Dupuytren’s Disease

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educational objectives

As a result of reading this article, physicians should be able to:

1. Identify the basic science associated with the pathogenesis of Dupuytren’s disease as it is defined to date.

2. Define the anatomy, pathologic and nonpathologic, involved with Dupuytren’s disease.

3. Identify the clinical presentation of patients with Dupuytren’s disease.

4. Discuss traditional and cutting-edge operative and nonoperative treatment options, including postoperative care.

ABSTRACT

Dupuytren’s disease is a benign contractile disorder of the hand. The condition commonly affects older men of Celtic descent. Although fibroproliferation and collagen alteration play a role in its etiology, defining a cause remains elusive. Nonoperative intervention for advanced disease has shown only short-term benefit. Therefore, open fasciectomy has become the mainstay of treatment. Associated morbidity and recurrence have prompted investigation into less invasive techniques, including needle aponeurotomy and enzymatic fasciectomy. Data from phase III studies using injectable collagenase are changing treatment algorithms. Postoperative rehabilitation includes nighttime splinting and immediate active range of motion exercises to facilitate return to function.

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Felix Plater of Switzerland is credited with presenting in 1614 the first accurate description of the palmar fibromatosis now known as Dupuytren’s disease. In 1777, English surgeon Henry Cline suggested a role for the palmar fascia in this contractile disorder and, in 1808, specified involvement of the palmar aponeurosis. Cline further proposed operative release of the contracted aponeurosis as a treatment option while stressing the importance of understanding the proximity of abnormally positioned neurovascular structures during dissection.

Sir Astley Cooper, a student and apprentice of Cline, theorized that a chronic inflammatory condition of the palmar aponeurosis could lead to contracture of the involved digits, a theory proposed years before Dupuytren’s work on the disorder was formally published. Cooper proposed repetitive trauma as an etiologic factor for the onset of the contracture and agreed with his mentor’s suggestion that operative release of diseased tissue could be beneficial.

In 1831, Guillaume Dupuytren, a French anatomist and military surgeon, presented his detailed anatomic and pathologic description of what is now known as Dupuytren’s disease. Given his stature in the medical community, the contracture came to bear his name.

**Basic Science**

A large influx of information over the past 3 to 4 decades regarding the basic science of Dupuytren’s disease has improved our understanding of the primary role fibroblasts and myofibroblasts play in disease development and progression. Myofibroblasts are the primary cell type found in diseased tissue. These cells have been shown to be biochemically similar, if not identical, to myofibroblasts found in normal tissue. This information brings into question the former theory that an altered form of myofibroblast is responsible for Dupuytren’s disease.

Studies of collagen type and deposition in diseased tissue have shown higher concentrations of type III collagen resulting in a higher ratio of type III collagen to type I collagen. These data have confirmed the role alterations in collagen development and deposition play in Dupuytren’s disease.

Cellular ischemia results in cytokine-mediated transformation of fibroblasts to myofibroblasts in diseased tissue. This mechanism of transformation is thought to be a cause of increased myofibroblast proliferation leading to eventual contracture. In this algorithm, an episode of local ischemia stimulates fibroblast proliferation in a mechanism still incompletely understood but thought to be related to the production of free radicals. While a cause-and-effect relationship has yet to be proven with Dupuytren’s disease, the higher prevalence of the disorder seen in patients with characteristics such as alcohol consumption, cigarette smoking, and human immunodeficiency virus (HIV) is well established.

This association may be due to free radical development in such patient populations. With an increase in free radical formation, a cycle of myofibroblast proliferation leading to local ischemic events is created, further contributing to disease progression. More recent literature has studied the roles genetic predisposition, enzymatic pathways, and biochemical profiles play in Dupuytren’s disease development, progression, prognosis, and treatment. DNA microarrays have been used to evaluate the expression of genes in diseased tissue. In one such study on abnormal collagen expression in Dupuytren’s disease, an upregulation of collagen synthesis gene expression combined with simultaneous down regulation of collagenase gene expression explained the alteration of collagen types seen in Dupuytren’s disease.

