

# *T H E C L A S S I C*

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## *A Clinical Staging System for Adult Osteomyelitis*

\_\_\_\_ **George Cierny III, MD; Jon T. Mader, MD;** \_\_\_\_  
**and Johan J. Penninck, MD**

**J**on Terry Mader (Fig 1) was born on March 21, 1944 in Madison, WI. He earned his BA and MD degrees at Wabash College at Indiana University in 1966 and 1970, respectively. He trained in Internal Medicine at the University of Texas Medical Branch in Galveston, TX and made his career there in the Division of Infectious Disease in the Department of Internal Medicine. At the time of his death on October 25, 2002, he had risen to the positions of Professor of Medicine, Professor of Pathology, and Adjunct Professor of Orthopaedic Surgery. During his life, he published more than 145 peer-reviewed papers on osteomyelitis, antibiotic therapy, hyperbaric oxygen, joint infections, the foot in patients with diabetes, and the use of the Ilizarov technique for the treatment of musculoskeletal infections. He also was the principle investigator in numerous funded research projects and the coauthor of *Musculo-Skeletal Infection*. He died when the book was in its final stages of production.

In addition, Dr. Mader was a gifted athlete, Eagle Scout, captain in the U.S. Naval Reserve, and regarded with respect and affection by his patients and colleagues.

*Henry H. Sherk, MD*

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### **INTRODUCTION**

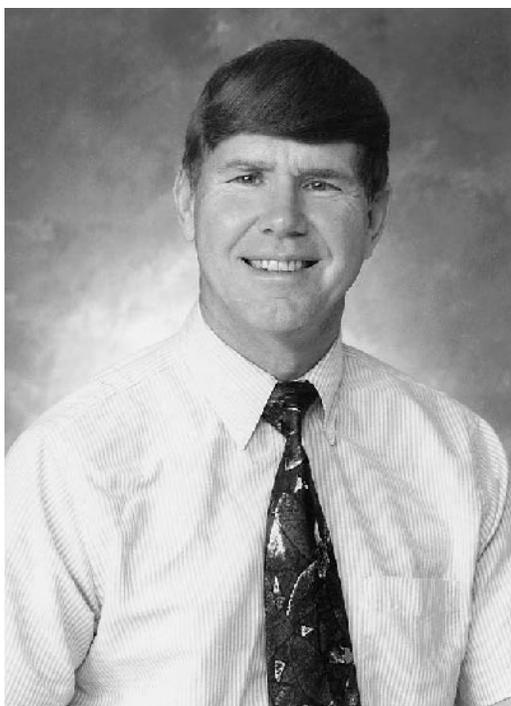
At the University of Texas Medical Branch (UTMB) in Galveston, Texas, the adult osteomyelitis service now treats ten new cases of osteomyelitis each month. Since January 1981, 425 patients have been evaluated and 900 procedures performed to treat 240 lesions.

In our experience, the treatment of adult osteomyelitis is influenced by four factors: the condition of the host, the functional impairment caused by the disease, the site of involvement, and the extent of bony necrosis. Without reference to these factors, it is difficult to compare the results of different treatment protocols<sup>1-5</sup> and the effectiveness of new therapeutic modalities.<sup>6-9</sup>

The UTMB classification of adult osteomyelitis<sup>10</sup> combines four anatomic types

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**Fig 1.** John T. Mader, MD

(the disease) with three physiologic classes (the host) to define 12 clinical stages (Table I). A distinction between the acute and chronic process has not been necessary.<sup>11</sup> The classification system incorporates the four prognostic factors, delineates treatment for progressive stages of the disease, and provides guidelines for the use of adjunctive therapies.

#### **THE UTMB TREATMENT PROTOCOL FOR ADULT OSTEOMYELITIS (TABLE II)**

The UTMB prospective study of adult osteomyelitis began in June 1981. The methods

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(Reprinted with permission from Cierny III G, Mader JT, Penninck JJ: A clinical staging system for adult Osteomyelitis. *Contemp Orthop* 10:17-37, 1985.)

**TABLE I The UTMB Staging System for Adult Osteomyelitis**

<b>Anatomic Type</b>		
Type I	—	Medullary osteomyelitis
Type II	—	Superficial osteomyelitis
Type III	—	Localized osteomyelitis
Type IV	—	Diffuse osteomyelitis
<b>Physiologic Class</b>		
A-Host	—	Good immune system and delivery
B-Host	—	Compromised locally (B <sup>L</sup> ) or Systemically (B <sup>S</sup> )
C-Host	—	Requires suppressive or no treatment; minimal disability; treatment worse than disease; not a surgical candidate

#### **Clinical Stage**

Type + Class = Clinical Stage

Example:

Stage IVB<sup>S</sup> osteomyelitis = a diffuse lesion in a systemically compromised host

have remained unchanged except for the antibiotic recommendations for emergency coverage and for acrylic bead mixtures. The protocol itself is unique: patient selection and medical/surgical treatment are predetermined by the clinical staging system; the preoperative antibiotic program is based on outpatient biopsy data, and the patient follow-up system correlates a clinical and biologic response to therapy.

#### **Patient Evaluation**

*Condition of the host.* Host deficiencies influence treatment options, prognosis, and the interpretation of treatment results. Following debridement surgery, the host must be able to impede infection, resist contamination, heal surgical wounds, and tolerate the metabolic stress of sequential surgeries. A list of factors influencing the ability of the host to elicit an effective response to infection and treatment is found in Table III. Local factors lead to a vascular compromise of bone and soft tissue. Systemic compromise affects immune surveillance, metabolism, leukocyte function, and/or large vessel disease. Local and sys-

temic factors may combine (e.g., diabetes mellitus).

*Disability of the patient.* The functional impairment caused by the disease, the reconstruction options, and the metabolic consequences of aggressive therapy influence the selection of treatment candidates. A draining sinus with minimal pain and/or dysfunction is not, by itself, an indication for surgical treatment. At times, the procedures required to arrest or palliate the disease are of such magnitude that treatment can lead to loss of function, limb, or life. In these latter instances, *quality of life* is the major factor influencing the decision to pursue therapy.

