Surgical Treatment of Osteomyelitis

George Cierny, III, M.D.
San Diego, Calif.

Summary: Chronic osteomyelitis is refractory to nonsurgical treatment due to a resilient, infective nidus that harbors sessile, matrix-protected pathogens bound to substrate surfaces within the wound. Curative treatment mandates physical (surgical) removal of the biofilm colony, adjunctive use of antibiotics to eliminate residual phenotypes, and efforts to optimize the host response throughout therapy. Patient selection, therapeutic options, and the treatment format are determined by the Cierny/Mader staging system, while reconstruction is governed by the integrity/stability of the affected bone(s) and quality/quantity parameters of the soft-tissue envelope. (*Plast. Reconstr. Surg.* 127 (Suppl.): 190S, 2011.)

Chronic osteomyelitis is a biofilm infection wherein only a small fraction of the microorganisms are free-swimming (planktonic), available (for culture), and sensitive to systemic agents. Instead, the overwhelming majority of pathogens are sessile-based, resiliently attached (to dead bone, implants, or foreign bodies) and embedded within a microbial-based slime (biofilm). Once in the sessile state, the organisms are invulnerable to both the host's defenses and circulating concentrations of antibiotics.[1,2] With time, microbial toxins and caustic by-products of the host's cellular defenses accumulate to cause a profound local and systemic compromise (sepsis, tissue loss, and chronic edema). It is this pathophysiology that mandates a multidisciplinary approach to treatment: complete surgical removal of the biofilm burden (débridement), medical optimization of host defenses, and administration of antimicrobial agents at concentrations to kill pathogen phenotypes within the wound following débridement.[3,4]

Over the last three decades, treatment strategies to counter mechanisms of surface colonization by potential pathogens have set the foundation for today's limb salvage protocols.[4–15] Innovative technologies introduced versatile fixation devices,[16–18] methods of tissue regeneration,[19,20] antimicrobial agents and isolation strategies,[21–26] local antibiotic delivery systems,[27–31] negative-pressure applications,[32] and function-sparing ways to transfer living tissues site to site.[33–38] With the ability to convert dirty wounds to clean wounds[39] and restore both form and function, the treatment of musculoskeletal sepsis entered a new era wherein the prospects of a successful reconstruction derailed the *fear of failure* that had been blocking surgeons from initiating treatment at all.[40]

CLASSIFICATION

Today, over 90 percent of patients suffering from chronic osteomyelitis can expect a successful treatment outcome.[41] As in musculoskeletal oncology, treatment selection is now guided by a staging system to articulate treatment with pathophysiology, stratify patient cohorts, and facilitate the comparison of therapeutic options. Published in 1985, the Cierny/Mader classification of adult osteomyelitis[10] was, historically, the first system to articulate treatment with the natural history of the disease. Though usually more descriptive in nature, previously published classifications had not proven helpful in the management of individual patients.[42,43] In the Cierny/Mader system, a clinical stage of osteomyelitis is formulated to aid in planning the course of treatment, identify progressive stages of the disease, act as a prognostic indicator, and assist in selecting the surgical approach.[10,44–47] The system is reproducible, can be used to compare protocols and/or treatment centers, and is also appli-

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cable to infections of the hand, head, and/or spine for which therapeutic options may vary.[4,48–52]

The Cierny/Mader classification stratifies the primary factors affecting treatment: the extent of osseous necrosis, the health of the patient, and the impact of disease on function. Three host classes (A-hosts, B-hosts, and C-hosts) and four anatomic variants (types I, II, III, and IV) are combined to designate one of 12 clinical stages. (i.e., stage IVB osteomyelitis). The anatomic type determines the surgical approach, whereas the host class governs the selection of therapeutic options. The interplay of the three factors (above) with the anatomic site of the infection and experience of caregivers will mandate treatment to be palliative or curative, simple or complex, limb-sparing or ablative.

The Host

The staging system combines the health status of the patient with the physical impact of disease (on function) into a physiologic host class: healthy patients are designated as A-hosts, and patients with comorbidities affecting their response to stress, trauma, or infection are classified as B-hosts. The B-host is at risk for treatment failure due to a high incidence of metabolic deficiencies, immune compromise, episodes of bacteremia, wound slough, and excessive bleeding (Table 1).[47,53] If the risks and/or morbidity of treatment outweigh the benefits, the patient is classified a C-host and not offered definitive care. Instead, the C-host is palliated or simply treated expectantly.

Host optimization (the reversal/optimization of comorbidities) throughout treatment will improve B-host outcomes to more closely parallel those for A-hosts.[47,54] Treatment risk can also be downsized with the selection of low-morbid surgical solutions and avoidance of surgical implants (Table 2).[44–47,54–59] If, however, an implant is needed, reconstruction is best staged to follow treatment with an antibiotic depot to decrease the strain on host defenses, clear residual pathogens, safeguard coverage, and increase success rates.[14]

Disability

To offset the morbidity/sequelae of treatment, treatment for cure must offer the patient significant advantage over palliation or observation alone (see C-host, above). In this regard, persistent infection, pain, deformity, and even chronic drainage are not absolute indications for treatment, as the primary goal of therapy is to improve a patient's quality of life.

Table 1. Patient Comorbidities Affecting Treatment and Outcomes*

Local factors
Chronic edema
Venous stasis
Large vessel disease
Arteritis
Extensive scar
Radiation fibrosis
Obesity
Foreign body excess†
Systemic factors
Malnutrition
Immune deficiency
Hypoxia
Malignancy
Diabetes
Old age
Organ failure
Bleeding diathesis
Nicotine abuse
Intravenous drug abuse
Drug inhibitors‡
Skin colonization

*Comorbidities include systemic and regional (local) host comorbidities associated with wound-healing deficiencies in patients undergoing treatment for chronic osteomyelitis.
†Foreign body excess includes an amount or distribution of foreign material at the site of infection that prohibits treatment for cure (i.e., a diffuse aray of shotgun pellets/shrapnel).
‡Drug inhibitors are essential medications affecting bony union (i.e., Dilantin or fluoroquinolones).

Table 2. Adult Osteomyelitis (Initial Treatment Success Rates)*

Method	No. of Procedures	First Treatment Success Rates	
		A-Host (%)	B-Host (%)
Muscle flaps[44]	130	95	75
Cancellous grafts[55]	150	94	79
Papineau grafts[56]	20	80	20
Free osseous grafts[44,57]	42	83	59
Revision total joints[46]	74	96	76
Osteosynthesis[45]	458	96	75
Bone transposition[58]	*78*	*98*	*92*
Simple closure[46,59]	*142†*	*96*	*91*
Bone transport[54]	*120*	*76*	*72*
Permanent spacers[47]	*44*	*93*	*88*

*Various prospective cohort studies (evidence: level II) are listed that illustrate how first treatment outcomes for chronic osteomyelitis were statistically inferior in B-hosts versus A-hosts when reconstruction was associated with high surgical morbidity. Following low-risk, low-morbidity methods (italicized items), there were no significant differences between cohort groups. Adapted with permission from Cierny G, DiPasquale D. Treatment of chronic infection. *J Am Assoc Orthop Surg.* 2006;14:105–109.
†Simple closures, apposition of adjacent tissues without formal transposition or transfer.