Specific biomarkers have been identified in Dupuytren’s tissue and are now being used to provide laboratory values for prognosis and treatment. While not necessarily unique to Dupuytren’s disease, a disintegrin and metalloproteinase domain 12 (ADAM12), periostin (POSTN), and tenascin C (TNC) are all abnormally expressed in diseased tissue. In addition, ADAM12 and TNC are associated with the transforming growth factor beta (TGF-β) pathway, a pathway known to have direct and indirect effects on fibroblast/myofibroblast contraction and proliferation.

Myofibroblast contractility is also associated with and enhanced by sphingosine-1-phosphate (SIP) activation of non-muscle myosin II (NMII). Identification of the SIP pathway is being studied as a possible target for nonoperative treatment of Dupuytren’s disease.

Finally, chromosomes 6, 11, and 16 have been identified as being the loci for many, if not all, of the genes associated with Dupuytren’s, thus providing a genetic basis for evaluation and treatment.

**Anatomy**

An understanding of normal palmar fascial anatomy is necessary for proper evaluation and treatment of Dupuytren’s disease. The changes seen in diseased tissue are a result of progressive pathologic changes in normal anatomy. Nomenclature used for normal structures includes bands and ligaments, whereas diseased tissues are referred to as nodules and cords (Figure 1).

The palmar fascial complex as described by Rayan is composed of 5 fascial structures: the radial, ulnar, and central aponeuroses and the palmodigital and digital fascia. All are involved in Dupuytren’s disease to a varying degree. Of these, the triangular-shaped fascial layer known as the central or palmar aponeurosis is thought to be the center of Dupuytren’s disease activity. Classically, with disease progression, the more distally located palmodigital and digital fascia become increasingly involved, resulting in the metacarpophalangeal (MCP) joint and proximal interphalangeal (PIP) joint contraction seen clinically.

The palmar aponeurosis is composed of fibers oriented in longitudinal, trans-
The most superficial layer of each tendinous band is composed of 3 distinct central digits in the distal palm. Each prenous bands fan out and bifurcate to the 3 pretendinous bands. These pretendinous bands are part of discretely organized structures known as pretendinous bands. Vertical fibers, in conjunction with slips from the transverse fibers, constitute the septa of Legueu and Juvara. Seven compartments are formed by these septa that surround flexor tendons, lumbricals, and joint capsule with connections to the tendon sheath, periosteum, and subcutaneous tissue and skin. Those fibers attach to the tendon sheath, periosteum, and joint capsule with connections to the subcutaneous tissue and skin. Those fibers oriented volar to the neurovascular bundle are called Grayson’s ligaments and those passing dorsal are Cleland’s ligaments.12,16

PATHOANATOMY

Pathologic tissues in Dupuytren’s disease are grouped broadly into being either nodules or cords. Controversy exists as to which structure develops first or whether they develop simultaneously.

Early descriptions of the myofibroblast and its role in Dupuytren’s disease were made by Gabbiani et al17 and Tomasek et al.18 Newer data confirming the role of myofibroblasts in cord contraction have studied myofibroblast distribution within nodules and cords. Nodules were found to have an increased density of myofibroblasts that contributed to the contraction of diseased cords. In contrast, cords were found to be relatively hypocellular structures.

Contraction of myofibroblasts in diseased nodules results in deposition of an extracellular matrix that, through a process of remodeling, leads to shortening of the cord. In addition, myofibroblastic apoptosis during this process leads to relative hypocellularity of the cord and worsening of the flexion deformity.19

Cords can shorten in a progressive manner, leading to joint and soft tissue contracture. Also, cords develop from or along the same 3-dimensional pathways as normal tissue and are fairly predictable in their location and effect on surrounding soft tissues, joints, and neurovascular structures. Proper management acknowledges the fact that the orientation of diseased tissue is found not to be isolated to a longitudinal plane but involves a 3-dimensional space from the palm to the digit.

A thorough understanding of normal fascial anatomy of the palm and involvement of the neurovascular bundle is necessary for safe and effective operative treatment of Dupuytren’s disease. Terminology used when defining diseased vs normal tissue defines bands, sheaths, and ligaments as normal anatomy, whereas nodules and cords are reserved for diseased tissues (Table).

