Hand Infections: Treatment Recommendations for Specific Types

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Abstract

Hand infections can be associated with considerable morbidity. Expeditious treatment is needed to minimize permanent dysfunction, loss of work, and medical cost. Hand infections can affect the skin, subcutaneous tissues, fascia, subfascial and synovial spaces, joints, and bone. Pathogens include a variety of bacteria, viruses, yeasts, fungi, and mycoplasmata. Management frequently involves rest, elevation, incision and drainage, and appropriate antibiotic therapy. The orthopaedic surgeon must be knowledgeable about the pertinent anatomy and how this influences the behavior of specific types of infections, the role of immunocompromise, and the importance of early mobilization.

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Expeditious and appropriate treatment can minimize the morbidity associated with infections of the hand. The typical infections that occur in the hand include subcutaneous abscesses, septic joints, and cellulitis. The complex anatomy of the hand also makes it susceptible to unique infections. Optimal care requires the clinician to understand this anatomy and how it influences the behavior of certain infections, so that they can be recognized and treated appropriately. Management usually necessitates adequate incision and drainage, appropriate antibiotic therapy, elevation and edema control, and mobilization. The purpose of this review is to discuss the general principles of treatment and to delineate more specifically the management of infections typically unique to the hand.

Microbiology

The most common bacteria involved in infections of the hand are *Staphylococcus aureus, Streptococcus,* and Gram-negative species. *Staphylococcus* is the principal organism in 50% to 80% of infections.[1] Industrial and home-acquired injuries usually involve a single Gram-positive organism, while infections due to intravenous (IV) drug use, bite wounds, and mutilating farm injuries and those associated with diabetes mellitus often are polymicrobial, involving Gram-positive, Gram-negative, and anaerobic species.[1-7] α-Hemolytic *Streptococcus* and *S aureus* are the most commonly isolated pathogens in human-bite infections.[1-4] *Eikenella corrodens* is isolated in approximately one third of human-bite wounds.[1,3,4] *Pasteurella multocida* commonly infects animal-bite and scratch wounds.[8] Chronic indolent infections may suggest fungi or atypical mycobacteria.

Knowledge of the likely pathogens is based on the history, nature, and course of infection and can direct culture and staining requests. Routine aerobic and anaerobic cultures and Gram stain should be done. If atypical mycobacteria are suspected, Ziehl-Neelsen staining and cultures at 28˚C to 32˚C in Löwenstein-Jensen medium should be performed.[9] Examination for fungi is accomplished with a potassium hydroxide preparation.[10] A Tzanck smear can be useful in the diagnosis of herpes simplex virus infection by demonstrating typical giant cells.[11]

Antibiotic Therapy

Table 1 gives some recommendations for initial antibiotic therapy based on the type of infection. Antibiotics should be administered empirically after performing cultures

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Table 1
Empiric Antibiotic Recommendations for Hand Infections*

Infection	Possible Organisms	Antibiotic Dosage	Alternative Antibiotic Dosage
Felon / paronychia	*S aureus*, oral anaerobes	Dicloxacillin, 250-500 mg PO q6h, *or* Nafcillin, 1-2 g IV q4-6h, *or* Clindamycin, 150-300 mg PO q6h	Cephalexin, 250-500 mg PO q6h, *or* Cefazolin, 1 g IV q8h, *or* Erythromycin, 250-500 mg PO q6h
Flexor tenosynovitis	*S aureus*, *Streptococcus*, Gram-negative rods	Cefazolin, 1 g IV q8h	Nafcillin, 1-2 g IV q4-6h, or vancomycin, 1 g IV q12h, plus gentamicin,† *or* Imipenem, 0.5-1.0 g IV q6h
Herpetic whitlow	Herpes simplex virus	Consider acyclovir, 400 mg PO tid x10 days	
Subcutaneous or deep-space abscess	*S aureus*, anaerobes, Gram-negative rods	Cefazolin, 1 g IV q8h, *or* Ampicillin-sulbactam, 1.5 mg IV q6h	Nafcillin, 1-2 g IV q4-6h, plus gentamicin,† *or* Imipenem, 0.5-1.0 g IV q6h
Cellulitis / lymphangitis	*Streptococcus*, *S aureus*	Dicloxacillin, 250-500 mg PO q6h, *or* Nafcillin, 1-2 g IV q4-6h, *or* Cephalexin, 1 g IV q8h	Cephalexin, 250-500 mg PO q6h, *or* Erythromycin, 250-500 mg PO q6h
IV-drug-abuse–related	Gram-positive, Gram-negative, or mixed[6] or methicillin-resistant *S aureus*[1]	Nafcillin, 1-2 g IV q4-6h, plus gentamicin†	Vancomycin, 1 g IV q12h (for methicillin-resistant *S aureus*), plus gentamicin,† *or* Imipenem, 0.5-1.0 g IV q6h
Human bite	*S aureus*, *E corrodens*, *Streptococcus*, anaerobes	Cefazolin, 1 g IV q8h, plus penicillin, 2-4 million U IV q4-6h, *or* Clindamycin, 300 mg PO q6h, plus ciprofloxacin, 250-500 mg PO q12h, or trimethoprim-sulfamethoxazole (Septra DS) PO q12h	Nafcillin, 1-2 g, plus penicillin, 2-4 million U q4-6h, *or* Amoxicillin-clavulanate potassium, 250-500 mg PO q8h, *or* Ampicillin-sulbactam, 1.5 g IV q6h
Animal bite‡	Gram-positive cocci, anaerobes, *P multocida*	Cefazolin, 1 g IV q8h, plus penicillin, 2-4 million U IV q4-6h	Nafcillin, 1-2 g, plus penicillin, 2-4 million U q4-6h, *or* Amoxicillin-clavulanate potassium, 250-500 mg PO q8h, *or* Ampicillin-sulbactam, 1.5 g IV q6h
Diabetes-related	Gram-positive cocci, Gram-negative rods[5]	Cefazolin, 1 g IV q8h, plus gentamicin†	Cefoxitin, 2 g IV q6h, *or* Ampicillin-sulbactam, 1.5 g IV q6h
Osteomyelitis	*S aureus*, *Streptococcus*, (rarely) Gram-negative rods	Cefazolin, 1 g IV q8h, plus gentamicin†	Nafcillin, 2 g IV q4h, *or* Vancomycin, 1 g IV q12h, *or* Clindamycin, 600-900 mg IV q8h, *or* Doxycycline, 100 mg PO q12h
Septic arthritis	*S aureus*, *Streptococcus* (*Neisseria gonorrhoeae*)	Cefazolin, 1 g IV q8h, *or* Ceftriaxone, 1 g IV q24h (for *N gonorrhoeae*)	Nafcillin, 2 g IV q4h, *or* Vancomycin, 1 g IV q12h, *or* Clindamycin, 600-900 mg IV q8h, *or* Doxycycline, 100 mg PO q12h
Traumatic / contaminated wound§	*S aureus*, *Streptococcus*, anaerobes, Gram-negative rods	Imipenem, 0.5-1.0 g IV q6h	Cefazolin, 1 g IV q8h plus gentamicin†