*Physiologic Classification (Host).* At UTMB, the condition of the host and the relative disability caused by the disease are combined in a physiologic classification (Table I). A patient with a normal physiologic response to infection and surgery is designated an A-Host; a compromised patient is classified a B-Host and will have either local ( $B^L$ ), systemic ( $B^S$ ) or combined ( $B^{L,S}$ ) deficiency in wound healing (Table III). When the treatment or results of treatment are more compromising to the patient than the disability caused by the disease itself, the patient is classified a C-Host. Thus, the selection of surgical candidates may vary from institution to institution until there has been a standardization of concepts, methods, and techniques.

### Disease Assessment

Persistent osteomyelitis is a surgical disease. Since debridement is the unchallenged cornerstone of successful therapy, a classification of osteomyelitis based on the site of necrosis will have specific implications for surgical management. Using such a system, four anatomically defined types of osteomyelitis become apparent: medullary, superficial, localized, and diffuse (Fig. 1). However, the condition of the host, regional vascularity, local milieu, and extent of necrosis will influence the natural history of the disease.<sup>11</sup>

*Anatomic classification.* The medullary and superficial types of osteomyelitis share a

pathophysiologic component: soft tissue compromise. In *medullary osteomyelitis* the primary lesion is endosteal. The etiology of the disease is variable but the nidus remains constant: ischemic scar, chronic granulations, and splinter sequestra within the medullary canal.

In *superficial osteomyelitis* the problem is on the surface of the bone. This is a true contiguous focus lesion. A compromised soft tissue envelope either begins or perpetuates an exposure of the bone. The involved surface may be on an old saucerization, and healed Pappineau graft, the prominent callus of a healed open fracture, or the metatarsal head in a neuropathic foot ulcer.

The hallmark of *localized osteomyelitis* is full-thickness, cortical sequestration and/or cavitation. It is a discrete lesion within a stable bony segment. Although localized osteomyelitis usually follows trauma, it often has the combined features of medullary and superficial osteomyelitis and may even result as an extension of either of these two entities.

*Diffuse osteomyelitis* is a permeative, circumferential, or through-and-through disease of hard and soft tissue. In this type, an intercalary segment of the skeletal unit must be removed in order to resect all the compromised tissue. Instability is present either before or after a thorough debridement. Stabilization is an essential factor in the treatment and separates the diffuse lesion from the other types of osteomyelitis. Infected nonunions, end-stage septic joints, and through-and-through metaphyseal/epiphyseal lesions of the proximal femur are examples of this type of osteomyelitis.

*Clinical staging of adult osteomyelitis.* The four anatomic types of osteomyelitis are numerically ordered according to the complexity of the disease and/or its treatment: I—medullary; II—superficial; III—localized; IV—diffuse (Table I). In our classification system, the anatomic type (I-IV) is combined with the physiologic class (A-C) to designate one of 12 clinical stages of adult osteomyelitis. The clinical stage can change during the course of treatment (Table IV).

**TABLE II UTMB Treatment Protocol for Adult Osteomyelitis**

- 
- I. PATIENT EVALUATION
    - A. Old records and films documenting treatment and history
    - B. Physical examination, review of system, social history, medical history
    - C. Laboratory values: CBC, platelet count, sedimentation rate (Wintrobe), serum protein electrophoresis, total iron binding capacity, PT and PTT, 12/60, serum iron, transcutaneous oxygen tensions (direct Coombs—investigational antibiotics)
  - II. PHYSIOLOGIC CLASSIFICATION (HOST)
    - A. A-Host: Good systemic defenses; good local vascularity
    - B. B-Host: Local compromise (B<sup>L</sup>) systemic compromise (B<sup>S</sup>)
    - C. C-Host: Minimal disability; not a surgical candidate; treatment worse than the disease
  - III. DISEASE ASSESSMENT
    - A. Anatomic classification:
      - 1) Type I: Medullary osteomyelitis
      - 2) Type II: Superficial osteomyelitis
      - 3) Type III: Localized osteomyelitis
      - 4) Type IV: Diffuse osteomyelitis
    - B. Physical examination:
      - 1) Sinus tracts, old scars, condition of extremity, reconstruction options
      - 2) X-rays, tomograms, CT scans (if indicated)
      - 3) Arteriograms: Complicated anatomic status, microvascular reconstruction planned
      - 4) Technetium 99 (<sup>99m</sup>Tc) bone scan and indium-111 chloride scan<sup>12</sup> (whole body and pinhole analysis)
  - IV. IDENTIFICATION OF ORGANISMS
    - A. Based on biopsy of deep granulations
    - B. Aerobic and anaerobic cultures taken
    - C. Histologic correlation (document tissue sampling, validate culture results, confirm pathogenesis)
  - V. SELECTION AND ADMINISTRATION OF ANTIBIOTICS
    - A. Quantitative sensitivity testing by the tube-dilution technique
    - B. Antibiotic selection: Low MIC/MBC activity relative to expected serum concentration. Optimal: MIC at least eight times less than the expected serum concentration.
    - C. Antibiotics and patient carefully monitored seven to ten days prior to surgery:
      - 1) Assure adequate antibiotic coverage
      - 2) Check host tolerance
      - 3) Marginate the wound
      - 4) Monitor social adjustment to treatment
      - 5) Support host alteration
    - D. Unless the patient is seriously ill, the antibiotics are not started until cultures and sensitivities have been returned. In these instances, and in a severely compromised host, Ticarcillin 3gm q4h, Cefazolin 2gm q6h, and Gentamicin (5mg/kg/day) are begun empirically after the biopsy. This antibiotic regime covers the three most common isolates in our series: *Staphylococcus aureus*, *Pseudomonas aeruginosa* and anaerobic organisms. The antibiotics are modified by culture and sensitivity results.
    - E. Hickman catheter placement<sup>13</sup>
    - F. Antibiotics are given 42 days after the last major debridement surgery
    - G. Laboratory values and antibiotic levels (when indicated) checked weekly or every other week during therapy
  - VI. METHODS OF HOST ALTERATION
    - A. Patient education—No smoking
    - B. Nutritional supplementation:
      - 1) Malnutrition
      - 2) Alcohol use
      - 3) Immune compromise
      - 4) Renal/liver failure
      - 5) Diabetes
    - C. Hyperbaric oxygen: advanced age, chronic hypoxia, arteritis, major vessel disease, extensive scarring, radiation fibrosis, extensive granulation beds

(continues)