The pretendinous cord develops from the pretendinous band and palmar fibrofatty fascia. It is responsible for much of the MCP joint flexion seen in Dupuytren’s disease and is the most common cord seen clinically. However, it can also contribute...
to concomitant MCP and PIP joint contracture as it progresses distally as a central cord. Its insertion distally includes the skin overlying the proximal phalanx, the tendon sheath distal to the PIP joint, the periosteum at the middle phalangeal base, and the lateral digital sheet. Although intimately associated with the neurovascular bundle along its course, it does not cause bundle displacement as often as other cords. Occasional bifurcation of the pretendinous cord to separate digits can be seen as a commissural cord.

The spiral cord is a compilation of 4 fascial structures including the pretendinous band, spiral band, lateral digital sheet, and Grayson’s ligament (Figure 3).20,21 These structures coalesce in the digit and contract, causing predictable displacement of the neurovascular bundle proximal, central, and superficial to its in situ location.20,22,23 Early in the disease process, the cord will spiral around the neurovascular bundle following lines of normal fascial anatomy. However, with disease progression, the cord becomes taut and straight, causing the neurovascular bundle to spiral around the cord. The spiral cord can cause increasingly severe PIP joint contracture to the point that the likelihood of a spiral cord being present is increased if PIP contracture is seen clinically.

The natatory cord arises from the natatory ligament and is the only structure at the palmodigital junction not to be directly involved with the spiral cord. Progression of the natatory cord leads to contracture of the second through fourth web spaces. Passive abduction of the involved digits with subsequent flexion of 1 and extension of the other at the MCP joint causes the cord to become more prominent.24 The central cord is a distal extension of the pretendinous cord found in the palm and can cause contraction of the PIP joint. Involvement and contracture of the lateral digital sheet in the digit is termed a lateral cord. Its insertion to the skin or flexor tendon near Grayson’s ligament leads to contracture of the PIP joint. In addition, contracture of the distal interphalangeal (DIP) joint should raise awareness for the possibility of this cord being present. It may cause displacement of the neurovascular bundle depending on its mass within the digit. Of note, the small finger aspect of the small finger does not contain a lateral digital sheet as a discreet structure. However, the tendon of the abductor digiti minimi muscle can serve as a nidus for an abductor digiti minimi cord found in 25% of patients with a small finger contracture.20 This cord can displace the neurovascular bundle midline. Care must be taken when approaching the small finger to avoid damage to the dorsal ulnar sensory nerve that can be displaced from its normal anatomic position into one more susceptible to injury during dissection.20

A retrovascular cord involves tissue just dorsal to the neurovascular bundle, excluding Cleland’s ligament, which is normally spared in Dupuytren’s disease. This cord alone does not directly cause contracture of the PIP joint, but may be a cause of residual PIP contracture if not completely removed due to its effect on the surrounding soft tissues. In addition, it is the most common cause of DIP joint contracture.

Commissural cords are products of natatory and transverse ligament extensions near the first web space. If the thumb is involved, contraction of these cords can lead to difficulty with pinch and grasp and may require excision if severe disability is present.20,24

CLINICAL FEATURES

Dupuytren’s disease is a disorder of autosomal dominant inheritance with
variable penetrance. Multiple factors including age, sex, ethnicity, and geography play a role in Dupuytren’s prevalence. Specifically, the disorder is present in Scandinavia, Great Britain, Ireland, and Australia to a greater degree than in Africa, Greece, and Asia. It is speculated that the high predilection of the Vikings for the disease and their routes of travel through Europe may be related to such a geographic distribution.