* Abbreviations: PO, orally; q6h, every 6 hours; tid, three times a day.
† Loading dose, 2 mg / kg of body weight; then follow serum levels.
‡ Consider rabies prophylaxis.
§ Consider tetanus prophylaxis.

and Gram stain. In general, initial antibiotic therapy should be aimed only at suspected organisms, since excessive coverage can select for resistant organisms, is costly, and unnecessarily exposes patients to side effects. Some hand surgeons feel empiric IV antibiotic coverage of aerobic and anaerobic organisms is necessary (e.g., cefazolin and penicillin G) for infections serious enough to require incision and drainage and hospitalization.[12] If IV drug abuse is involved or if the patient is diabetic, Gram-negative coverage is added (gentamicin) with the awareness that the adverse effects of aminoglycosides include nephrotoxicity and ototoxicity.[5,12,13]

In instances of methicillin-resistant *S aureus* infection, vancomycin is the drug of choice. Even when in vitro sensitivity tests suggest sensitivity to cephalosporin, the organisms are still resistant; thus, cephalosporins have no role in their treatment.[14] Only if there is a high suspicion of methicillin resistance (e.g., recurrent infection and a history of previous methicillin-resistant *S aureus* infection) should vancomycin be used empirically, since it is not as effective as the penicillinase-resistant penicillins (e.g., nafcillin) or the cephalosporins in this setting. Occasionally, methicillin-resistant *S aureus* is sensitive to trimethoprim-sulfamethoxazole, rifampin, and ciprofloxacin.

Patient Factors

Immunosuppressive therapy, acquired immunodeficiency syndrome (AIDS), and diabetes mellitus can predispose patients to hand infections with typical and opportunistic pathogens. In series of hand infections in diabetic patients,[5,13,15] treatment was often delayed, resolution was slowed, repeated debridements often were necessary, Gram-negative organisms were common, and amputations were performed frequently (rates of 20% to 63%), either to control infection or because of poor function. In diabetic patients with renal transplants, morbidity was particularly high.[15]

In one series of hand infections in AIDS patients, the infections were not opportunistic, but most had atypical presentations and unusually virulent courses. Herpetic lesions were also more virulent and did not resolve unless treated with antiviral agents.[16]

Intravenous-drug abusers commonly present with dorsal hand abscesses, usually due to *S aureus*, which has a propensity for methicillin resistance (although this is not the case at our institution [personal communication, Leland Rickman, MD, 1995]).[1] Infections in IV-drug abusers and in diabetic patients have a high incidence of Gram-negative organisms.[1,6,12,15] Risk factors for necrotizing fasciitis include IV drug or alcohol abuse and diabetes mellitus.[17]

Incision and Wound Management

Successful management of hand infections predates the advent of antibiotic therapy. The mainstay of treatment of deep hand infections is adequate incision and drainage. In general, straight incisions are preferred, as they cause less flap necrosis. Contaminated wounds should be left open for later closure or allowed to heal by secondary intention. Certain special situations are amenable to wound closure over irrigation drainage.

After initial incision and drainage, wounds are dressed with moist gauze and a bulky splint, usually immobilizing the digits in the intrinsic-plus posture, maintaining the first web space. The wound is examined at 24 to 48 hours, and daily wet-to-dry dressing changes are instituted, incorporating whirlpools or soaks to encourage continued drainage. Hand therapy is begun as early as possible, after initial inflammation and swelling have begun to resolve but usually before the wound has healed.

Specific Types of Infection

Cellulitis

Cellulitis is a spreading, diffuse inflammation characterized by hyperemia, leukocytic infiltration, and edema (by definition, without abscess formation, although it may extend peripherally from an area of suppuration) and may be accompanied by acute lymphangitis. Although often initiated by skin trauma, ulceration, dermatitis, or lymphedema, in many instances no predisposing condition exists. Cellulitis is most evident in the skin or subcutaneous tissues but can involve deeper structures.

Group A β-hemolytic *Streptococcus* is usually the causative organism. Occasionally, *S aureus* causes less extensive cellulitis.[18]

The diagnosis of cellulitis is based primarily on clinical findings. A subcutaneous abscess or septic joint or deep-space infection may lie beneath an apparent area of cellulitis, or there may be concern that apparent cellulitis actually represents necrotizing fasciitis. Examination should concentrate on ruling out these entities before initiating medical treatment. We have found no clinical usefulness in aspirating the leading edge of the area of cellulitis to identify an organism.

An attempt to treat early cellulitis with oral antibiotics is reasonable. If resolution is not apparent within 24 to 48 hours, however, IV antibiotic treatment is indicated. Oral agents of choice are penicillin V and

cephalexin. If the causative organism is not known, coverage for both *Staphylococcus* (including penicillinase-producing organisms) and *Streptococcus* is necessary, with cephalexin being the most appropriate single drug. For patients with severe penicillin allergies, erythromycin is an alternative.

Reassessment, suspecting an underlying infection (such as a subcutaneous abscess, deep-space infection, or septic joint), is indicated if there is no improvement despite appropriate treatment. For extensive cellulitis, IV antibiotic therapy is indicated. Intravenous penicillin G and cefazolin are the medications of choice, with cefazolin being preferred if the organism is unknown. If the patient is severely allergic to penicillin, vancomycin is an alternative.[18] We prefer to observe patients in the hospital while they are receiving IV antibiotic therapy, at least until early resolution. After discharge, IV antibiotics can be continued in the home, if necessary. Oral agents can be used if the causative organisms are sensitive.

Subcutaneous Abscess

A subcutaneous abscess usually results from a puncture wound that inoculates the subcutaneous layer. The most common organism is *S aureus*. Physical examination frequently demonstrates surrounding edema, cellulitis, and a central area of fluctuation. A specimen for culture and Gram stain can be obtained by aspiration through a large-bore needle. Expeditious incision and drainage before instituting antibiotic therapy can provide the necessary specimen while simultaneously achieving definitive treatment.