**TABLE II UTMB Treatment Protocol for Adult Osteomyelitis (Continued)**

- D. Special considerations:
- 1) Local compromise: pressure garments, tissue transfers
  - 2) Pressure sores: force distribution
  - 3) Diabetes: blood-glucose control
  - 4) Immune compromise: gamma globulin therapy
  - 6) Major vessel disease: arterial bypass surgery
  - 7) Chemical suppression: discontinue or alter medications
  - 8) Sepsis/toxicity: Emergency decompression/drainage
- VII. SURGICAL TREATMENT OF OSTEOMYELITIS
- A. First look:
- 1) Seven to ten days after initiation of appropriate antibiotics
  - 2) Debridement approach direct and atraumatic
  - 3) Stabilization provided prior to or after debridement when indicated
  - 4) All avascular scar and bone removed
  - 5) Biopsies and cultures obtained of all foci encountered
  - 6) Wound left open (stent dressing) Exceptions: compromised hosts (B<sup>S</sup>); ankle, hand, spine; Type II lesions (primary soft tissue reconstruction and/or host alteration); minimal necrosis osteomyelitis
  - 7) Cultures checked and antibiotics adjusted if necessary
- B. Second look:
- 1) Performed five to seven days after first debridement
  - 2) Biopsies and cultures repeated
  - 3) Dead space management:
    - a. Open cancellous grafting (Papineau technique)<sup>14</sup>
    - b. Primary closure with local tissue (+/- cancellous grafts)
    - c. Primary closure with transferred tissues (+/- cancellous grafts)
    - d. Primary closure over antibiotic impregnated beads
  - 4) Cultures checked and antibiotics adjusted if necessary
- VIII. FOLLOW-UP OF ALL PATIENTS
- A. Monthly laboratory values for six months (CBC, ESR, 12/60)
  - B. Indium scans and laboratory values repeated every six months or until normal
  - C. Repeat x-rays as indicated to follow union and graft incorporation

**Microbiology**

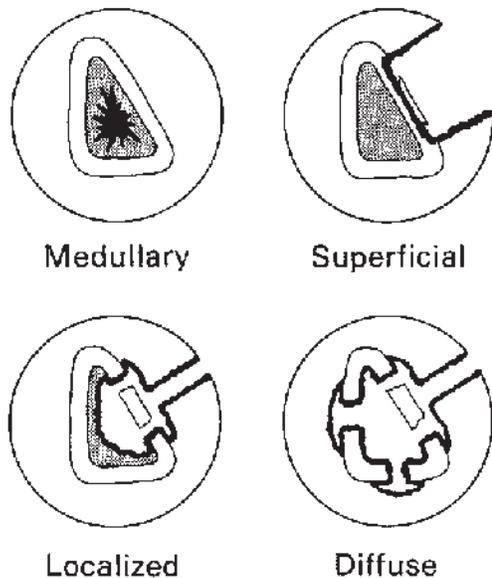
The bacteria responsible for the infection may be reliably isolated in two ways: preoperative

**TABLE III Systemic or Local Factors that Affect Immune Surveillance, Metabolism, and Local Vascularity**

Systemic <sup>(S)</sup>	Local <sup>(L)</sup>
Malnutrition	Chronic lymphedema
Renal, liver failure	Venous stasis
Alcohol abuse	Major vessel compromise
Immune deficiency	Arteritis
Chronic hypoxia	Extensive scarring
Malignancy	Radiation fibrosis
Diabetes mellitus	
Extremes of age	
Steroid therapy	
Tobacco abuse	

biopsies, or from tissue sampled at the time of debridement surgery.<sup>15</sup> All isolated pockets of granulation tissue or necrosis must be sampled. Whenever possible, an antibiotic regime tailored to the sensitivities of all organisms isolated from biopsy material obtained in the outpatient setting is begun prior to debridement.<sup>16</sup>

At the first and all subsequent debridements, multiple biopsies are obtained again for aerobic/anaerobic cultures and histologic evaluation. During therapy, antibiotic coverage may be changed or modified on the basis of clinical findings, serial debridement isolates and their sensitivities, inadequate serum bactericidal levels, abnormal laboratory studies, and/or patient intolerance. Antibiotics are given for six weeks after the last major debridement surgery.<sup>16</sup> At UTMB, all isolated



**Fig. 1** Anatomic classification of adult osteomyelitis.

organisms are placed in defibrinated sheep blood and stored at  $-70^{\circ}\text{C}$  for future reference. Outpatient intravenous antibiotic therapy is utilized once serum bactericidal levels and/or surgical wounds permit.<sup>13</sup>

### Surgical Treatment

Osteomyelitis surgery is disciplined and demanding. The average number of operations for a limb-salvage patient in our 1983 series as 3.8 procedures. Depending on the clinical stage of the disease and the planned reconstruction, the diagnostic biopsies, debride-

ments, and reconstructions may be combined or performed separately. Biopsies are usually performed in the outpatient setting under local anesthesia. The organisms are thereby established, and questionable areas of involvement are assessed histologically.

**Debridement.** As in musculoskeletal tumor surgery, careful preoperative planning is critical to achieve a high rate of success and to minimize wound complications in the patient with osteomyelitis. The debridement is direct, atraumatic, and executed with the reconstruction in mind. Whenever possible, the incisions are laced between myocutaneous territories, at times disregarding previous incisions. Soft tissue retraction is minimized by careful wound planning. Sinus tracts are excised if present for more than one year.

All dead or ischemic hard and soft tissues are excised unless a palliative procedure has been chosen from the start. The extent of the debridement is predictable from the preliminary assessment.<sup>16</sup> If complete excision will threaten stability, external fixation and/or a bypass graft may be necessary prior to or during debridement surgery. At UTMB, the instruments used in the debridement procedures include scalpels, curettes, straight stem and angled dental mirrors, and a pneumatic bone scalpel. Because of the speed and gentle efficiency of this pneumatic system, osteotomes are rarely used. Tetracycline labeling, fluorescein, and other dyes have not been useful.

The debridement process begins in a centrifugal fashion. This technique retains an outer ring of bone that shares its circulation with the attached soft tissues. This shell of bleeding bone is dressed with either bone grafts, antibiotic beads, or soft tissue at the time on reconstruction. The residual cortical and cancellous bone must bleed uniformly (Fig. 6). Definitive wound management usually takes place five to seven days after the last debridement. In the interim, the wound usually is left open.