Dupuytren’s disease is more common as one ages, with patients older 40 years being at increased risk.25 It is also more common in men than women, at a ratio of 9 to 1.26 While a cause-and-effect relationship has yet to be proven, diabetes mellitus, alcohol use, smoking, and HIV have all been associated with a higher risk of Dupuytren’s disease development.5,20

Presentation for clinical evaluation is many times delayed secondary to the relatively painless nature of Dupuytren’s disease progression. Patients are often referred by their primary care physician for evaluation of a trigger finger, tendon adhesions, or joint arthritis due to decreased finger range of motion (ROM) or contraction. Classically, the first clinical signs reported by the patient are skin pitting and thickening or nodule formation near the MCP joint on the palm of the hand. Patients may also present with more advanced disease secondary to difficulty with placement of the hand in a pocket or inability to flatten their fingers on a table.

Following skin pitting and thickening, early disease progression presents as nodules and cord formation in the palm. The ring and small fingers are affected first, and most often in that order. Following the ring and small fingers, the long finger, index finger, and thumb may also be affected, in that order.22,27 In women and patients with diabetes mellitus, initial presentation and progression may differ from that found in men. Isolated PIP or web space involvement in women and radial digit involvement in patients with diabetes mellitus may be misdiagnosed as something other than Dupuytren’s disease early in the process.20

As cord development progresses, significant findings in the palm and digits can become clinically impressive and disabling. A failed tabletop test, as described by Hueston,28 in which the patient cannot lay his or her palm and fingers simultaneously flat on a hard surface, helps in the screening process for Dupuytren’s disease. A positive test has been shown to correlate with alterations in hand function and MCP joint contracture of approximately 40°.28

In patients younger than 40 to 50 years at onset with early, aggressive cord formation and multiple physical findings including knuckle pads (aka Garrod’s nodes), plantar fibromatosis (Lederhose’s disease), and penile involvement (Peyronie’s disease), a diagnosis of Dupuytren’s diathesis should be considered. These patients are at higher risk for recurrence, poor operative outcomes, significant complications, and less satisfying rehabilitation. While operative treatment is not contraindicated in this population, such risks should be thoroughly discussed prior to treatment.

The risk of recurrence after treatment is controversial due in part to the lack of standardized definitions of recurrent disease. Documented from 8% to 54%, the risk of recurrence and difficulty in predicting outcomes should be discussed with every patient prior to operative and nonoperative intervention.20 While the risk of disease recurrence seems high at first, the risk of recurrence requiring further intervention is much lower.20 Whether recurrence is due to incomplete excision, new disease formation, or disease persistence and progression is also debatable. Younger patients and those with strong genetic tendencies (ie, diathesis) are at higher risk of recurrence. Finally, while the operative technique used has not been associated with higher recurrence risk,20 the use of full-thickness skin grafting to areas of excision has been postulated to decrease the rate of recurrence.4

**MANAGEMENT**

Operative intervention continues to be the gold standard for treatment of Dupuytren’s disease and is indicated in cases of advanced disease, contractures interfering with activities of daily living, and in cases where patient preference dictates operative intervention. Specifically, a positive tabletop test is correlated with MCP joint contractures of 30° to 40° and reflects the stage at which inability to use the hand in a normal fashion is present.20

Proximal interphalangeal joint contracture of ≥20° has traditionally been an indication for operative or invasive intervention. However, due to the difficulty of correcting severe PIP joint deformity, caution should be observed when suggesting operative intervention simply due to PIP joint contracture.

Numerous nonoperative modalities, including splinting, physical therapy, and corticosteroid injections, have not been found to be beneficial with regard to long-term outcomes. In addition, recent interest in medicinal treatment of Dupuytren’s disease,29 the role of matrix metalloproteinase in fibroblast contracture,30 and the use of the combination of an angiotensin converting enzyme (ACE) inhibitor/N-acetyl-L-cystein in TGF-β1 inhibition for Dupuytren’s disease prevention31 have added to an already long list of nonoperative treatment options for Dupuytren’s patients. However, due to the variable course of early Dupuytren’s disease progression, those patients with less severe clinical findings or symptoms should be encouraged to continue to use the affected hand in a normal fashion, as observation is the mainstay of treatment for early disease.

Options for operative intervention are numerous and include radical, regional, or limited fasciectomy, dermatofasciectomy, segmental fasciectomy, percutaneous needle fasciectomy, and fasciectomy using local collagenase injection.