The Disease

With few exceptions, a thorough débridement is the unchallenged cornerstone of successful therapy.[4,45] The anatomic types of osteomyelitis

escalate in complexity from type I to type IV and articulate the natural patterns of disease with treatment (Fig. 1).

Medullary Osteomyelitis (Type I)

In type I osteomyelitis, the biofilm nidus is confined to the endosteum as dense scar, infarcted marrow, dead bone, or a medullary implant. Soft-tissue involvement is usually reactive in nature and responsive to removal of the nidus and a short course of antibiotics. The hematogenous variant is most commonly found in compromised hosts, tends to be diaphyseal rather than metaphyseal, and accounts for less than 2 percent of the cases treated in our registry since 1981 (Table 3).

Superficial Osteomyelitis (Type II)

Type II osteomyelitis is a true form of the contiguous-focus osteomyelitis described by Wadvogel et al. in 1970.[43] Here, the nidus is an exposed, bony surface at the base of a chronic, open wound. The medullary contents are not involved. Common examples include bone at the base of a pressure sore (decubitus) and chronic wounds associated with Papineau bone grafts.[60]

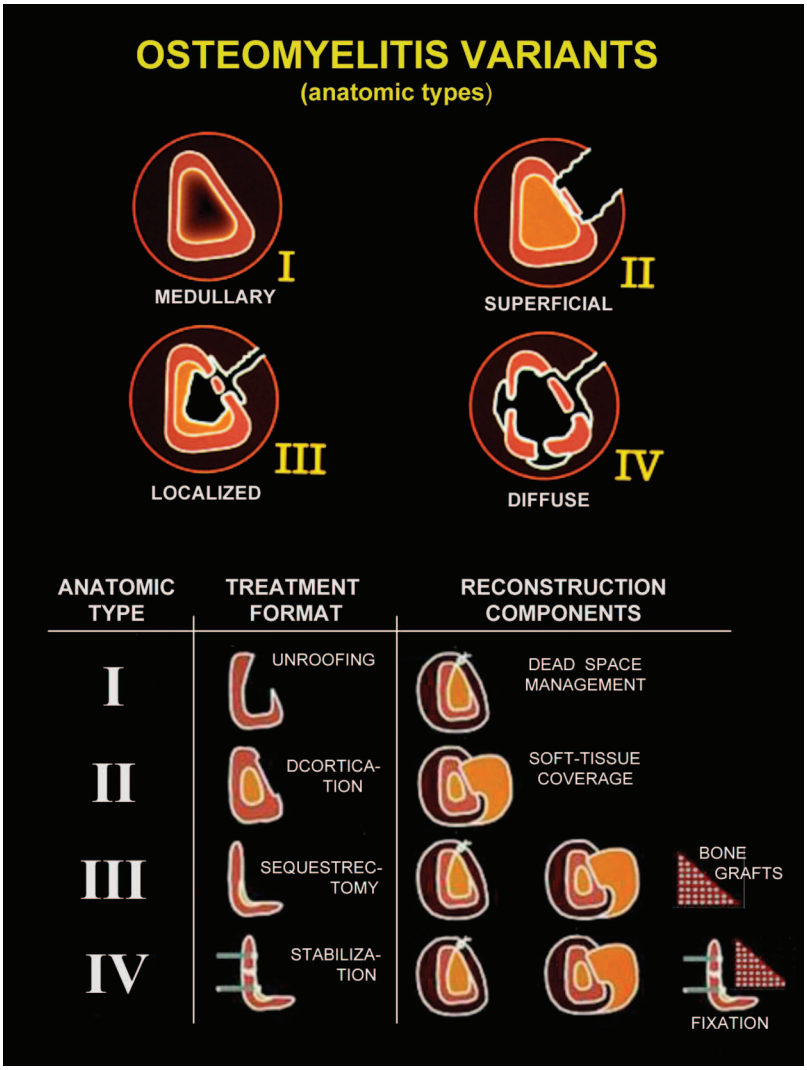

Fig. 1. A graphic depiction of the four anatomic types of osteomyelitis matched with a surgical and reconstruction format for each. Adapted from Cierny G. Chronic osteomyelitis: Results of treatment. In: Greene WB, ed. *Instructional Course Lectures.* Vol. 39. Rosemont, Ill.: American Academy of Orthopaedic Surgeons. 1990;39:495.

Table 3. Treatment Candidates (*n* = 2207): 1981 to 2007 (Success at >2 Years)*

Stage	No. of Cases	Success Rate (%)
IA	51	100
IIA	88	100
IIIA	209	97
IVA	595	99
Total	943	99
IB	71	94
IIB	194	89
IIIB	139	95
IVB	860	90
Total	1264	90
Overall 2-year success rate		95

*The relative incidence of anatomic types of adult osteomyelitis treated by the author from 1981 through 2007 was 6, 13, 16, and 66 percent for types I, II, III, and IV, respectively. There were 106 C-hosts in this patient population. The 2-year success rates are shown for each clinical stage.

Localized Osteomyelitis (Type III)

The hallmark of type III osteomyelitis is presence of a full-thickness, cortical sequestrum (Figs. 2 and 3). The canal is involved (type I pattern), there may be a soft-tissue deficit (type II pattern), and indwelling hardware is commonly present. Examples would include an infected fracture union with plate fixation and presence of a sequestered, butterfly fragment. To distinguish this from a type IV osteomyelitis (see below), the involved bony segment will still be stable following a complete débridement.

Diffuse Osteomyelitis (Type IV)

This is a permeative, through-and-through type of infection combining the characteristics of types I, II, and III osteomyelitis with the additional feature of instability (Figs. 4 through 10). These lesions are either intrinsically unstable (i.e., an infected nonunion) or rendered unstable with débridement (i.e., a peri-prosthetic, total joint infection, or a hematogenous osteomyelitis requiring diaphysectomy for cure).