**Dead space management.** The techniques of managing the dead space created by debridement surgery are illustrated in Fig. 7.

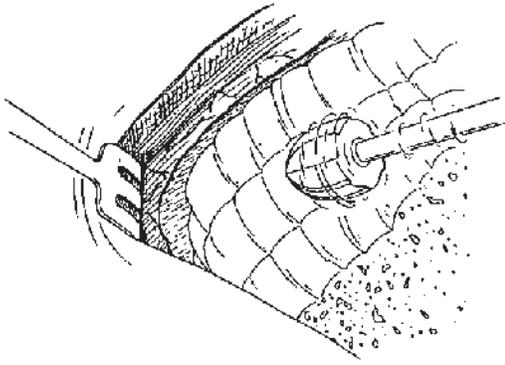
**TABLE IV** Clinical Stage Manipulation

#### Physiologic Considerations: (Examples)

B-Host	→	A-Host (Hyperalimentation)
B-Host	→	C-Host (Orthosis/antibiotics)
A-Host	→	C-Host (Compression garment)
A-Host	→	B-Host (Drug toxicity)

#### Anatomic Considerations:

Stages III/IV	→	Stage I (IM rod)
Stage IV	→	Stage III (Bypass or union)
Stages III/IV	→	Stage III (Papineau ulceration)
Stage I/II	→	Stages III/IV; stage progression in the B-Host

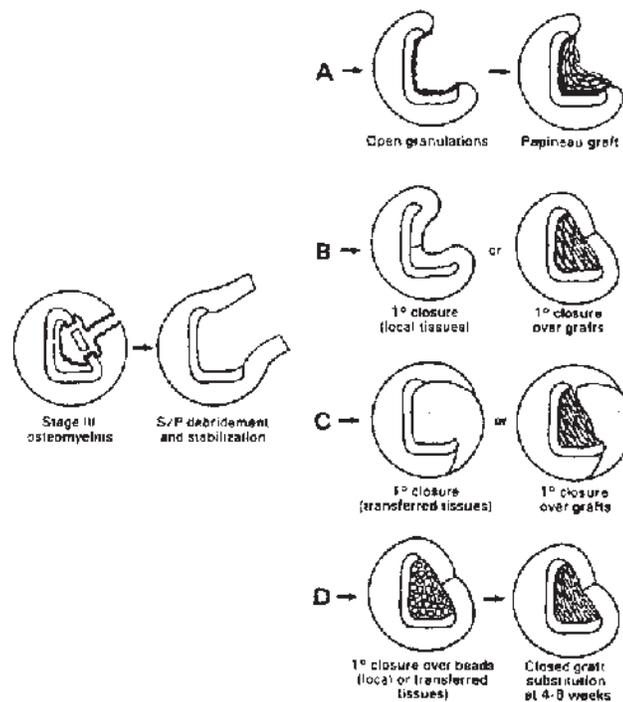


**Fig. 6** Tangential excision with the bone scalpel is carried down to uniform haversian or cancellous bleeding (the paprika sign<sup>17</sup>).

Secondary intention healing is discouraged; the scar tissue that fills the defect later becomes avascular and may lead to recurrent drainage. Similarly, suction/drainage systems are rarely used. The goal of surgery is to replace dead bone and scar with durable, vascularized tissue.

A complete wound closure is secured whenever possible. Cancellous bone grafts are placed beneath local or transferred tissues when structural augmentation is necessary or a significant dead space will otherwise persist in the bone. Bypass grafts are performed when an in situ reconstruction will prove inadequate or is not feasible (Fig. 8). Open cancellous grafts<sup>14</sup> are used sparingly as the epithelial coverage is not durable and may lead to superficial ulceration following minor trauma or persistent venous stasis.<sup>18</sup> They are, however, simple to do, effective, and particularly useful when a free or local tissue transfer is not an option.

Antibiotic-impregnated acrylic beads<sup>6</sup> have been used to sterilize and/or temporarily maintain a dead space created by debridement surgery. In our experience, any patient-compatible, powdered antibiotic may be safely delivered in this manner; it must first be adequately pulverized and then thoroughly mixed with the powdered cement prior to adding the monomer.<sup>10</sup> Thermal stability of the antibiotic(s) is not necessary when the beads are



**Fig. 7** Methods of dead space management.

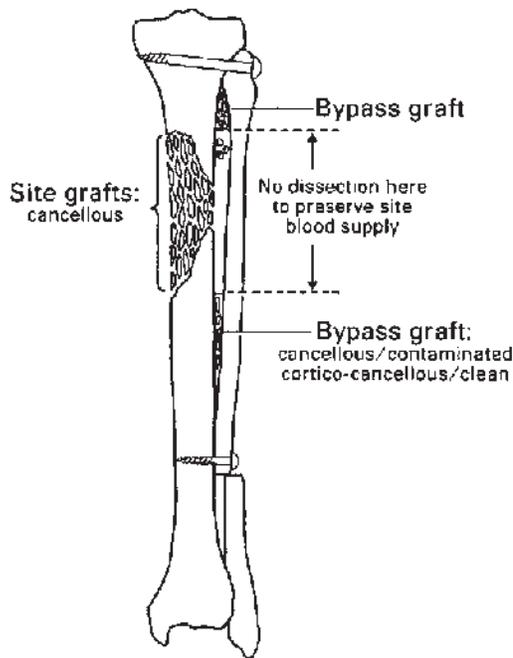


Fig. 8 Bone graft techniques.

fashioned in the dough phase.<sup>19</sup> Two or three antibiotics may be combined in a single mix. Before using this technique, the debridement must first be thorough and the wound flora ideally sensitive to the antibiotic mixed with the cement. The beads usually are removed within two to four weeks and replaced with cancellous bone grafts. If strung on a line,<sup>6,20</sup> the beads are removed in ten to 12 days. The five antibiotics most commonly used in beads and their mixing ratios are listed in Table V. If the volumetric ratio of the powders exceeds 24cc/120cc (antibiotic/40gm cement), the cement will not harden reliably.