Postoperatively, the wound is left open with a moist bulky dressing and subsequently managed as previously discussed. Initial empiric antibiotic treatment should be with a first-generation cephalosporin. If the infection originated from a farmyard or similar injury, penicillin should be added for anaerobic coverage. If the patient is diabetic or the abscess originated from IV drug abuse, Gram-negative coverage (e.g., with gentamicin) is included.

Paronychia

Acute paronychia refers to an infection beneath the eponychial fold[19] (Fig. 1). Disruption of the seal between the nail plate and the nail fold allows entry of bacteria into the eponychial space. The abscess that forms can track around beneath the entire nail fold superficial to the nail plate (runaround abscess), or it can track proximally around and deep to the nail plate between the nail and the matrix.[19] The infection is often seen after manicures, in persons with artificial nails, in nail biters, and in those with hangnails. Physical examination demonstrates swelling, erythema, and tenderness about the nail fold. The offending organism is usually *S aureus.*

Although early infections can sometimes be successfully treated with oral antibiotics, the safest approach is to provide drainage. After a metacarpal block and sterile preparation, a No. 15 scalpel, Freer elevator, or fine hemostat can be used to lift the nail fold off the nail plate to allow egress of the abscess. The nail fold is held open with a small piece of gauze. If the abscess has spread proximally underneath the nail plate, either a longitudinal portion or all of the nail is removed, depending on the extent of the spread. A Freer elevator or similar instrument is placed between the nail plate and the matrix (taking care not to damage the latter) on the involved side. The nail can then be cut longitudinally with scissors, and a portion of the nail can be removed, thereby decompressing the nail fold volar and dorsal to the nail plate. If necessary, the entire nail can be removed. A small piece of gauze can maintain drainage. An eponychial fold incision should be avoided, if possible, because an unsightly deformity of the nail fold can result. Within 24 to 48 hours, the dressing should be removed with the aid of a warm soak. The dressing should be changed daily thereafter, again with the use of soaks.

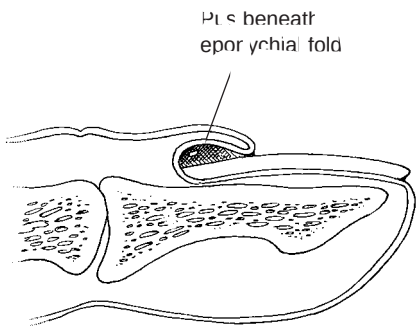

Fig. 1 Sagittal section of the digital tip demonstrates the space beneath the eponychial fold under which an abscess (paronychia) can develop.

Chronic paronychia is characterized by intermittent periods of inflammation about the eponychium. It is usually seen in patients exposed to constant moisture. Eventually there is separation of the nail fold from the underlying nail plate. There may be a cheeselike drainage from beneath the eponychium. Fungi (particularly, *Candida albicans*), atypical mycobacteria, and Gram-negative bacteria have all been implicated.[1,19] Treatment can be frustrating, with the most successful interventions being marsupialization and total nail removal.[19] Application of a topical corticosteroid-antifungal ointment (3% clioquinol in a triamcinolone-nystatin mixture [Mycolog]) has been recommended as an adjunct.[19]

Felon

The digital pad is a closed, poorly compliant compartment containing

a latticework of fascial septa extending from the distal phalanx to the skin.[19] Distal phalangeal vascularity can be compromised by distention within this compartment.[19] Felons are closed-space infections of the digital pulp. Symptoms include intense throbbing pain and swelling of the entire pulp, usually after penetrating trauma. The most common organism is *S aureus*.[19]

In the cellulitic stage, elevation, soaks, and antibiotics are often adequate treatment. Once abscess formation has occurred, drainage is necessary. If inadequately treated, necrosis of the digital pad, osteomyelitis of the distal phalanx, and extension to the flexor sheath can occur.

Incisions for drainage of felons have been controversial. Fishmouth incisions have been condemned because of resultant vascular compromise of the digital pad.[19] Midvolar and high lateral incisions are preferred, with the point of maximal tenderness guiding placement[19] (Fig. 2). Proximal probing should be avoided because of the risk of inoculation of the flexor sheath.

Postoperatively, loose-gauze packing facilitates drainage. Removal of the dressing and reexamination are recommended in 24 to 48 hours. Empiric *Staphylococcus* coverage should be provided until definitive cultures are available to guide treatment. Warm soaks and dressing changes will facilitate further drainage. The wound should then be allowed to close by secondary intention.

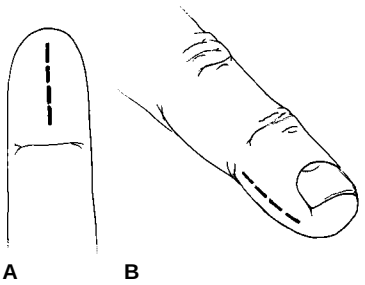

Fig. 2 Recommended incisions for drainage of felons. **A,** Midvolar. **B,** High lateral.

Herpetic Whitlow

The term "whitlow" (synonymous with felon) has continued to be used when describing herpesvirus hand infections. In fact, these infections are not felons, although they are often clinically confused with them and with paronychias. Most occur on the digital tip, but infections of the more proximal hand and forearm have been reported.[11]

The causative agents are the clinically indistinguishable herpes simplex virus types 1 and 2. Infection is an occupational hazard of those exposed to orotracheal secretions, but can also occur through autoinoculation from genital or oral lesions. A painful cytolytic infection occurs 2 to 14 days after exposure. It matures over the subsequent 14 days, with formation of vesicles that coalesce, unroof, and form characteristic ulcers. Patients may demonstrate concomitant lymphadenitis, fever, and malaise.

The disease is self-limited, resolving over a period of 7 to 10 days. Viral shedding, and thus the ability to infect other hosts, persists until lesion epithelialization is complete.[11] As the infection subsides, the virus avoids immune clearance by achieving a latent state and retreating to nervous ganglia.[11] Recurrences can be induced by psychological or physical stress, other infections, fever, sun exposure, and other precipitants. Patients sometimes perceive a prodrome of pain, tingling, or numbness. Diagnosis is usually made on the basis of the history and physical examination findings, but can be confirmed with cultures of the vesicular fluid (requires 1 to 5 days), Tzanck smear (less sensitive than cultures), or a rise in serum antibody titers.