TREATMENT PROTOCOL

The staging process begins with a review of the medical/surgical history, laboratory tests, and physical findings to establish host status, integrity of the musculoskeletal system, and extent of disease (Table 4). Assessment of the soft-tissue envelope is carried out in anticipation of problems related to the surgical approach, skeletal fixation, and wound closure. Clinical signs and symptoms of vascular compromise will guide a perfusion workup with vascular indices, transcutaneous oxygen tensions, and/or an angiogram.[61–77]

The diagnostic imaging of osteomyelitis often requires a combination of diverse imaging techniques for accurate clinical staging.[78] Conventional radiography provides an overview of the anatomy and the pathologic conditions of the bone and soft tissues of the region of interest. However, because the specificity of plain radiographs for detection is higher than its sensitivity, other more reliable methods of imaging are needed.[79–81] Ultrasonography is most useful in the diagnosis of fluid collections, periosteal involvement, or abnormalities in the surrounding soft tissues and may provide guidance for diagnostic or therapeutic aspiration, drainage, and/or tissue biopsy. Though not particularly sensitive in detecting osteomyelitis, a computed tomography scan is a useful method to detect early osseous erosion and document the presence of sequestra, cloacae, foreign bodies, and/or gas formation (Fig. 4, *below, left inset*).[80] Bone scintigraphy provides a nonspecific but sensitive detection of osseous disease, while an indium-111–labeled white blood cell scan is more specific in identifying infection, can be used to help diagnose and localize osteomyelitis, and is useful in evaluating the response to treatment.[81] Magnetic resonance imaging is the most sensitive and most specific imaging modality for the detection of infection in bone (sensitivity/specificity, 82 to 100 percent/75 to 95 percent), provides superb anatomic detail and gives accurate information on the extent of the infectious process in bone and soft tissue (Figs. 2, *right*, and 4, *right*).[58,80,82–84]

Treatment Format

To justify the morbidity and risk of limb salvage, the expected outcome must offer distinct advantage(s) over an amputation or observation, alone. If treatment for cure is contraindicated or excessive, the patient is classified a C-host and offered palliation (incision/drainage, oral antibiotics, ambulatory aides, and pain medication). Amputation is indicated when limb salvage and palliation are neither safe nor feasible.

Surgical Algorithms for Osteomyelitis of the Extremities (Excluding Hand)

Amenable comorbidities are reversed and/or minimized (host optimization) before surgery. Thereafter, soft tissues are resected to supple, well-perfused margins, and the bone is tangentially excised until exposed surfaces bleed in a uniform, Haversian (cortical), or sinusoidal (cancellous) pattern (the paprika sign).[85] All foreign bodies and surgical implants are removed. Frozen-section biopsies are taken and sent for histologic examination to confirm the presence of inflammation, validate culture setups for fungi and micobacterial species, and rule out

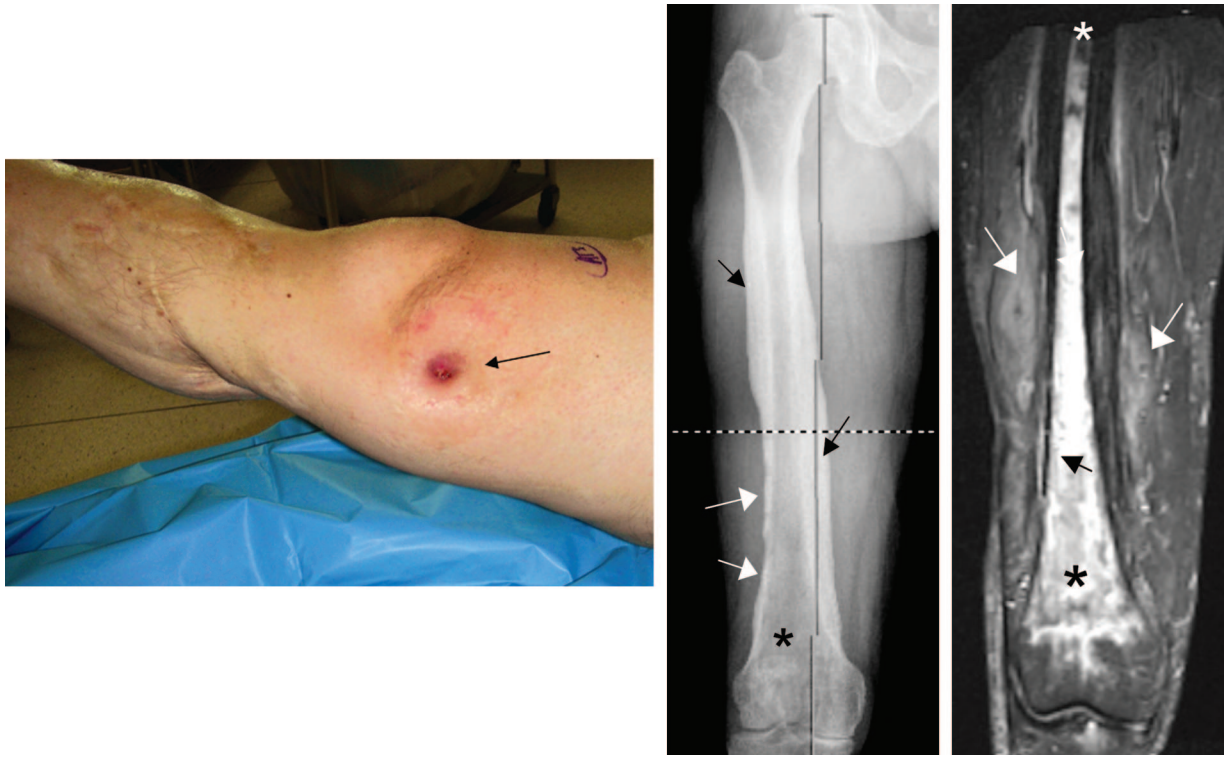

Fig. 2. (*Left*) A draining sinus in the distomedial thigh of a patient with an infected fracture union (femur) seen 30 years after an open fracture initially treated with medullary and then plate fixation. (*Center*) Anteroposterior radiograph showing reactive callus (*black arrows*), sequestered lateral cortex (*white arrows*), and distal osteopenia (*). (*Right*) A coronal T2, magnetic resonance imaging cut showing the medullary extent of the lesion (*), the sequestered lateral cortex (*black arrow*), and several abscesses in the soft tissues (*white arrows*).

