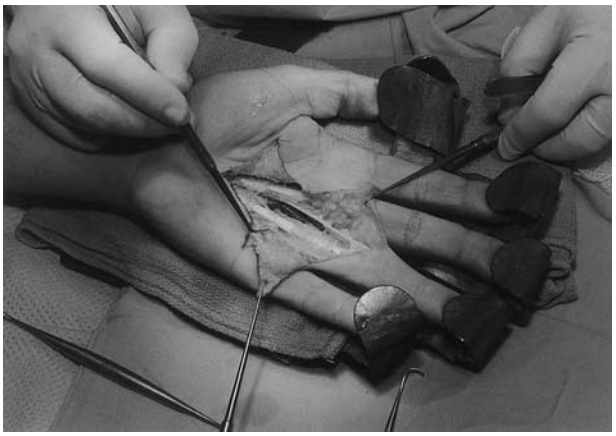

FIGURE 11. Full dissection completed with neurovascular bundles that should be carefully protected.

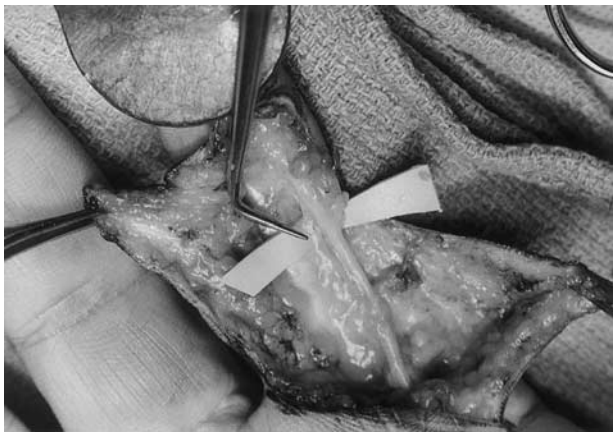

FIGURE 13. Neurovascular bundle seen after excision of diseased cord.

the disease will proceed distally to cause a distal interphalangeal contracture.

Once the fasciectomy is complete, attention must be focused on the joint contractures. MCP contracture usually resolves with fasciectomy alone. Proximal interphalangeal contracture, however, is much more difficult to resolve.[39] If, after fasciectomy, PIP extension is not restored, a transverse incision in the flexor sheath is made at the level of the A-3 pulley. This alone may provide some increase in extension, but usually a volar plate release is required. If complete extension is not obtainable after volar plate release, the collateral ligaments may be released to make further progress. Once a complete volar plate and collateral ligament release is performed, no further soft-tissue release should be performed (Fig 14). Transection of further soft-tissue elements may compromise the vascularity and viability of the digit. Any remaining PIP flexion should be addressed after surgery with stretching in hand therapy.

Therapy

Patients should be seen by a hand therapist with several days of surgical fasciectomy. Our protocol has patients in therapy by postoperative day 5. At the time of their first visit, a forearm-based extension splint is fashioned to be worn at all times. Therapists assess vascular changes after splint application. If distal ischemia is noted in full extension, the fingers are allowed to flex until adequate blood flow returns, and

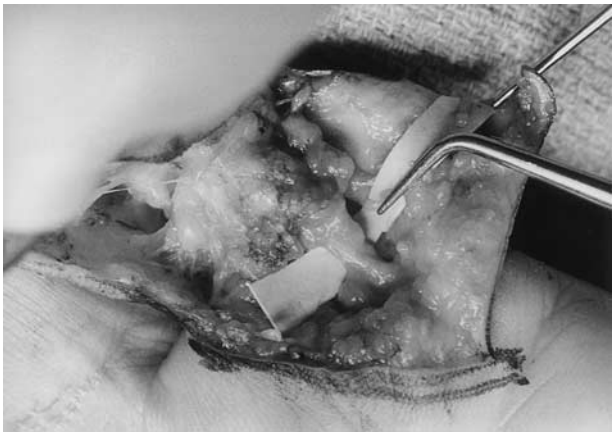

FIGURE 12. Spiral cord with neurovascular bundle pulled midline.

FIGURE 14. Small finger fully released at the PIP joint with drain in place. Skin closure using a flat silastic drainage and Brunner incision.

then the splints are extended serially over the next several visits. Active and active-assisted motion is begun and full passive range of motion should be tolerated by the end of the first week. The dressing is changed at this first visit, with dressing change instruction given to patients that have undergone an open-palm technique.