The cornerstone of treatment is to not mistake the infection for a paronychia or felon. Unnecessary deep incision and drainage can lead to superinfection. To treat excessively painful early lesions, some have recommended superficial unroofing of the vesicles, which has been followed by some diminution of the pain (but no change in the natural history of the infection).[19] Treatment with acyclovir has been recommended to prevent recurrences, abort recurrent infections in patients who have a prodrome, decrease the clinical course in particularly protracted cases, and induce remission in patients with AIDS.[11]

Flexor Tenosynovitis

The flexor sheath is a mesothelial investing structure with a closed space between the visceral layer adherent to the flexor tendon and the parietal layer. The sheath extends from the proximal end of the A1 pulley to the level of the distal interphalangeal joint. The sheath of the thumb is contiguous with the radial bursa, and the sheath for the small finger is contiguous with the ulnar bursa. The radial and ulna bursae extend proximally to the carpal tunnel. In 50% to 80% of persons, the radial and ulnar bursae communicate[1,20] (Fig. 3).

Pyogenic flexor tenosynovitis is a bacterial infection of the flexor sheath. It usually results from penetrating trauma, although hematogenous spread is usual for gonococcal infections. *Staphylococcus aureus* is the most common causative organism. Streptococcus and Gram-negative organisms are frequently involved. In rare cases, atypical mycobacteria are responsible for a chronic indolent infection characterized by abundant tenosynovitis.[9,20]

Kanavel described four signs characteristic of pyogenic flexor tenosynovitis: (1) flexed resting position of the involved digit, (2) ten-

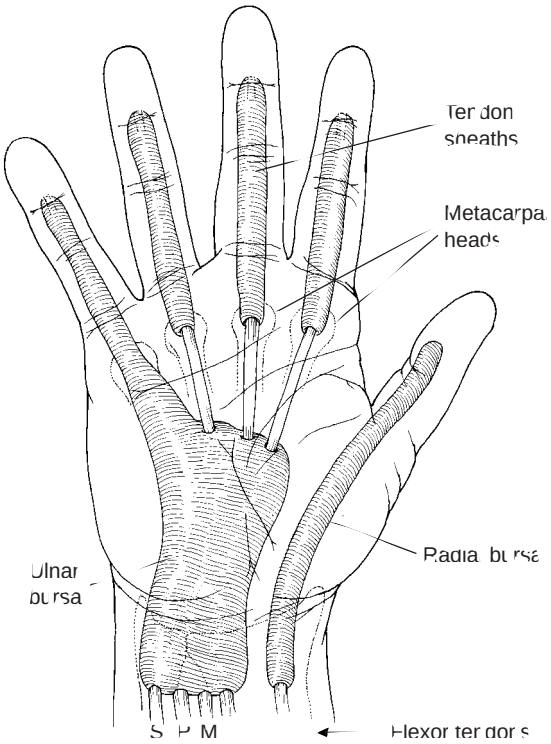

Fig. 3 Anatomy of flexor sheath and radial and ulnar bursae. Note that the radial bursa is contiguous with the flexor pollicis tendon sheath, and the ulnar bursa is contiguous with the small-finger flexor sheath. In 50% to 80% of persons, the ulnar and radial bursae communicate. S = small finger; R = ring finger; M = middle finger; I = index finger; T = thumb.

derness over the flexor sheath, (3) fusiform swelling, and (4) severe pain on passive extension.[1,21] All signs may not be present, especially early in the course. Treatment delay can result in tendon vascular compromise and necrosis, eventuating in adhesions and poor gliding.

Spread to contiguous deep spaces can also occur. Infection of the thumb-flexor sheath can, via contiguous spread through communication of the radial and ulnar bursae, result in infection of the small-finger sheath, and vice versa ("horseshoe abscess"). More extensive proximal spread can lead to infection of Parona's space (the quadrilateral potential space at the wrist bordered by the pronator quadratus, digital flexors, pollicis longus, and flexor ulnaris), sometimes resulting in acute median-nerve compression.[20] Contiguous spread from the index-finger flexor sheath can cause infection of the thenar space.

Early infections (within 24 hours) can be treated with elevation, splinting, and IV antibiotics.[1] If improvement is not noted in the first 24 hours, surgical treatment is necessary. Limited incisions and catheter irrigation have become popular, allowing more rapid rehabilitation and recovery. A proximal incision is made just proximal to the A1 pulley, near the distal palmar crease. A midlateral or volar transverse distal counterincision is made in the distal interphalangeal joint flexion crease to allow through-and-through irrigation.[1,20,22] A 5F pediatric feeding tube or an angiocatheter is placed into the flexor sheath, usually in the proximal incision, and sutured in place. Care must be taken to ensure that the catheter is within the sheath so that adequate irrigation of the infection can occur; otherwise, there is the potential for a compartment syndrome in the digit due to the presence of irrigating fluid in the interstitial tissue.

The sheath must be copiously irrigated. The hand is placed in a splint with absorptive padding in the palm. We prefer a simple closed system for irrigation that is easily understood by the floor nurses and minimizes contamination. One end of a piece of IV extension tubing is connected to the flexor sheath catheter. The other end is attached to a port of a three-way stopcock. A 5-mL syringe is placed in the second port of the stopcock. An IV tube connected to a bag of normal saline is attached to the third port of the stopcock. Three times a day, 10 to 20 mL is drawn from the IV bag into the syringe by using the stopcock. The stopcock is then directed toward the patient, and the fluid is injected through the extension tubing into the flexor sheath. After 48 hours, the wet dressing and the irrigation catheter are removed, and digital motion is started. Daily warm soaks and wet-gauze dressing changes are continued until the wound has healed by secondary intention. Physical therapy and use of a whirlpool are often beneficial early in the mobilization process.

If, at the time of initial incision and drainage, the purulent material in the sheath is too viscous to be evacuated by irrigation, open drainage is necessary. It is imperative to perform this with the use of a midlateral incision instead of a Brunner-type incision. The latter has a propensity for flap-tip necrosis, eventuating in exposed flexor tendons. Once adequately debrided, the midlateral incision can be loosely tacked closed over the irrigation drainage system.