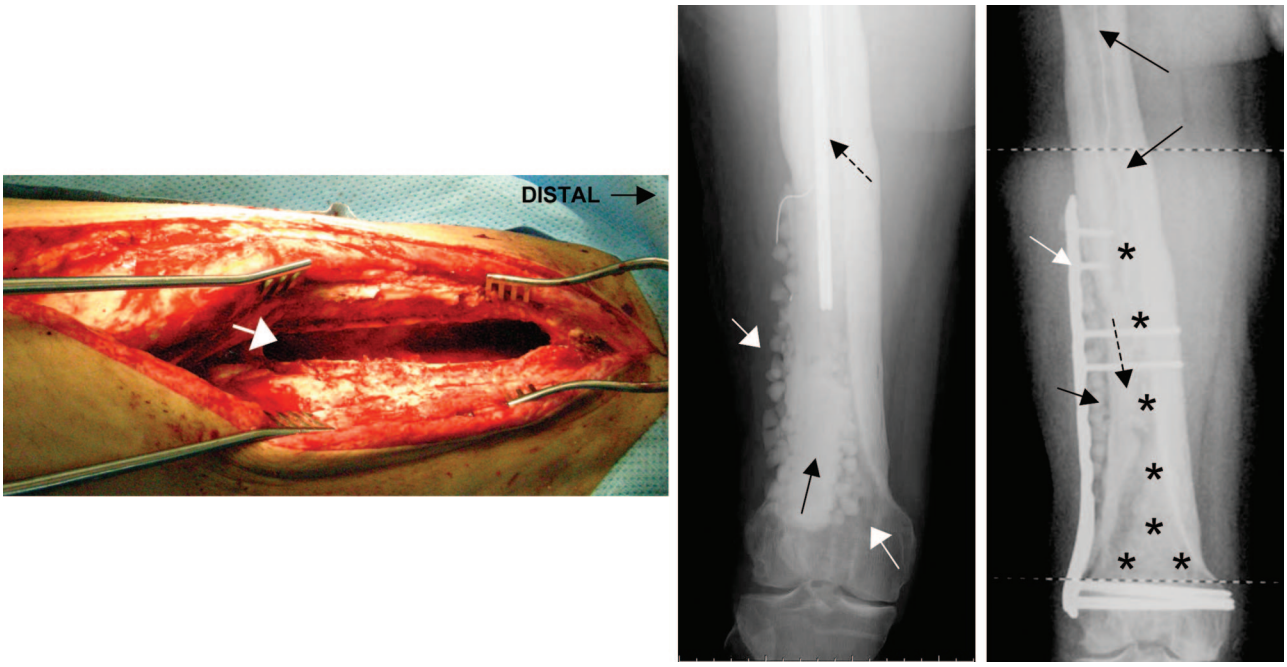

Fig. 3. (*Left*) To treat the patient, the lateral sequestra were excised to unroof (*white arrow*) and débride the metaphysis. (*Center*) The wound was closed over antibiotic beads (*white arrow*) and an antibiotic spacer (*black arrow*) reinforced with a small rod (*broken arrow*). (*Right*) Three months later, reconstruction was performed using a locked plate (*white arrow*), antibiotic beads (*black arrows*), cancellous grafts (*), and a permanent antibiotic spacer (*broken arrow*).

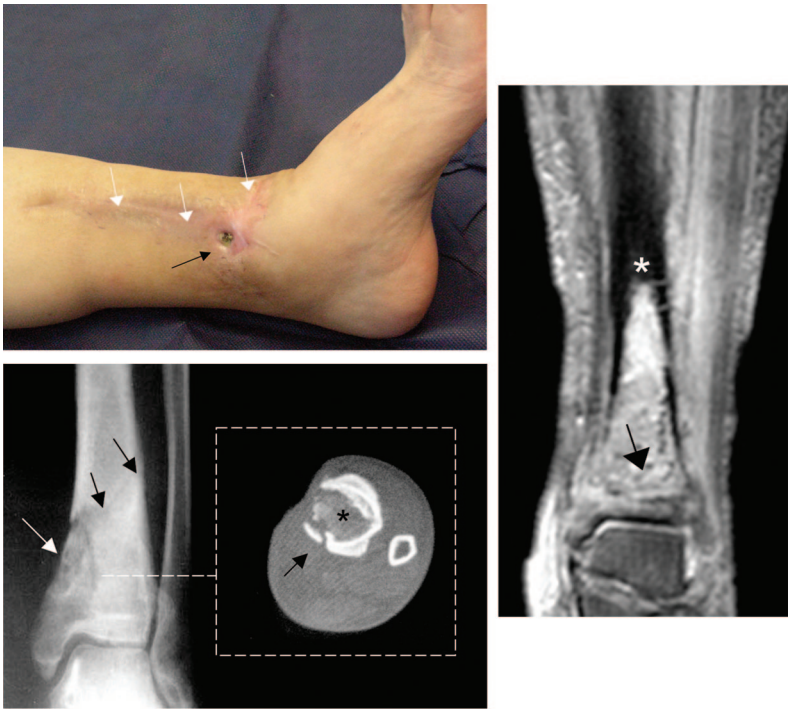

Fig. 4. (*Above, left*) Appearance of the distal leg of a woman with infection following internal fixation of her distal tibia while she was immunosuppressed for lupus erythematosus. Soft-tissue compromise (*white arrows*) and a chronic sinus (*black arrow*) persisted following hardware removal. (*Below, left*) An anteroposterior radiograph at presentation demonstrated a nonunited fracture (*black arrows*) and a cortical defect beneath the open sinus (*white arrow*). (*Inset*) A computed tomography scan cut at the level of the medial defect disclosed a cortical sequestrum (*black arrow*) at the cloaca and cavitation (*) of the distal tibial metaphysis. (*Right*) A coronal T2 magnetic resonance imaging cut mapped the extent of the medullary component (*) and sequestration of allogeneic bone grafts (*black arrow*) placed at the initial fracture fixation.

other pathologies mimicking infection (neoplasms, autoimmune disorders, and dysplasias).

Pathogen Identification

Culture of a microorganism on solid or liquid media remains the accepted standard for diagnosing osteomyelitis. Using this approach, multiple tissue samples are collected from separate regions of the wound and sent to microbiology for culture and sensitivity testing on various media, as indicated.[86–88] If orthopedic implants, foreign bodies, and/or sequestra are removed during débridement, successful, and quantitative culture yields can be enhanced up to twofold using a sonication process (available in most laboratories) to shear still biofilm-bound, planktonic organisms for processing.[2,89–91] However, because only a very small percentage of the biofilm colony is planktonic (culturable), even the most sophisticated flask cultures often fail to identify an inciting organism, even in the face of an overwhelming clinical infection.

With the genomes of medically important microorganisms now well characterized, the introduction of mass spectrometry, pulsed field gel electrophoresis and DNA pyrosequencing into clinical microbiology laboratories has ushered in a new era in molecular diagnostic pathogen detection and characterization.[92–94] This rapid and highly accurate technology can assess fresh, dry, or frozen specimens, applies to all pathogen classes and species, and can detect any organism regardless of its culturability, prior antimicrobial treatment, or metabolic state. It is our current practice to use quantitative polymerase chain reaction pyrosequencing is used to assess all wounds associated with a previous or ongoing infection, as we have documented a high incidence (23 to 30 percent) of microorganisms present even after interval treatment with high concentrations