### Application

*Stage IA,B,C: Medullary Osteomyelitis (Fig. 9A, 9B)*

Once the medullary process extends into the soft tissues, the usefulness of medical management alone will depend on the site and extent of the process, the physiologic class of the patient, and the functional disability expected from disease and treatment. The majority of the patients with

**TABLE V Antibiotic Bead Cocktails  
June 1984**

Cephalosporins	
Cefazolin	3-6gm
Moxalactam	3-6gm
Cefotaxime	5-10gm
Tobramycin	4.8-9.6gm
Vancomycin	2-4gm
Ticarcillin	6-12gm
Lower dosages used in combinations or 1/2 pk (PMMA) preparations.	

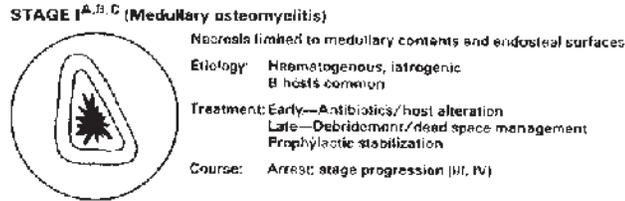
Stage I osteomyelitis are systemically compromised hosts and suffer stage progression to Stage IIIB or IVB. When extension occurs, the process usually becomes intraarticular and the subchondral bone and articular cartilages sequester.

Protection (stick, cast, or orthosis) usually is necessary until remodeling occurs and/or bone grafts mature. This stage often is debrided and closed on the first look. Closure over the obliterated dead space frequently can be achieved with simple approximation of the soft tissues. The organism is obtained preoperatively, and wound closure is protected by appropriate antibiotic coverage.<sup>23</sup>

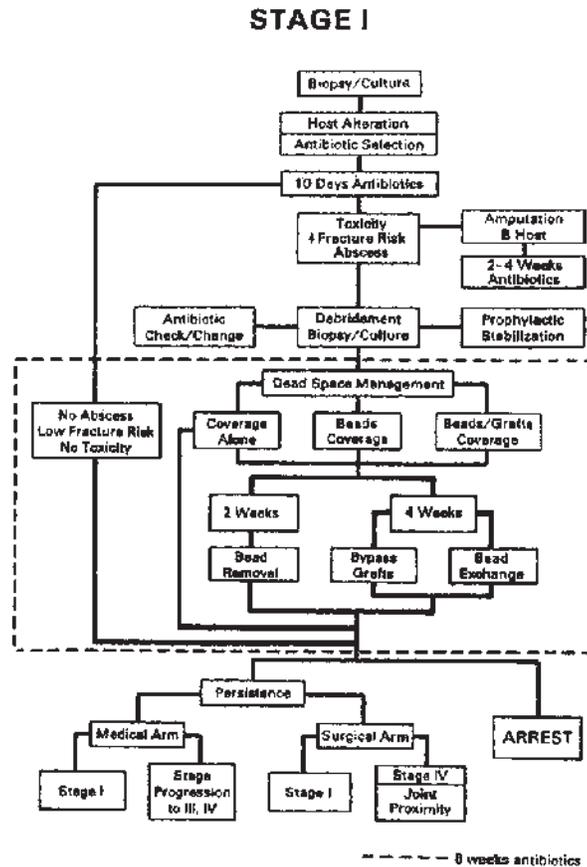
To completely excise the infected contents of the medullary canal, a small unroofing of the cortex is required. This *minimal access* to the canal is sufficient for an extended curettage<sup>21</sup> and/or medullary reaming.<sup>22</sup> When using the latter technique, the disease must be situated within an isthmus or limited to the tract of an intramedullary nail, otherwise a combination of reaming and unroofing will be necessary. The bony entry should be placed slightly askew of the anticipated soft tissue closure to prevent inversion of the wound margins and persistent drainage. The operations for treating Stage I lesions apply whenever medullary involvement is present (Fig. 10).

### Stage IIA,B,C: Superficial Osteomyelitis (Fig. 11A, 11B)

The management of superficial osteomyelitis requires considerable experience with complex soft tissue transfers. Ischemic soft tissues



**Fig. 9A** Stage I classification of osteomyelitis.

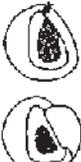


**Fig. 9B** Treatment algorithm for Stages IA and IB.

are excised and the exposed bony surface is tangentially removed (decortication) until the paprika sign<sup>17</sup> is observed. A pedicle flap or free tissue transfer is performed at the same sitting or as a delayed procedure. The key to the success of this method is a live and clean prior to soft tissue coverage. (sic)

If the Stage II lesion is caught in the acute or subacute stage, treatment with pressure gar-

ments, orthoses, and/or local wound care may be sufficient. The wounds heal unless tissue oxygen tensions, local mechanical factors, or patient cooperation are not favorable. At UTMB, the septic joint is classified as a superficial osteomyelitis (osteochondritis). The soft tissue component of the process is the compromised synovium. The disease progresses and responds to treatment as do the other Stage II

<b>Surgical Components</b>	
<b>STAGE I</b>  <b>Minimal access</b>	
<b>STAGE II</b>  <b>Decortication</b>	
<b>STAGE III</b>  <b>Saucerization</b>	
<b>STAGE IV</b>  <b>Stabilization</b>	

**Fig. 10** Debridement techniques are unique to each stage but combine as the complexity of the disease increases.

lesions; they resolve with early host alteration and progress to cavitation and full-thickness sequestration (subchondral), if untreated.

The operations for treating Stage II lesions apply whenever superficial involvement is present (Stage II, III, IV).

### **Stage IIIA,B,C: Localized Osteomyelitis (Fig. 12A, 12B)**

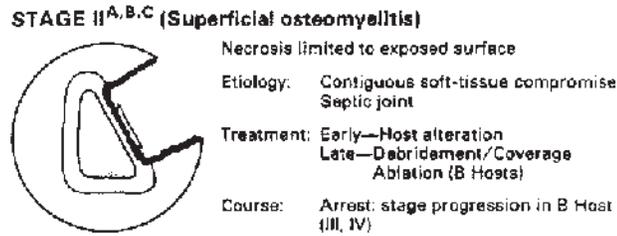
The hallmark of this process is cortical sequestration and cavitation. Debridement surgery usually involves sequestrectomy, saucerization, medullary decompression, scar excision, and superficial decortication. The reconstruction will depend on the dead space created, the integrity of the residual bone, and the site of involvement. The procedures include viable hard and soft tissue transfers, cancellous bone grafts, bypass procedures, and simple wound approximation. Prophylactic stabilization is provided when the extent of the debridement places the bone at risk for fracture.<sup>24,25</sup>