Deep-Space Infections

The deep spaces of the hand include the dorsal subaponeurotic, thenar, midpalmar, Parona's quadrilateral, and interdigital subfascial web spaces. Deep-space infections usually occur via penetrating inocu-

lation or contiguous spread; occasionally, hematogenous seeding is responsible. *Streptococcus*, *S aureus*, and coliform organisms are the most common infectious agents.[1]

The dorsal subaponeurotic space is contained by the extensor tendons and fascia dorsally and the metacarpals and interosseous muscles palmarly. Infection of this space is accompanied by considerable dorsal hand swelling and may be difficult to distinguish from a subcutaneous abscess. A preferred method of incision and drainage is with two longitudinal incisions (over the second metacarpal and over the interspace between the fourth and fifth metacarpals), followed by local wound care and early motion.[23] Incisions directly over the digital extensor tendons should be avoided, so as to prevent compromise of their coverage.

The thenar space is bordered dorsally by the adductor pollicis, palmarly by the index-finger flexor tendon, and ulnarly by the midpalmar septum (which extends from the middle-finger metacarpal to the palmar fascia).[23] Radially, the thenar space is contained by the adductor pollicis insertion into the proximal phalanx and the thenar muscle fascia (Fig. 4). Infection of this space usually results from penetrating trauma or from contiguous spread from index-finger flexor tenosynovitis. Infection can spread around the distal edge of the adductor pollicis and first dorsal interosseous muscles to involve the dorsal first web, or it can invade the interval between the adductor pollicis and first dorsal interosseous, causing a "pantaloon" effect.[23] Examination reveals considerable thenar and first-web swelling. Since the volume of the thenar space is greatest with the thumb palmarly abducted, this is the usual resting posture. Pain is exacerbated by passive adduction or opposition.

Dorsal, palmar, and combined incisions have been described for surgical treatment[22] (Fig. 5). Many surgeons prefer the combined dorsal-palmar two-incision approach to ensure adequate drainage.[1,20,22] Another reasonable incision is one that perpendicularly traverses the first-web commissure and extends dorsally and palmarly.[23] Incisions that parallel the first-web commissure are discouraged to avoid web contracture. The palmar cutaneous branch of the median nerve, the digital nerves and arterial branches to the thumb and index finger, and the dorsal ulnar digital nerve to the thumb must be considered when making these incisions.

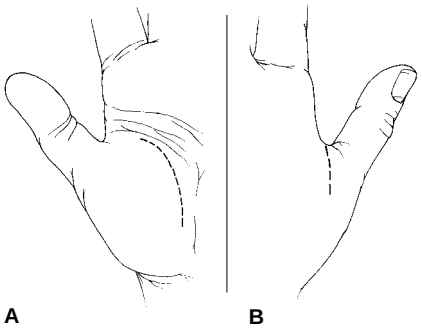

Fig. 5 Recommended incisions for drainage of thenar-space infections. Palmar **(A)** and dorsal **(B)** incisions are preferred by many surgeons to ensure adequate decompression of purulence, which can track dorsally from the thenar space to the interval between the adductor pollicis and the first dorsal interosseous muscles or to the subcutaneous area dorsal to the first dorsal interosseous muscle (pantaloon effect).

With adequate debridement, in the absence of necrotic tissue, wounds can be loosely approximated over a catheter with a similar bulky absorptive dressing and irrigation setup as described for flexor tenosynovitis.[1,22] Alternatively, continuous irrigation can be used.[22] If there is any doubt regarding the adequacy of the drainage or tissue viability, the wound should be left open and dressed with loose wet-gauze packing, which must be changed daily.

The deep palmar space is bordered dorsally by the middle- and ring-finger metacarpals and the second and third palmar interosseous muscles, palmarly by the flexor tendons and lumbricals, radially by the midpalmar septum, and ulnarly by the hypothenar muscles (Fig. 4). Direct penetration or, in rare cases, contiguous spread from the flexor sheaths of the middle and ring fingers may cause infection.[22,23] On presentation, the palm is markedly tender. The normal concavity of the palm is lost, so that it instead appears flattened or convex. Dorsal

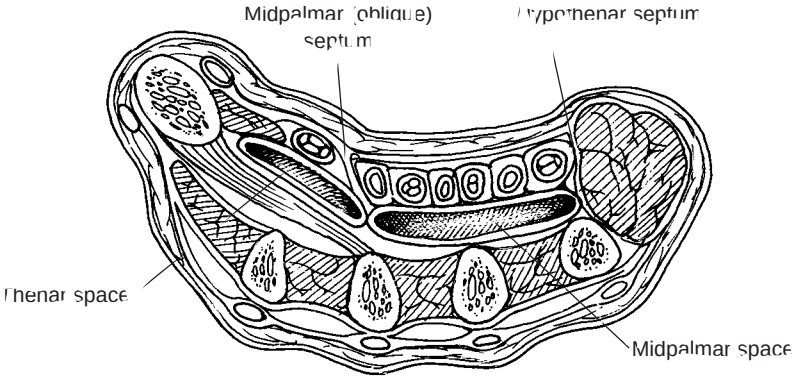

Fig. 4 Cross section through the hand depicts the thenar and deep palmar spaces. (Adapted with permission from Neviaser RJ: Infections, in Green DP [ed]: *Operative Hand Surgery*, 3rd ed. New York: Churchill Livingstone, 1993, vol 1, pp 1021-1038.)

hand swelling may be so impressive that the palmar process can be overlooked. Passive motion of the middle and ring fingers elicits pain.

Transverse and oblique longitudinal incisions have been described for drainage, as well as a distal approach in the third interspace, which allows evacuation of the abscess through the lumbrical canal[22] (Fig. 6). Wide exposure and drainage is preferred, after which the wound is either left open and dressed with loose wet-gauze packing (which should be changed daily) or closed over irrigation catheters, as previously described.

Involvement of Parona's space usually results from contiguous spread from the radial or ulnar bursa.[21] Symptoms may include acute carpal tunnel syndrome and pain with finger-flexor motion. Management is similar to that of other deep-space infections, emphasizing wide exposure and thorough drainage, and avoiding placement of incisions directly over the flexor tendons and the median nerve to avoid desiccation.

Old-fashioned collar buttons were dumbbell shaped; the collar closure was secured by passing the ends of the collar button through overlapping buttonholes. "Collar-button abscess" describes a subfascial infection of a web space that tends to spread peripherally at the palmar and dorsal ends and remains narrow in the middle. It usually results from contiguous spread, most often from an infected palmar blister or skin fissure. Because of the adherence of the palmar skin and underlying fascia, the infection cannot spread peripherally; rather, it is forced to expand dorsally through the space in the palmar fascia (just distal to the bifurcation of the neurovascular bundle) to involve the subcutaneous tissue of the dorsal web.[1,23] In addition to the findings of a palmar infection, there is considerable dorsal web-space swelling. In contrast to a simple dorsal subcutaneous abscess, a collar-button abscess is characterized by an abducted resting posture of the adjacent digits.