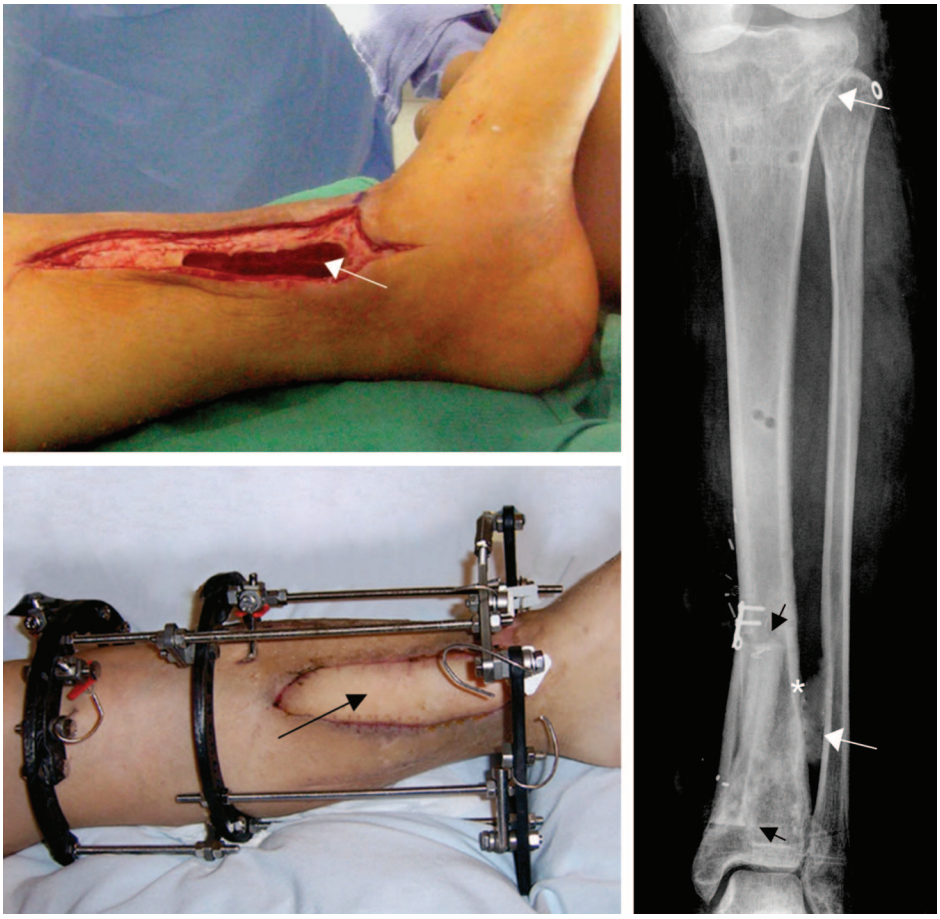

Fig. 5. (*Above, left*) At the initial debridement, the extent of a distal unroofing and careful preservation of the periosteal blood supply is evident. (*Below, left*) Three days later, the patient underwent reconstruction with an external fixator, a vascularized fibula with skin paddle (*black arrow*), and autogenous cancellous grafts. (*Right*) An anteroposterior radiograph at the time of external frame removal shows the healed fibular graft (*black arrow*), healing of the original fracture (*), and union of an ipsilateral fibular synostosis (*white arrows*) performed during her initial hospitalization.

of local antibiotics (Table 4, tissue specimens).[46,68] Based on these studies, we exchange out all potentially contaminated surfaces in the surgical field before implantation/reconstruction (the double setup; Table 4): whenever possible, the wound is closed following débridement/lavage and then reprepared and redraped; soiled instruments, equipment, and tubing are removed from the room; surgical personnel change gowns and gloves; and clean instruments are brought to the field prior to initiating the reconstruction.

Each reconstruction must take into consideration the integrity and mechanical stability of the débrided bony segment(s) as well as deficiencies in the soft-tissue envelope that will affect treatment options and/or outcomes.[57,95] Efforts to secure definitive closure are warranted when vital structures (vessels, nerves, and tendons) will otherwise be exposed to injury or if the intended reconstruction will require either a sterile field (surgical implants) or an enhanced vascularity (surgical implants and/or massive cancellous bone grafts) (Fig. 3, *center*). Delayed primary closures are performed when a second-look débridement and/or an additional surgical sitting is needed (i.e., free-tissue transfers by a second team, Fig. 5, *below, left*, and *right*). To close the wound primarily, pathogen-specific antibiotics should be on board and all dead space eliminated by living tissue (transpositions, acute shortening to contact, and so on) and/or implants containing antimicrobial agents that are free to elute into the wound (antibiotic beads, gels, sponges, or spacers). The large amount of antibiotic eluted into a closed space filled with a highly concentrated antibiotic depot will eliminate planktonic and most sessile pathogens remaining in

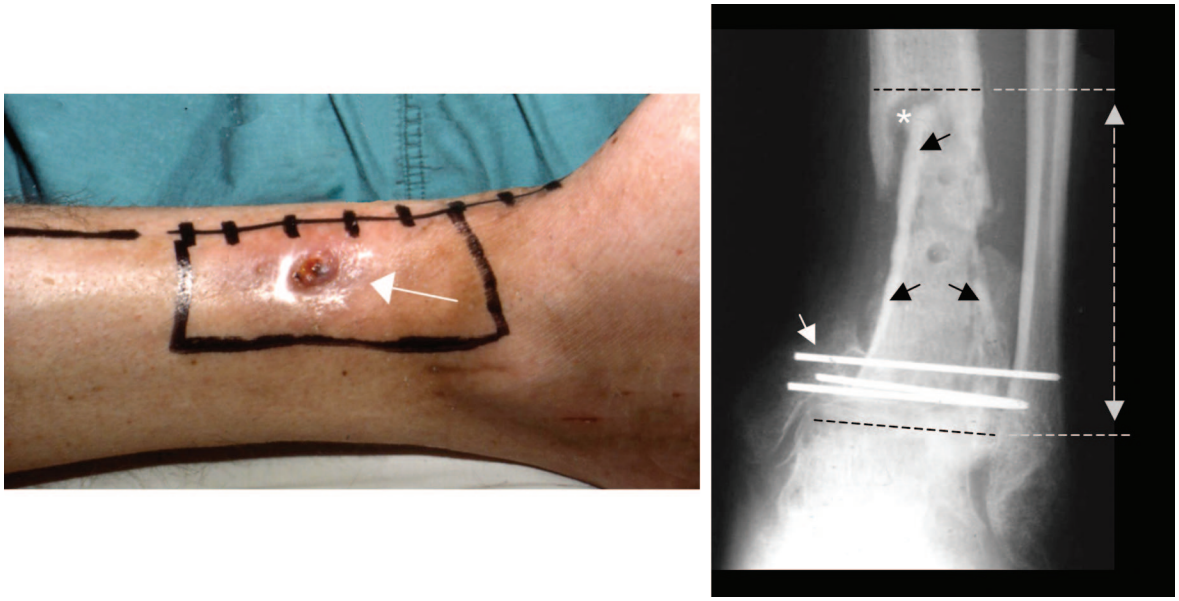

Fig. 6. (*Left*) A soft-tissue deficiency (*inked box*) and draining fistula (*white arrow*) were associated with an infected nonunion of the distal tibia. (*Right*) This anteroposterior radiograph shows pin fixation of the tibial plafond (*white arrows*), sequestered cortical fragments (*black arrows*), a nonunion (*), and the anticipated osseous resection for infection (*broken lines and arrows*).

the wound, safeguard closure, and maintain a workable space for later use (the spacer effect).[2,27,28,44] After wound healing and sufficient patient resuscitation, the depot is removed, the space reclaimed and the reconstruction performed following a double setup as if it is a clean surgical procedure.[39,46] When flaps fail and/or circumstances preclude their use, bone transport,[19,20,49,54,69–71] combined methods of shortening and lengthening,[51,66,67] and negative pressure–assisted closures are invaluable tools.[32]

Although a live, clean wound will heal by secondary intention, an open reconstruction has limitations: because the wound is exposed to contamination, all components of the reconstruction must also be live; there is limited surface area for closed bone grafting; exposures outside the open field are difficult and risky. Examples of these open methods include the use of external rather than internal fixation strategies, open cancellous bone grafting, acute limb shortening, vascularized bone flaps with or without skin paddle, and open methods of bone transport (Figs. 6 through 10).[54,96]

Antibiotic Selection

Once adequate tissue samples are secured for processing, broad-spectrum antibiotics are initiated. Coverage is based on knowledge from previous cultures, frozen sections taken at the time of surgery, and/or patient demographics. Thereafter, antibiotics are tailored to the sensitivities of *all* wound isolates and/or suspected microoragnisms. Ideal coverage would deliver an agent exhibiting a 1:1 kill ratio between its mean inhibitory concentration and mean bactericidal concentration for a particular pathogen and capable of safe serum concentrations at least six times the organism's mean bactericidal concentration.[3] The duration of coverage will vary according to the condition of the host and methods used.