Most Stage III lesions are posttraumatic and satellite or skip foci of infection may be present within the treatment zone secondary to prior surgeries (internal or external fixations). The preliminary evaluation (protocol steps I, II, III) usually identifies these lesions and directs an appropriate staging and treatment.<sup>16</sup> The operations for treating Stage III lesions apply whenever cavitation occurs (Stage III, IV) and often include procedures from Stage I and II treatment protocols (Fig. 11B)

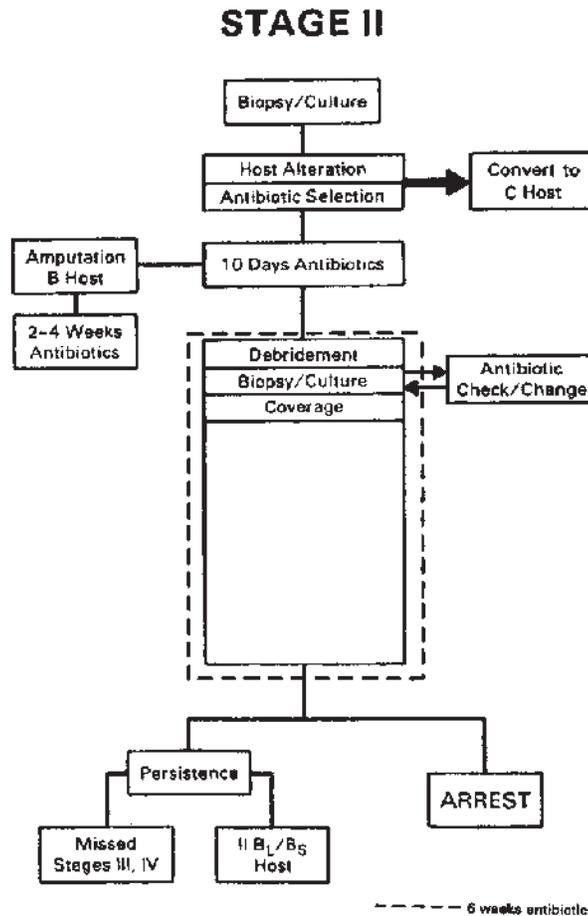
### **Stage IVA,B,C: Diffuse Osteomyelitis (Fig. 13A, 13B)**

B-Hosts and segmental defects are common in this stage and potentiate the risk for developing a wound healing disturbance, i.e., nonunion, central bone graft necrosis, opportunistic infections, and stress fractures. The techniques used in managing Stage IV osteomyelitis are conceived and executed with a *stabilization* procedure in mind.

The preoperative planning must be precise and exhaustive to avoid tissue devitalization, unnecessary hardware in the wound, and in inefficient use of cancellous bone reserves. External fixation devices, medullary rods, and cortical plates are used selectively and are listed according to our preference and frequency of application. In our experience, external fixation is the safest and most versatile system.



**Fig. 11A** Stage II classification of osteomyelitis.



**Fig. 11B** Treatment algorithm for Stages IIA and IIB

**Extent of Necrosis**

When the host is unable to resorb or expel infected, nonviable tissues, the infection becomes chronic. The nidus will persist until the pathophysiology of the process is reversed by appropriate therapy. Although debridement surgery usually is the treatment of choice, some lesions consistently respond to alternate

forms of therapy. The common denominator in these wounds is infected scar tissue, not bone sequestration. This entity is called *minimal necrosis osteomyelitis*. Manipulation of host parameters frequently is the treatment of choice rather than debridement surgery.

*Vertebral osteomyelitis.* Hematogenous osteomyelitis of the vertebral column is an

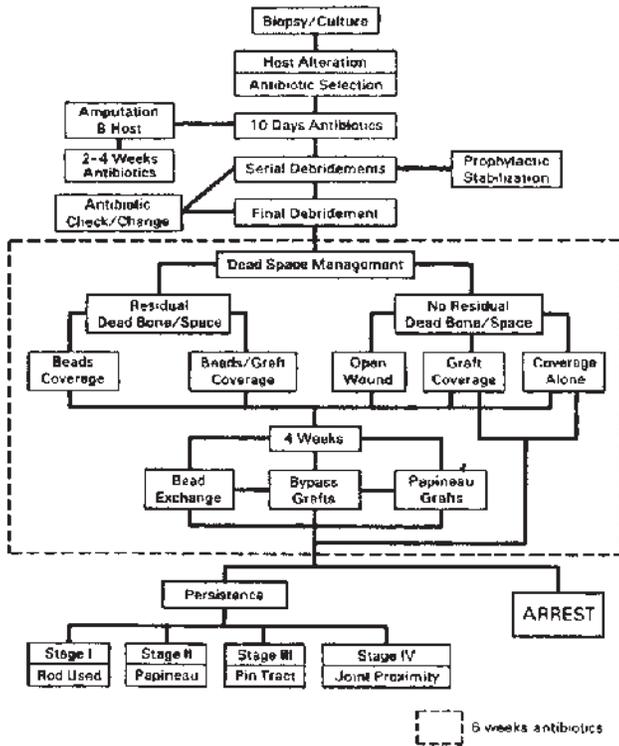
**STAGE III<sup>A,B,C</sup> (Localized osteomyelitis)**



Well-marginated  
Stable before and after debridement  
Etiology: Trauma; evolving Stages I, II; iatrogenic; bypassed Stage IV  
Treatment: Debridement/Dead-space management  
Stabilization, bone graft (optional)  
Course: Arrest; downstage (I-III) prophylaxis (III Papineau)  
Failure: missed Stage IV (joint proximity)