Some recommend a palmar incision with excision of the palmar fascia in the interdigital space to allow drainage of the palmar and dorsal extension of the infection through the same wound, while others advocate palmar and dorsal incisions.[22,23] Transverse incisions should be avoided because a web-space contracture may result. Postoperative management includes daily changes of the loose wet-gauze dressings and early range-of-motion exercises. Healing is by secondary intention.

Septic Arthritis

The usual mechanisms of hand-joint infections include direct penetration and extension from contiguous infection or hematogenous spread. Bacterial toxins, proteolytic enzymes, bactericidal enzymes of synovial and reticuloendothelial origin, and even proteoglycanolytic enzymes of cartilaginous origin are released during joint sepsis and instigate cartilage destruction.[24] As the intra-articular exudate increases, the intra-articular pressure rises, impeding synovial blood supply and causing direct cartilage damage, which in turn can result in capsule and bone erosion, sinus formation, and osteomyelitis.[24]

Staphylococcus aureus and *Streptococcus* organisms are most prevalent.[1,24] *Haemophilus influenzae* should be considered in the young child, and *Gonococcus* infection should be considered in the young adult with monarticular nontraumatic septic arthritis.[1,24] The pres-

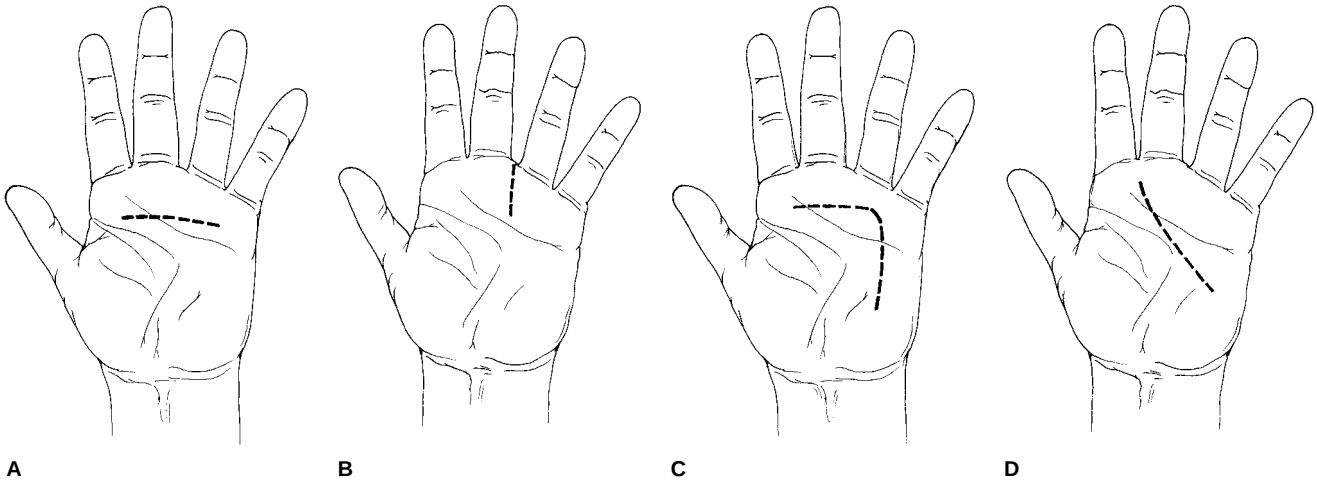

Fig. 6 Recommended incisions for drainage of deep palmar-space infections. **A,** Transverse distal palmar incision. **B,** Approach through the third lumbrical canal. **C,** Combined longitudinal-transverse approach. **D,** Oblique longitudinal approach.

ence of *S aureus* organisms, Gram-negative rods, or anaerobes; polymicrobial infections; positive blood cultures; and associated osteomyelitis all portend a poor outcome.[24] Outcome is closely related to the duration of the infection before adequate treatment.[23,24]

At physical examination, the joint is painful, swollen, and erythematous, usually assuming the posture that accommodates maximal joint volume. There is pain with motion and axial loading. A puncture wound may be identified. Hematogenous spread should be suspected in patients with systemic symptoms. Noninfectious conditions, such as crystalline arthropathies, may present similarly and should be ruled out. Joint aspiration should be performed if possible. Fluid findings characteristic of joint sepsis include a friable mucin clot; a white blood cell count of more than 50,000, of which more than 75% are polymorphonuclear lymphocytes; and a glucose level 40 mg less than the fasting blood glucose level.[1,24] Gram stain may or may not show organisms.[24]

While joint aspiration may be diagnostically useful, and there are those who recommend treating joint sepsis with serial aspirations, it is therapeutically unreliable in the hand.[24] Definitive incision and drainage allows evacuation of the offending exudate, removal of pannus and necrotic debris, and reduction of intra-articular pressure. Incisions should be straight to avoid flap-tip necrosis. The wrist is drained dorsally, with the entry usually between the third and fourth dorsal wrist compartments. Over the carpometacarpal joints, incisions should be dorsal and adjacent to extensor tendons. For the finger metacarpophalangeal joints, incisions should be placed dorsally, entering the joint through the extensor slip or (as we prefer) through the proximal portion of the sagittal band. For the interphalangeal joints and the thumb metacarpophalangeal joint, a midaxial incision is preferred, entering the joint at the junction between the accessory collateral ligament and the volar plate. This preserves the extensor mechanism and helps to avoid septic boutonniere and mallet deformities.[1,24]

Postoperatively, the wound can be left open for delayed primary closure or can be allowed to heal by secondary intention. In cases in which an exceedingly clean joint is achieved (especially if there is concern about desiccation of underlying structures), the wound can be loosely closed over an irrigation catheter, with intermittent or continuous irrigation and fluid egress allowed through the wound margins into a padded dressing. The hand is splinted for 48 to 72 hours in a functional position, after which drains and splints are removed and motion is encouraged.

Parenteral antibiotics are continued until local and systemic signs have resolved. Thereafter, parenteral or oral antibiotics are given for 2 to 4 weeks after the initial debridement.[24] Home IV therapy has facilitated hospital discharge in cases in which antibiotic treatment was the sole reason for hospitalization.