Once the mainstay of treatment for Dupuytren’s disease, McIndie and Beare’s radical fasciectomy32 has fallen out of fa-
Dupuytren’s disease varies significantly due to the persistence of recurrence, the invasive nature of the procedure, and adequate outcomes using a less invasive approach.

More often, a selective or regional palmar fasciectomy is performed. The procedure requires less invasive dissection than a radical approach and removes only diseased tissue in the region affected by the disease process. Despite its invasive nature, open fasciectomy remains the gold standard for treatment of Dupuytren’s contracture. Skin incisions in open excision must be planned with care, as closure of contracted skin may prove difficult. Midline longitudinal incisions with z-plasty closure, Brunner zigzag incisions, V-Y flaps, and transverse incisions have all been described. Although many of these incisions can be closed primarily, wounds left open to heal by secondary intention, such as the open palm technique of McCash, are also well-accepted options. Initial splitting in a bulky dressing for the first 3 to 7 days postoperatively, followed by active and passive ROM exercises, is advocated. Nighttime splitting for up to 4 months is often necessary to maintain an extended posture of the affected digits during the rehabilitation period.

A recent review article discussing complications following open fasciectomy over 20 years reported an overall complication rate of 3.6% to 30.1%. On average, wound-healing complications were the most prevalent at a rate of 22.9%, followed by digital nerve injury (3.4%), infection (2.4%), and digital artery injury (2.0%). A higher rate of complications associated with revision surgery for recurrent disease was also reported.

Recurrence rates for open treatment of Dupuytren’s disease vary significantly due to the lack of standardized guidelines defining recurrence and the variable nature of follow-up. It must be noted that recurrence rates requiring revision or repeat operative intervention are lower than recurrence rates alone. A recurrence rate of 0% to 71% was reported in an outcomes review of operative treatment for primary Dupuytren’s disease. Full-thickness or “firebreak” flaps of tissue for coverage after open fasciectomy have been studied as a means to decrease recurrence rates. In a comparison between the open technique of McCash and a full-thickness skin graft, Skoff et al reported a 50% recurrence rate with the open palm technique vs 0% recurrence with the full-thickness graft. After 3 years, outcomes comparing dermofasciectomy with full-thickness skin graft and z-plasty over the PIP joint were reported in a prospective randomized study. No statistical significance between the 2 and a combined recurrence of 12.2% was reported.

Finally, concomitant fasciectomy and carpal tunnel release has traditionally been avoided due to an unacceptably high rate of complex regional pain syndrome (CRPS). More recent data, however, retrospectively indicated that the rate of CRPS may be much lower than originally thought and that simultaneous undertaking of 2 procedures may be advantageous.

Incisions for open procedures range from midaxial longitudinal incisions extending into the digit from the palm and Brunner zigzag incisions to V-Y advancement flap closure. A longitudinal incision in theory may put the centrally displaced neurovascular bundle at higher risk of damage. However, careful dissection coupled with thorough knowledge of the diseased anatomy makes this approach relatively safe. Closure of the incision is preformed using a z-plasty technique to provide more pliable tissue for closure in addition to allowing for lengthening of the incision.

The relative simplicity of planning, excellent exposure, and the option to leave a portion of the incision open to be closed with skin grafting if necessary has popularized the Brunner zigzag incision for the majority of operative cases.

With exposure, great care must be taken to create full-thickness flaps that respect not only the diseased tissue and endangered neurovascular bundle but also the necessity of closure and wound healing. Avoidance of skin and wound-edge necrosis through atraumatic technique, suturing of the flap edge to adjacent skin to avoid vigorous retraction, and closure void of any undue tension are requisite for a successful outcome.

Originally described by Russ in 1908, segmental fasciectomy involves multiple small incisions along the course of the diseased cord. Through these incisions, segments of diseased tissue are removed, thus disrupting the structure of the cord. Recurrence rates as low as 21% have been reported using this technique, but open operative fasciectomy remains the standard of care.