Antibiotic Depots

Only high-dose antibiotic composites currently of poly-methyl-methacrylate or calcium sulfate are used to treat infection[27–31,44,95]; commercially available antibiotic-loaded cements (in the United States) are low-dose mixtures intended only for prophylactic use when revising prosthetic arthroplasties.[97] Although the use of $CaSO_4$ beads (OsteoSet beads; Wright Medical, Memphis, Tenn.; Stimulan beads, Biocomposites Inc.; Wilmington, N.C.) would obviate the need to later remove spent depots as potential foreign bodies, these biodegradable products, too, have drawbacks: (1) they are not sturdy enough to act as load-bearing spacers; (2) the amount and type of antibiotic(s) they can carry are limited due to intrinsic chemical intolerances preventing the product(s) from hardening; and (3) spontaneous wound fistulas can occur in as high as 20 to 28 percent of cases due to inflammatory by-products released during their degradation.[30]

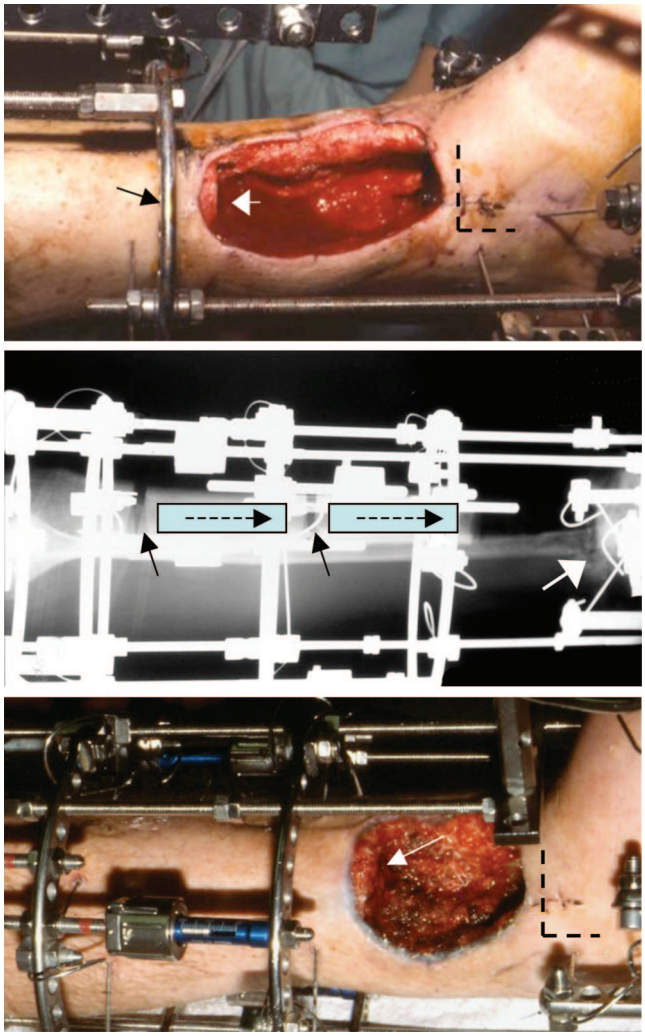

Fig. 7. (*Above*) A clinical photograph taken after debridement and external fixation shows a 12-cm composite defect, the distal-most transport ring of a ring fixator (*black arrow*), the docking end of the tibia (*white arrow*), and an outline of the recessed docking site at the talus (*dotted lines*). (*Center*) A lateral radiograph illustrates the bifocal frame used to transport (pull) two bony segments (*blue blocks*) away from their respective corticotomies (*black arrows*) toward (*dotted arrows*) the talar docking site (*white arrow*) to restore and reconstruct the tibia and fuse the ankle. (*Below*) In this clinical photograph taken after 8 weeks of transport, the wound is reduced in size by 50 percent; the docking end of the distal transport segment (*white arrow*) and the talar profile (*dashed lines*) are illustrated.

STAGE-DIRECTED LIMB SALVAGE

Medullary Osteomyelitis (Type I)

In my experience, there are few indications for an exclusively medical management of type I osteomyelitis other than in an asymptomatic lesion discovered coincidentally during a medical workup, infections caused by sensitive micobacteria and/or fungi, or in a hematogenous, vertebral osteomyelitis.[3,10] Otherwise, type I osteomyelitis requires surgical excision of the nidus through a cortical window. The approach is either direct (unroofing the lesion) or indirect (reaming through the canal) from above or below the nidus. Truncated lesions, at a diaphyseal-metaphyseal junction (isthmus and beyond), require combined reaming and unroofing to complete the excision (Fig. 3).

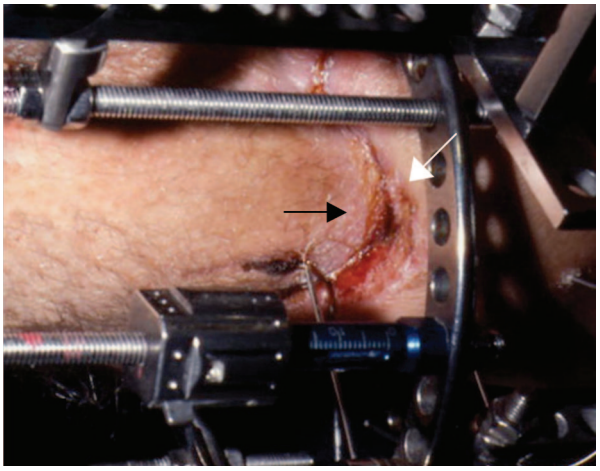

Fig. 8. This photograph was taken just before a docking with the talus (*white arrow*). One week earlier, opposing bony and soft-tissue margins were surgically contoured to maximize contact at the docking and avoid an undermining of the advancing skin margin (*black arrow*).