Human Bites

Infected human-bite wounds that present late (more than 24 hours after the bite) are usually infected. Two typical injuries occur: clenched-fist injuries, with a wound over the metacarpal head, and true bite-induced wounds. Clenched-fist injuries may involve the metacarpophalangeal joint. The early clenched-fist injury wound may appear innocuous if examination of the involved finger is performed with the metacarpophalangeal joint in extension. Retraction of lacerations in the extensor mechanism and joint capsule proximal to the skin laceration may render a traumatic arthrotomy undetectable on casual inspection. A wound on the dorsum of the metacarpophalangeal joint must be considered a clenched-fist injury with possible joint involvement until proved otherwise because of the grave consequences that ensue if such an injury is not properly treated.[2,3]

Forty-two bacterial strains have been isolated from human-bite wounds.[4] Group A *Streptococcus, S aureus,* and *E corrodens* organisms have been most commonly associated with infection.[4] *Eikenella corrodens* has been associated with 7% to 29% of human-bite infections and is variably susceptible to cephalosporins but is not susceptible to penicillinase-resistant penicillins.[4] *Bacteroides* species are the most commonly isolated anaerobes and are usually associated with mixed cultures.

The workup of bite wounds includes aerobic and anaerobic cultures. Radiographs may show a fracture, foreign body (e.g., a tooth), or osteomyelitis.

Noninfected bite wounds should be extended, explored, irrigated, and debrided with appropriate instruments, adequate light, tourniquet control, and assistance. Splinting, elevation, hospitalization, and IV antibiotic therapy have been recommended for 48 hours, especially if a joint was entered.[2,3] In the absence of tendon or joint injury, the combination of debridement and antibiotic therapy has been found to lead to better results than debridement without antibiotic therapy, with no significant difference in outcome between intravenous and oral administration.[25]

Infected bites require irrigation and debridement, hospitalization, and IV antibiotic therapy, with repeat debridement at 48 hours if necessary. Empiric antibiotic coverage includes IV penicillin G and a first-generation cephalosporin; some au-

thors also recommend coverage with an aminoglycoside.[1,2] Warm soapy water soaks or whirlpools and wet-gauze dressing changes are started 24 to 48 hours after initial debridement.[2] Tendon repairs are performed in a delayed fashion, and wounds are left open and allowed to heal by secondary intention.[21]

Animal Bites

In the United States, animal bites are most frequently inflicted (in decreasing order of prevalence) by dogs, cats, and rodents, and often involve the hand.[4,26] In one study, dog bites rarely became infected, while 50% of cat bites did.[27] *Pasteurella multocida* is commonly associated with infections from dog and cat bites. *Staphylococcus*, *Streptococcus*, and some anaerobes are also common pathogens.[4,26,27]

As with human bites, meticulous wound irrigation decreases the risk of subsequent infection.[26] Wound extension and exploration are indicated if there is suspicion of joint, bone, or tendon-sheath penetration. Antibiotic prophylaxis, although controversial, is probably indicated for all but the most trivial hand wounds.[26]

Infected wounds usually require hospitalization for definitive incision and drainage, wound care, and parenteral antibiotic therapy as described for human bites. Empiric antibiotic therapy should provide coverage for *P multocida*, *Staphylococcus* and *Streptococcus* organisms, and anaerobes (penicillin G and a first-generation cephalosporin).

Rabies, although uncommon in the United States, warrants brief mention. Criteria for prophylaxis have been established by the Centers for Disease Control. No treatment is required for bites inflicted by healthy dogs or cats that have been observed for 10 days. Victims of bites from a skunk, bat, fox, coyote, raccoon, or bobcat or from a rabid or suspected rabid domestic animal should be treated with human diploid cell vaccine and rabies immune globulin. Other animals (e.g., mice, rats, hamsters, rabbits, and squirrels) should be considered individually, but their bites almost never call for antirabies prophylaxis.[26]

Osteomyelitis

Open fractures are the most common cause of osteomyelitis in the hand.[1,24] Contiguous spread is another common mechanism. Hematogenous spread is rare and is more common in children than in adults. *Staphylococcus aureus* and *Streptococcus* are the most common organisms. There is a higher incidence of Gram-negative, anaerobic, and polymicrobial infections in immunosuppressed patients, diabetic patients, and those with mutilating injuries or injuries that occurred in a contaminated environment.[5,24] The infection rate for open hand fractures ranges from 1% to 11% and increases with wound severity.[1,24] Osteomyelitis in this setting can be minimized with adequate debridement and staged reconstruction.[24]

Physical findings include pain, erythema, and swelling. Systemic symptoms are rare unless there is a severe fulminant infection, which should raise the suspicion of antecedent bacteremia and hematogenous spread. In the acute phase, nuclear medicine studies may depict pathologic changes before they are visualized on x-ray films. Early radiographic findings may include local osteopenia and periosteal reaction, although even these findings are delayed from initial onset. In the chronic phase, sequestra and involucra are present and may be visible radiographically. The index of suspicion should be heightened when a presumed soft-tissue infection does not respond or intermittently resolves, only to be followed by recurrent drainage.

Before sequestrum formation, acute osteomyelitis can sometimes be successfully treated medically. Periosteal or intramedullary aspiration can yield the offending organisms. However, even in the acute stages, most surgeons prefer debridement and cortical windowing followed by antibiotic treatment.[24] When there is sequestrum formation, medical management alone is insufficient, and surgical debridement to the level of viable soft tissue and bone is necessary.

Initial high-dose IV antibiotic therapy is based on suspected organisms and usually provides coverage at least for *Staphylococcus.* When culture and sensitivity results become available, parenteral antibiotic coverage is amended if necessary. Intravenous antibiotics are continued until local erythema, swelling, and pain resolve, wounds close, and drainage stops. Oral antibiotics are usually continued for 4 weeks in cases of acute osteomyelitis and for 4 to 6 weeks in cases of chronic osteomyelitis.[24] Normalization of a previously elevated erythrocyte sedimentation rate is a good indicator for completion of the antibiotic course.

In cases of infected open fractures less than 6 weeks after injury, internal fixation should remain in place if it is providing stability. If by 6 weeks union has not occurred, any inadequate fixation should be replaced or an external fixator applied or both.[24]

Necrotizing Fasciitis

Necrotizing fasciitis is a life- and limb-threatening soft-tissue infection. It occurs frequently, but not exclusively, in indigent abusers of IV drugs and alcohol.[17,28] The extremities are the most common sites of infection, which is usually initiated by major or minor trauma.[28] In one study, 63% of cases were the result of IV drug abuse.[17]

The infection can be caused by one or more organisms. In about half of the cases in one large series,[17] only one organism was isolated—

most frequently group A *Streptococcus*.[17] Other organisms found in polymicrobial infections include α- and β-hemolytic *Streptococcus*, *Staphylococcus* species, and anaerobes.[17,28]

Necrotizing fasciitis is characterized by intensely painful, rapidly advancing, poorly demarcated cellulitis with tensely swollen, shiny skin. Within a few days, bullae and ecchymoses appear. Early in the course, the usual systemic signs of infection may be lacking, although leukocytosis is consistently present.[17] Hemodynamic instability in the face of otherwise trivial-appearing cellulitis should increase suspicion of necrotizing fasciitis.