**Fig. 12A** Stage III classification of osteomyelitis.

**STAGE III**

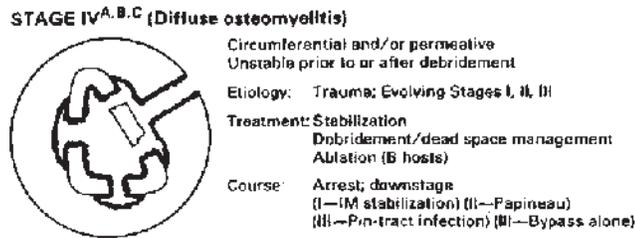


**Fig. 12B** Treatment algorithm for Stages IIIA and IIIB.

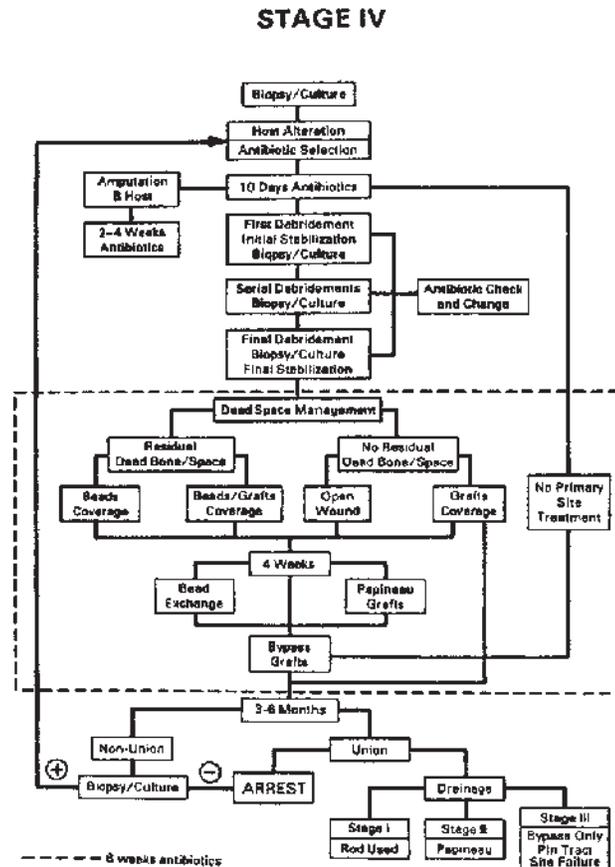
example of osteomyelitis with minimal necrosis. When the Stage I lesion progresses to Stage IV in the spine, the process usually becomes intraarticular and the disc sequesters within the septic joint. The rich blood supply and high cancellous to cortical bone ratio favor a rapid resorption of subchondral bone and medullary debris. The disease will arrest if motion is curtailed and the host augmented

with appropriate antibiotic therapy. The disc is a resorbable sequestrum.

*Deep/superficial osteomyelitis.* There is a deep Stage II lesion with minimal necrosis. Often these lesions are failures in management of Stages III or IV where treatment involved a suction-irrigation system or secondary intention healing. In the preliminary evaluation, no dead bone can be identified to



**Fig. 13A** Stage IV classification of osteomyelitis.



**Fig. 13B** Treatment algorithm for Stage IV.

account for a chronic sinus. Here the ischemic soft tissue adjacent to deep bony surfaces is the nidus that leads to persistent disease. Therapeutic options include exploration and soft tissue manipulation, hyperbaric oxygen therapy, or skillful neglect.

*Infected nonunion with minimal bony necrosis.* In this lesion, the problem again is infected

scar tissue. Surface resorption and union are impeded by constant motion and the resultant local compromise.<sup>26</sup> Immobilization and/or bypass surgery will relieve the soft tissue compromise, promote union, and enhance autosequestrectomy.<sup>27</sup> If the necrosis is more than superficial in septic pseudarthrosis, drainage often recurs despite bony union.

## THE UTMB EXPERIENCE—A PRELIMINARY REPORT

From June 1981 to December 1983, 357 patients with adult osteomyelitis were evaluated and staged on our service.<sup>10</sup> Definitive treatment was given to 189 patients with 192 lesions, and 747 surgical procedures were performed (Table VI). There were 46 amputations: 41 were a first procedure; five followed a limb-salvage attempt. Of the primary amputations 90% (37/41) were in B-Hosts: 86% Stage IVB, 14% Stage IIB. Two of the patients with late amputations were disease-free at nine and 12 months but were unsuccessfully rehabilitated. These two patients were a young woman (Stage IIIA) with Munchausen's syndrome and a young man (Stage IVB<sup>S/L</sup>) with an ipsilateral sciaticofemoral palsy. During the first six months of therapy there were three deaths, including one (IVB<sup>L</sup>) due to trauma at four months, one (IVB<sup>S</sup>) from hypoglycemia at three months, and the third (IIB<sup>S/L</sup>) from an induced bleeding diathesis at three weeks.

Of the patients evaluated, 15% (54/357) were C-Hosts. The remaining 110 of the 164 patients not treated by our service were either outside the consultations (46), IIB or IVB lesions we referred for amputations (50), or osteomyelitis with minimal necrosis (14).

**TABLE VI Classification of 192 Sites of Adult Osteomyelitis Treated Surgically at UTMB (1981–1983)**

Medullary		Localized	
Stage	Number of Lesions	Stage	Number of Lesions
IA	1	IIIA	35 (1 amp.)*
IB	7	IIIB	4
	8		39
	<b>Superficial</b>		<b>Diffuse</b>
IIA	6	IVA	69 (4 amps.)*
IIB	9 (5 amps.)*	IVB	61 (36 amps.)*
	15		130

\*amputations

## RESULTS

Sixty-three patients entered our limb-salvage protocol and were followed for a minimum of two years (Table VII). Among these limb-salvage candidates, 93.6% (59/63) were disease-free and ambulating without assistance at 24 months. Two patients had more than one lesion, bringing the total number of sites treated to 65. Treatment led to an arrest in 95.4% (62/65) of the lesions.

There were nine treatment failures (Table VIII): eight initial, one late, and four overall. These failures were defined by recurrent drainage (5/9), infected nonunion with minimal necrosis (2/9), tumor contamination (1/9), and unsuccessful rehabilitation (1/9). The reasons for initial failure included inadequate debridement (6), poor fixation (1), and stress fracture (1). Of the initial treatment failure group, 62% (5/8) were arrested with retreatment. Three of the four overall treatment failures were in the initial failure group: an occult carcinoma was undiagnosed until the third debridement/biopsy, and two compromised patients suffered stage progression and underwent a below-knee amputation after completing a limb-salvage protocol. The other overall failure (IIIA-Munchausen's) had an ablation elsewhere at 12 months despite disease arrest (pathology report).