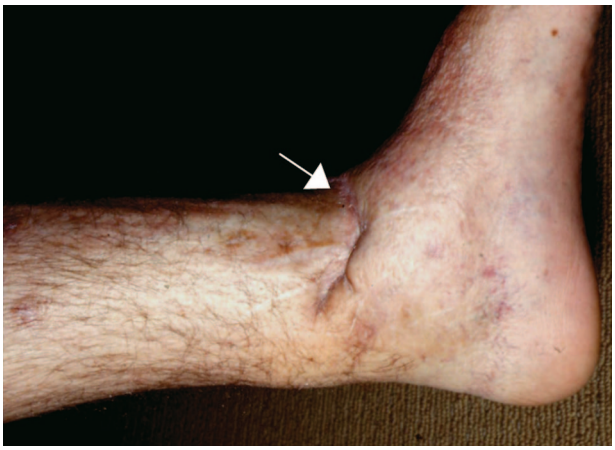

Fig. 9. Clinical photograph of healed wound after frame removal (*white arrow*).

Due to the limited involvement of investing soft tissues in type I lesions, the dead space remaining after débridement is usually confined to the medullary canal. A primary closure, an antibiotic depot within the canal and a short course of systemic antibiotics will, therefore, usually suffice for treatment. If the bone requires protection from an insufficiency fracture following treatment, various measures can be taken: ambulatory aids, a cast, a brace, external fixation, or antibiotic-coated hardware placed internally (Fig. 3, *center*).[31]

Superficial Osteomyelitis (Type II)

With a soft-tissue deficit in the etiology of this type of osteomyelitis, preoperative planning must focus on restoration of the soft-tissue envelope.

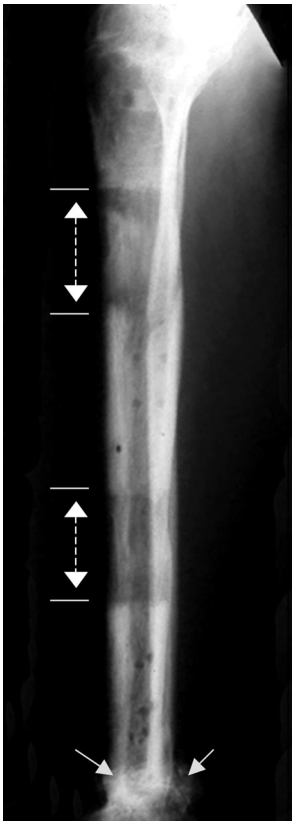

Fig. 10. A lateral radiograph shows consolidation of the two bony regenerates (*dotted arrows*), a solid ankle fusion (*white arrows*), and maintenance of both limb length and axial alignment.

The clinical examination, vascular indices, transcutaneous oxygen tensions, and angiograms are used to assess the deficit and potential pathways for its reversal. In the workup, the bony nidus is also mapped to establish the zone of injury and confirm that the nidus does not extend into the medullary canal to implicate a type III osteomyelitis (see below).

Surgical treatment begins with resection of soft tissues to viable/supple margins and the bone to the paprika sign. Thereafter, management will integrate knowledge of applied, vascular territories with experience translating perfusion deficits in the workup into durable coverage solutions. Local transpositions and free flaps are the most common methods used to restore and reconstruct type II osteomyelitis.

Localized Osteomyelitis (Type III)

Because type I and type II components of osteomyelitis are often concomitant in type III lesions, débridement commonly leads to a composite, hard, and soft-tissue deficit. If the excision will

Table 4. Treatment Algorithm for Adult Chronic Osteomyelitis, 2010*

I. Patient evaluation

II. Preoperative testing
–Laboratory testing: full metabolic panel, CBC with differential, coagulation panel, UA, ESR, CRP, colonization testing[61]
–Diagnostics: vascular indices; ultrasound; oxygen tensions (T_cPO_2)
–Radiology: plain films; magnetic resonance imaging, computed tomography, nuclear, and positron emission tomography scans; angiography studies
–Tissue specimens: cultures; histology sections; polymerase chain reaction pyrosequencing[62–64]

III. Clinical staging
–Anatomic type: I, medullary; II, superficial; III, localized; IV, diffuse
–Physiologic class: A-host, B-host, C-host

IV. Treatment format
–Limb salvage
–Amputation
–Palliation: C-hosts; no "treatment for cure"

V. Host optimization: reverse amenable comorbidities

VI. First surgery
A. One-stage treatment:
1. Débridement/tissue specimens/antibiotics (all treatment formats)
2. Dead space management (limb salvage, amputation)
Wound: secondary intention; primary versus delayed closures[65]
Bone: vascularized bone flaps; acute shortening[51,66,67]
Fixation: orthotics; external fixators
Depots: antibiotic beads[27–30,65]
B. First of multistage treatments:
1. Débridement/tissue specimens/systemic antibiotics
2. Double setup[46,68]: change instruments, repreparation and redraping, new gowns/gloves
3. Temporary fixation: external fixation; antibiotic-coated hardware
4. Dead space management:
Wound: secondary intention; primary versus delayed closures
Bone: bone transport[54,69–71]; vascularized bone flaps
Fixation: orthotics; external fixators; hardware (coated)[72–74]†
Depots: antibiotic beads, antibiotic spacers[72,75]

VII. Outpatient follow-up: wound surveillance; laboratory work (ESR/CRP); physical rehabilitation

VIII. Second surgery (second stage)
A. Definitive reconstruction:
1. Prophylactic antibiotics/hardware removal/débridement/tissue specimens (frozen biopsy negative—no inflammation)
2. Double setup: change instruments, repreparation and redraping, new gowns/gloves
3. Reconstruction:
Wound: primary closure
Bone: bone grafts, vascularized bone flaps, prosthetic joints
Fixation: orthotics; external or internal fixation, prosthetic joints
Depots: antibiotic beads, permanent spacers,[11,33] hardware (coated)†
B. Staged reconstruction no. 2:
1. Prophylactic antibiotics/hardware removal/débridement/tissue specimens (frozen biopsy positive—acute inflammation)
2. VI B (above) versus amputation

IX. Outpatient follow-up: wound surveillance; laboratory work (ESR/CRP); physical rehabilitation

X. Third surgery (third stage)
A. Definitive reconstruction: (frozen biopsy negative—no inflammation)
1. VIIIA (above)
B. Staged reconstruction no. 3: (frozen biopsy positive—acute inflammation)
1. VI B (above) versus amputation

XI. Fourth surgery: biologic reconstructions‡ (no hardware, no foreign bodies)

XII. Outpatient follow-up: wound surveillance; laboratory work (ESR/CRP); physical rehabilitation

CBC, complete blood count; UA, urinalysis; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.
*Treatment algorithm for adult osteomyelitis details the initial patient workup, clinical staging, format selection, and surgical protocols to manage both single and staged reconstructions. Patients receiving palliation or not requiring reconstruction following débridement complete a format after the initial intervention (see VI.A.2), whereas staged reconstructions follow interval treatment with both local and systemic antibiotics. All tissue specimens are cultured and examined histologically, and undergo quantitative polymerase chain reaction pyrosequencing.[62–64] Adapted with permission from Cierny G III, DiPasquale D. Adult osteomyelitis. In: Cierny G III, McLaren AC, Wongworawat MD, eds. *Orthopaedic Knowledge Update: Musculoskeletal Infection*. Rosemont, Ill.: American Academy of Orthopaedic Surgeons; 2009:135–153; Table 2, 144.
†Hardware (coated) includes a medullary rod, cortical plate, or prosthesis coated with antibiotic-impregnated bone cement.
‡Biologic reconstructions include bypass synostoses,[76,77] acute shortenings, bone transport, resection arthroplasties, and so on.