The definitive diagnosis is made at surgery when fibrinous necrotic tissue is found to be accompanied by liquefaction of the subcutaneous fat and a characteristic, often foul-smelling, thin fluid referred to as "dishwater pus." There is usually thrombosis of subcutaneous vessels. Depending on the stage of the disease, the skin may or may not appear viable. The muscle is usually spared. Wide debridement of the involved tissue, which often includes the overlying skin, is required.

Cultures and Gram-stain specimens are obtained at the first opportunity, and antibiotics are started emergently and empirically, including high doses of penicillin G as coverage for penicillinase-resistant *Staphylococcus*. Although Gram-negative organisms are rare, an aminoglycoside is also recommended because of the potential morbidity of this severe infection.[17]

In two studies, reports of mortality ranged from 8.7% to 33%, with the single factor most influencing morbidity and mortality being early and adequate debridement.[17,28] Negative prognostic factors include age over 50 years, underlying chronic illness, diabetes mellitus, and involvement of the trunk.[28]

Mycobacterial Infections

Atypical mycobacteria have been increasingly recognized as pathogens, and more than 75% of atypical mycobacterial infections involve the hand.[1,9] Of the atypical mycobacteria, *Mycobacterium marinum* is the most frequently identified organism. Inoculation usually occurs by direct penetration from contaminated swimming pools, fish tanks, piers, boats, fish bites, or injuries from fish fins or spines.[9,29] Infections can be cutaneous (verrucal), subcutaneous (granulomatous), or deep (involving tendon, joint, bursal synovium, or bone).[9]

There are no pathognomonic physical findings, although deep infections are frequently associated with abundant tenosynovitis or joint synovitis. Variation from a typical rheumatoid pattern should alert the clinician. Many cases are initially missed, and as a result, appropriate treatment is delayed.[9,29] Diagnosis is initially based on clinical suspicion. Skin tests and smears for acid-fast organisms are unreliable.[1,9,29] When smears are negative, fungal infections, although rare, should be ruled out.[29] Systemic symptoms are usually absent, and the white blood cell count and erythrocyte sedimentation rate are typically normal.[9] Granulomas are noted on histologic examination of biopsy specimens. Cultures must be incubated in Löwenstein-Jensen medium at 30˚C for up to 8 weeks.[9,29]

A superficial infection is usually self-limited unless the lesion has been "picked at" or a biopsy specimen has been obtained without postoperative antibiotic coverage, in which case it can be converted into a subcutaneous lesion.[9] A subcutaneous lesion should be debrided, and antibiotics should be administered for 2 to 6 months. Deep lesions should be treated with tenosynovectomy, synovectomy, or adequate incision and drainage of the infected bone or joint, with antibiotic administration for 4 to 24 months.[9,29]

In cases of hand tenosynovitis, the flexor tenosynovium in the fingers is completely removed, preserving the annular pulleys. On the dorsum of the wrist, a longitudinal incision is made to debride areas of extensor tenosynovitis. An extensile carpal tunnel approach is used to expose the flexor tenosynovium at the wrist.[20]

Minocycline is the antibiotic of choice because it is associated with fewer side effects than the alternatives. In cases of allergy or organism-insensitivity, ethambutol and rifampin may prove useful.[1,9,29]

Fungal Infections

Fungal infections of the upper extremities can be grouped in four categories: cutaneous, subcutaneous, deep, and systemic.[10] Cutaneous lesions involve the skin or nails. Keratinophilic fungi (dermatophytes) can colonize areas of glabrous skin (tinea corporis), the interdigital areas and palms (tinea manuum), or the nails (tinea unguium, or onychomycosis), causing pruritic scaling of the skin or a nail deformity. A presumptive diagnosis can be made on the basis of potassium hydroxide preparations; however, definitive diagnosis depends on the results of fungal cultures.

Uncomplicated skin infections are treatable with topical agents, such as tolnaftate and miconazole. Onychomychosis typically occurs in hands that remain moist; it begins as a minor paronychial infection and progresses to whole-nail involvement characterized by thickening, softening, discoloration, and cracking. Psoriatic nail deformities can mimic onychomycosis. Onychomychosis is relatively resistant to treatment and has a high recurrence rate. Nail removal and application of topical agents are less successful than oral administration of griseofulvin or ketoconazole for systemic effect.[10] Reported cure rates have ranged from 57% to 80%.[10]

Subcutaneous and deep fungal infections are often overlooked in the differential diagnosis of hand infections. Sporotrichosis, the most common subcutaneous fungal lesion in North America, occurs predominantly in the upper extremities.[10] Subcutaneous implantation is the usual mode of transmission and usually involves handling contact of a variety of plants (e.g., rose thorns, cactus, sphagnum moss). Ulceration occurs at the initial puncture site, and nodules eventually form along lymphatic vessels, which in turn can ulcerate. Despite the typical clinical presentation and exposure history, the condition is frequently neglected in the differential diagnosis, and as a consequence, treatment is often delayed. Because sporotrichosis is difficult to diagnose on the basis of standard stains, definitive diagnosis requires cultures. The treatment of choice is oral potassium iodide.

Deep fungal infections of the hand can involve tenosynovium, joints, or bones. Organisms can be virulent (histoplasmosis, blastomycosis, coccidioidomycosis) or opportunistic (mucormycosis, aspergillosis, candidiasis) with the latter affecting the immunosuppressed patient. Infectious agents usually enter via the pulmonary route, and musculoskeletal infections occur by hematogenous spread. Treatment requires debridement and administration of amphotericin B. Coccidioidomycosis arthritis is particularly resistant to treatment and can be managed only by amputation or arthrodesis.[10]

Summary

The morbidity of hand infections can be decreased with understanding of the different types of hand infections often unique to the particular anatomy of the hand. For optimal results, aggressive treatment must be instituted expeditiously, followed by appropriate aftercare. This frequently entails incision and drainage, antibiotic therapy, wound care, edema control, and mobilization.

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