Beginning at the third postoperative week, the splint is weaned to be worn at night only. Nighttime splinting may continue for up to 6 months. At this time the patient may begin to use the hand for light activities. If the scar is closed, scar massage is begun. By the sixth postoperative week, light resistive exercises are added and the splint is shortened to a hand-based splint for comfort. If PIP flexion contractures recur or persist, serial casting is begun at this time.

If patients are doing well after 8 weeks, they frequently can be discharged from therapy. A follow-up appointment, however, generally is made at the third or fourth postoperative month to ensure that range of motion, and particularly extension, is maintained.

Outcome

Many studies have reported the long-term results of surgical excision of Dupuytren's disease. Recently, motion was reported to increase from 46% of normal preoperatively to 96% postoperatively by using the open palm technique. At as much as 19 years of follow-up evaluation, motion remained 92% of normal.[40] Another study showed a 79.5% improvement in motion immediately after surgery and 83.5% maintained a 74% improvement at 5.6 years follow-up evaluation. Risk factors for poor results included early age at onset, severe PIP involvement, and involvement of the small finger.[41]

Recurrence of Dupuytren's disease has been reported to occur in up to 74% of patients. A report of 76 hands with over 12 years average follow-up evaluation showed a 47% recurrence rate requiring surgery and with up to 74% of patients having some form of recurrence.[42] Recurrence can be difficult to treat and is seen more commonly in patients with early disease onset, a positive family history, rapid disease progression, and distal disease. The pathoanatomy is less consistent in the previously operated field and there is a greater incidence of neurovascular injury. One study showed a 68% rate of decreased sensation using Semmes-Weinstein monofilament testing and there was an 11% incidence of completely anesthetic digits. This same study reported better results with local flap reconstruction than with full-thickness skin grafts. Of note, there was a 95% satisfaction rate after revision surgery despite the sensory deficits, again highlighting the importance of patient education and expectation management.[43]

It is important to differentiate between recurrence (new cord formation) and persistence of PIP flexion contracture. These postoperative PIP contractures are common after complete release and are best dealt with by making patients aware of this risk. These contractures can be secondary to a primary joint contracture or caused by attenuation of the central slip with resultant weakness of extension. Treatment of recurrent disease that causes new PIP contractures can be the most challenging revision surgery. In these instances, PIP joint fusion may be required. In cases of compromised vascularity or in the insensate finger, amputation may be indicated.

Complications

Major intraoperative complications include injury to a digital nerve or digital artery.[44] These usually may be avoided by a thorough understanding of the anatomy and patient dissection of the facia. Nerve transection should be treated with primary neurorrhaphy. Transient paresthesias normally occur owing to traction injuries to the nerve at the time of dissection. The most important factor leading to a satisfactory outcome from this complication is preoperative patient education. Patients tolerate this complication well when they are aware of the possibility and know that it is likely to resolve spontaneously. To be certain that a traction injury is responsible for sensory changes, careful tracing of the nerve should be performed after fascial resection to assure that the nerve remains in continuity.

Arterial transection should likewise be followed-up by primary repair.[45] Loss of digital perfusion can occur without direct arterial transection when severe flexion deformities exist. Excessive stretching of the vessel can lead to spasm or rupture. The first is treated with finger flexion, warm saline, local lidocaine application, and, if needed, systemic heparin application. The latter is treated by interpositional vein graft because traction injuries to the vessel cause significant

intimal damage proximal and distal to the rupture site.

Postoperative complications also include hematoma formation, infection, flap necrosis, and recurrence. Hematomas can be prevented by meticulous hemostasis combined with the use of a drain. The open palm technique also decreases the incidence of hematoma complications. Infections are best prevented by recognition of significant risk factors such as diabetes or chronic systemic steroid use. In these patients, perioperative antibiotics may be used to decrease the chance of infection. Infections, once recognized, are treated aggressively with debridement and parenteral antibiotics.

The incidence of flap necrosis can be minimized by appropriate incision choice, precise flap planning, careful elevation of flaps to prevent buttonholing through flap edges, and hematoma prevention. If this occurs, treatment usually is healing via secondary intention. In large defects where there is a well-vascularized bed, skin grafting may be performed. If there is exposed tendon or nerve, a local flap such as a cross-finger flap may be required for coverage. All of these procedures may result in increased stiffness owing to the need for immobilization.

Conclusion

Dupuytren's disease is a complex process that can significantly affect the function of the hand. Although surgical excision can provide improved function, there are limits to the efficacy of this treatment. With a complete understanding of the disease process and risk factors involved, the surgeon can guide the patient to a treatment plan that, despite its limitations, can lead to a satisfactory outcome.

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