Table 5. Segmental Osseous Defects (n = 314): Incidence of Methods Used, 2000 to 2008*

	Incidence of Method (%)
Internal fixation/prosthetic joints/grafts	32
Distraction osteogenesis	25
External fixation bone grafts	16
Vascularized bone grafts	11
Permanent acrylic spacers	8

*The relative incidence of methods used to reconstruct 314 consecutively treated segmental defects is shown for patients with chronic osteomyelitis treated from 2000 to 2008. The permanent spacers used in this series were all composites of various antibiotic-impregnated bone cements (poly-methyl-methacrylate) reinforced with a nail, pin, or plate.

be of such a magnitude as to threaten the mechanical stability of the remaining bony segment, the limb may be prophylactically stabilized with use of an osseous transfer,[58,76,77] an external fixator,[98] or stabilized in situ, following débridement, with an antibiotic depot (antibiotic-coated implant/spacers, antibiotic rods, and so on) (Figs. 2 and 3). Soft-tissue defects are addressed as discussed for type II lesions. If osseous reconstruction is indicated or a significant dead space exists following débridement, reconstruction will usually follow a course of local antibiotic therapy (Table 4).

Diffuse Osteomyelitis (Type IV)

Débridement of a type IV lesion always culminates in an unstable bony segment (i.e., a resection arthroplasty, nonunion, or segmental defect). Instability, an insidious zone of injury, bone loss, and a predominantly compromised (B-host) patient population (Table 3) make type IV lesions the most difficult to treat. Nearly all treatment protocols call for a staged reconstruction (Table 4) with the reconstruction later taking place as a clean procedure. Alternatively, soft-tissue restoration and bony reconstruction can take place simultaneously in the form of a vascularized (free) bone graft with skin paddle (Figs. 4 and 5), an open bone transport (Figs. 6 through 10), or various combinations of shortening and lengthening. A historical profile of methods used by the author to reconstruct a consecutive series of segmental, long-bone defects is found in Table 5.

RESULTS OF TREATMENT

Today, 91 percent of the patients referred to our treatment center in San Diego enter limb salvage protocols, and 63 percent of the patients treated are classified B-hosts (Fig. 11). Despite these trends, the long-term outcomes have improved as efforts to downsize patient risk have

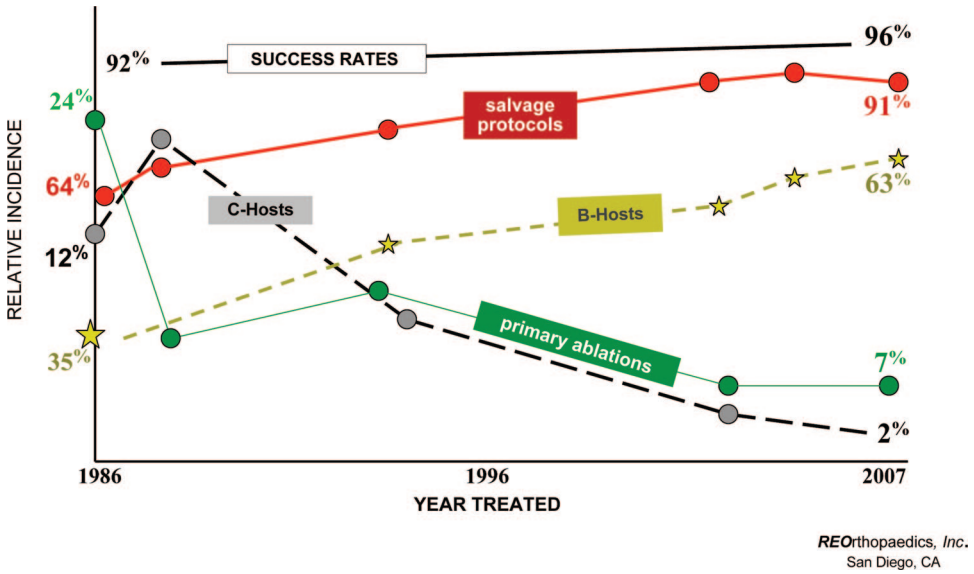

Fig. 11. Osteomyelitis: treatment outcomes and trends (1986 to 2007). A chronologic history of 1672 cases of chronic osteomyelitis profiling infection-free outcomes (2-year success rates), the percentage of B-hosts in the treatment population, and the utilization incidence of the three treatment formats: palliation (C-hosts), primary ablation (amputation), or a limb salvage attempt. Despite a twofold increase in the B-host cohort and a 26 percent rise in limb salvage attempts, success rates remained constant, due in part to our experience and the evolution of protocols to decrease patient risk and thereby broaden our selection criteria for curative treatment (see text).

evolved. These efforts include a multidisciplinary team approach to optimize the host response, identify and treat the entire biofilm colony, match the reconstruction to host and/or surgeon restraints, and maintain vigilant wound surveillance throughout treatment.

The long-term outcomes for 2207 sites of chronic osteomyelitis treated 1981 through 2007 are found in Table 3. In that series, there were 108 C-hosts, 1898 limb-salvage protocols, and 230 primary ablations. With success defined "an infection-free, functional reconstruction at the 2-year follow-up," the overall success rate following the initial treatment was 85 percent: 96 percent in A-hosts , 74 percent in B-hosts, 84 percent following limb salvage, and 91 percent following amputation, respectively. Of the 319 failures, there were aseptic nonunions (43 percent), wound sloughs (28 percent), an unanticipated impairment (15 percent), recurrent sepsis (12 percent), and deaths (2 percent) within a year of treatment. Of patients requiring retreatment, 82 percent ultimately had a successful outcome, elevating the overall success rate to 95 percent at 2 years (99 percent in A-hosts; 90 percent in B-hosts). There were no significant outcome differences found when comparing internal versus external methods of fixation, mono-microbial versus polymicrobial infections, oral versus parenteral antibiotic coverage, or sensitive versus resistant pathogens. On the contrary, when a surgical implant functioned to both restore and stabilize stage IVB osteomyelitis (i.e., an intercalary allograft or a prosthetic total joint), the overall success rate dropped 6 percent when compared with stage IVB reconstructions wherein restoration was biologic (tissue) and/or the implant could be removed without affecting the outcome (i.e., removal of an antibiotic rod following incorporation of cancellous grafts). This later finding again emphasizes the negative impact of local (hardware) and systemic comorbidities on outcomes.

George Cierny, III, M.D.
REOrthopaedics, Inc.
7910 Frost Street, Suite 120
San Diego, Calif. 92123
gcierny@reorthopaedics.com

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