Less invasive procedures, such as percutaneous needle fasciotomy (PNF) and injectable collagenase, have been developed in an attempt to match outcomes similar to those seen with operative treatment but avoid the morbidity associated with open procedures.

Percutaneous needle fasciotomy has gained popularity as being the least invasive of operative treatment options, making it a favorable choice for those patients not amenable to more invasive procedures. Performed under local anesthesia, PNF involves injection of anesthetic in an intradermal manner surrounding the cords to be released. Using the technique similar to that originally described by Sir Astley Cooper and modified by Eaton, a 25-gauge needle is used as a scalpel, sweeping in a path transverse to the cord from superficial to deep, causing transection of the cord. Constant tension on the cord during the procedure aids in avoiding injury to underlying neurovascular bundle and flexor tendon. Short-term results using PNF have been favorable, with contractures of the MCP joints showing the most improvement. Comparison of PNF with limited open fasciectomy was performed in 2006. Short-term results indicated a 63% improvement in total
passive extension deficit with PNF and 79% improvement with the limited open procedure. Whereas minor complications favored the open fasciectomy group, no major complications were reported with the PNF group, compared with 3 seen in the open group.45

Food and Drug Administration approval of injectable collagenase clostridium histolyticum as treatment for Dupuytren’s disease introduced yet another less invasive approach for management of advanced Dupuytren’s disease. Biochemical studies as early as 1987 suggested a possible role for a collagenase in the treatment of Dupuytren’s disease via the dissolution of collagen. Clinical studies by Hurst, Badalamente, and others through 2009 further replicated and refined the role of an injectable clostridium in the treatment of Dupuytren’s disease.46-48 Proponents of the injection cite the less invasive nature of the procedure, avoidance of extensive hand therapy after treatment, lack of significant adverse events, and promising short-term (8-year) outcomes as being strengths for the injection, especially with regard to treatment of MCP joint contracture.48-50 However, criticism of the clostridium injection relates a higher rate of minor adverse events, patient-reported apprehension for the procedure, lack of long-term outcome studies, and a current cost of $5400 per injection as being the downside to the procedure.49,51

Proximal interphalangeal joint involvement introduces an additional confounding factor in the treatment of Dupuytren’s disease. Controversy exists with regard to the degree at which PIP joint contracture should be corrected. Methods of correction include simple excision of diseased Dupuytren’s tissue, excision with extensive capsular release, and release of soft tissues surrounding the PIP joint. McFarlane et al reported poorer outcomes with operative intervention for PIP joint contractures of <30° and suggested release only when PIP joint contracture exceeded 30°. In addition, a dynamic PIP joint contracture must be considered when associated MCP flexion contracture is seen in the same digit. Due to the pathoanatomy of advanced cords in the digits, a well-developed spiral cord may cause contracture of both the MCP and PIP joints, and release of the MCP contracture may resolve deformity seen at the PIP joint. Postoperative maintenance of PIP correction is unpredictable and involves long-term extension splinting. Rives et al reported only 44% improvement in PIP joint extension for contractures of ≥45° undergoing additional PIP soft tissue release during Dupuytren’s fasciectomy.52 Strict adherence to the postoperative splinting protocol was predictive of outcome in their study. A compliant patient population is therefore necessary for successful treatment. Based on the idea that MCP joint compensation can overcome significant PIP joint contracture, Hueston suggested intervention for PIP contracture only if >40°.53 Due to the complexity in treating PIP joint contracture, a discussion must be had with the patient regarding the possibility that any PIP joint correction attained intraoperatively may not be long-standing. Also, by attempting to correct severe contracture at the PIP joint, the risk of complication during the procedure is elevated, especially if significant soft tissue dissection is necessary.

**Postoperative Care**

Postoperative care following clostridium injection is similar to that of an open technique, but allows for earlier return to use of the affected hand due to the less invasive nature of the procedure. After injection, a guided finger extension procedure is performed up to 3 times per day to facilitate cord disruption.48,50 Following complete cord release, an extension splint is worn for up to 4 months. A home therapy program consisting of active ROM and return to normal activities as tolerated is implemented immediately, with formal therapy introduced on an individual basis.49

**Complications**

The possibility of numerous complications exists for operative treatment of Dupuytren’s disease. Causes of such complications can be related to intraoperative or technical issues, patient anatomy and pathophysiology, and complications related to postoperative or rehabilitative care.

Altered anatomy found in Dupuytren’s disease, lack of experience with diseased tissue, and degree of contracture severity all play a role with regard to possible damage to neurovascular structures during contracture correction and tissue excision. The rate of digital nerve damage in
patients with primary disease undergoing fasciectomy has been quoted at an average of 3.4%. It is suggested that dissection in a proximal to distal direction from known to unknown beginning in the palm be the standard by which diseased fascia is excised. Meticulous care to identify neurologic structures in the field prior to further dissection and excision is paramount in avoiding nerve damage. If a nerve is acutely transected, primary repair should be performed.

Vascular insult can be related to intraoperative laceration or crush injury, arterial spasm, and/or intimal hemorrhage. Acute digital artery injury has been quoted at an average of 2.0% for primary disease. Acute transection requires immediate primary repair. Spasm and/or intimal hemorrhage are evaluated by first manipulating the joint in question, followed by irrigation with warm saline to encourage return of flow. Evaluation of digits and flaps in the proximity of a damaged vessel should be performed prior to dressing application.

Hematoma formation postoperatively can be a source of pain, delayed postoperative therapy due to stiffness, and a nidus for infection and skin necrosis. Care should be taken to evaluate the operative field for any bleeding vessels and provide hemostasis with the tourniquet deflated prior to wound closure.

Numerous flaps are often created during excision of diseased tissue. These flaps are often thin and subject to healing complications. Care to design flaps with adequate blood supply is required to avoid necrosis. The management of skin pitting and severe flexion contractures also poses a risk for skin breakdown or loss of adequate tissue for coverage. Small defects can be left open to heal by secondary intention. Full-thickness skin grafting should be used to cover areas of significant loss such as is often found over long-standing MCP and PIP joint contractures.

Reflex sympathetic dystrophy, or CRPS, deserves special attention. Defined by hyperemia, dysthesias, swelling, and pain out of proportion to what would be expected in a normal postoperative course, its risk is increased in women and patients undergoing simultaneous fasciectomy and carpal tunnel release. Careful dissection with specific attention paid to avoid overzealous retraction of neural structures may decrease the risk of development. If no other identifiable cause can be identified for such symptoms, treatment consists of pain control modalities that may include evaluation by a pain management specialist. Selective sympathetic nerve block may also prove useful in managing this perplexing outcome.

Postoperative therapy can be delayed by the aforementioned complications and can contribute to poor objective and subjective outcomes. Ability and willingness of the patient to comply with therapy plays a substantial role in the final result of operative care. Residual contracture, scar formation, stiffness, and the need for further intervention can be related not only to intraoperative technique and findings but also to postoperative care. In addition, a more severe preoperative contracture, disease affecting the little finger and/or multiple fingers, and PIP joint involvement increase the risk for residual or worsening postoperative contracture.

CONCLUSION

Dupuytren’s disease is a contractile disorder of the palmar fascia in the hand affecting cells of fibroblast origin. While the etiology remains unclear, it is most commonly seen in aging men of European descent and is inherited in an autosomal dominant fashion with variable penetrance. The current gold standard for treatment of severe disease is open limited fasciectomy followed by an aggressive course of therapy encouraging active ROM. Operative intervention is indicated with MCP joint contracture of at least 30° and/or a progressive PIP contracture of at least 20° to 30°. Due to residual postoperative contracture, controversy exists as to the minimum PIP joint contracture necessary for operative correction. The Brunner zigzag incision provides excellent exposure with adequate healing in the majority of cases. Injectable clostridium histolyticum collagenase offers a new and less invasive technique for patients with Dupuytren’s disease, with immediate return to function of the affected hand. Recurrence rates remain controversial independent of the technique used, and, as such, patient expectations should be evaluated and discussed prior to operative intervention, especially in those patients with Dupuytren’s diathesis.

REFERENCES