**TABLE VII Sixty-three Patients Followed ≥ Two Years (Clinical and Laboratory Evaluations at 24 Months) (A & B Hosts Combined)**

	Stage I	Stage II	Stage III	Stage IV
Patients	2	3	17	39
Sites	4	3	18	39
Drainage	0	0	0	0
+ E.S.R.	2	0	0	1 <sup>c.w.</sup>
+ Incl <sup>111</sup>	0	0	0	4 <sup>c.w.</sup>
Amputations	1	1	1	1
Hospital time (days)	47	38	56	97
Number of Procedures	3.4	2	3.5	5.7

**TABLE VIII Sixty-three Patients Followed  $\geq$  Two Years Treatment Failures**

Type	Class	No.	Mode	Course	Comment
I	A	0			
	B	1	Drainage (7 months)	BKA	IB-IVB Calcaneus/foot
II	A	0			
	B	1	Drainage (12 months)	BKA	IIB-IVB Calcaneus/foot
III	A	1	Pain (12 months)	AKA	Munchausen's syndrome
	B	1	Drainage (9 months)	Arrest	Inadequate debridement
IV	A	3	Drainage (8 months)	Arrest	Inadequate debridement
			Nonunion	Arrest	Stress fracture bypass/ORIF
			Nonunion	Arrest	ORIF
	B	2	Drainage (3 months)	Arrest	Inadequate debridement
	—	9	Tumor	BKA	

All 65 lesions were indium-positive prior to treatment. Since there were four amputations, only 61 sites were available for followup. Six of these 61 lesions (10%) had a normal sedimentation rate and an abnormal indium scan at 18 months (one IIIA, three IVA, and two IVB lesions). Three of these same six patients were still indium-positive with normal sedimentation rates at 24 months (one IVA and the two IVB lesions). One palliative procedure was performed in the original group of 63 patients (C.W.-IVB<sup>L</sup>); this patient's sedimentation rate and indium scan are still abnormal at 54 months.

The organisms isolated in 50 consecutive patients are listed in Table IX. *Staphylococcus aureus* was the most commonly isolated bacteria. More than two organisms were present in 46% of our patients. The number and type of organisms had no bearing on the outcome of treatment, providing an adequate debridement was performed. In all cases, appropriate antibiotic coverage was gained and maintained for the duration of the treatment protocols.

Representative sedimentation rate profiles for Stage IA/B, IIA, IIIA, and IVA are seen in Figs. 14A-14D. The Stage I profile is a composite of A and B hosts to illustrate the problem that occurs in using a sedimentation rate to follow compromised patients. These hosts are plagued by minor illnesses and peripheral sores that affect this index. In Stage IIA, the sedimentation rate did not fall before three

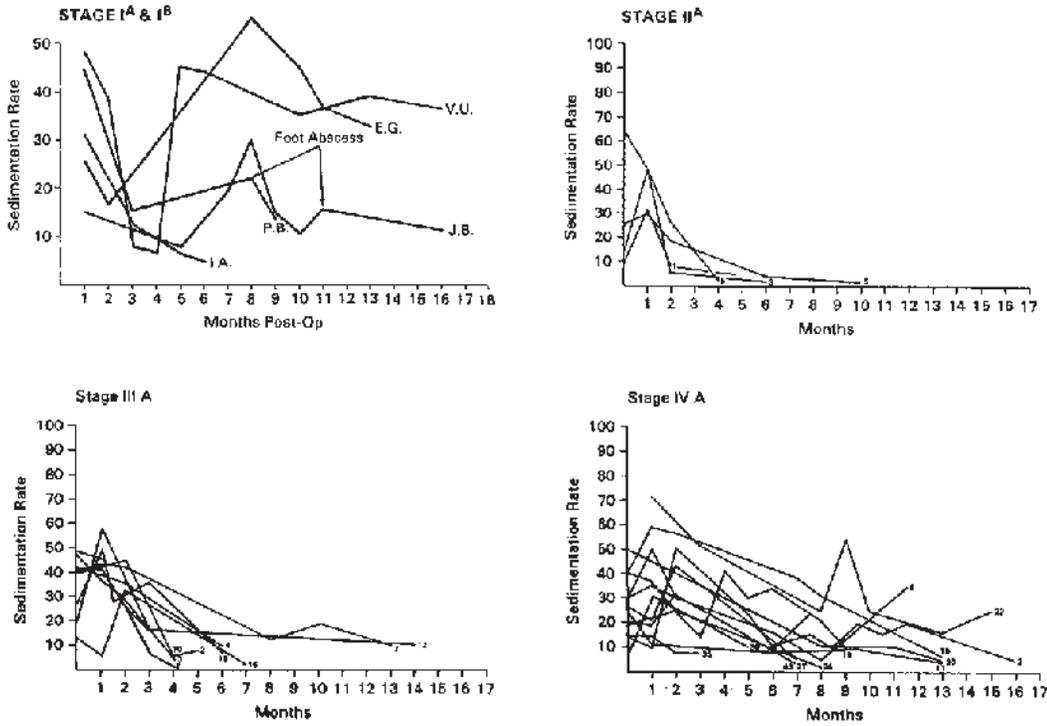
months, a profile identical to soft tissue infections. The profiles for Stages IIIA and IVA reflect the wound healing disturbances associated with bone grafts, internal hardware, pin tract infections, and nonunion.

**DISCUSSION**

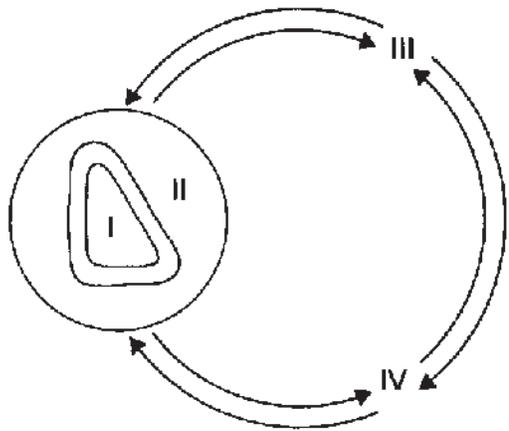
The clinical stages of adult osteomyelitis may interplay during both their natural history and their response to therapy (Fig. 15). An anatomic or physiologic stage progression (upstaging) increases the difficulty of treatment. The prognosis for disease arrest without

**TABLE IX Microbial Spectrum in 50 Consecutive Patients**

		Organisms Isolated	No. of Patients
<i>S. aureus</i>	29		
<i>Pseudomonas</i>	17	1	12
Anaerobes	12	2	15
<i>Enterococcus</i>	11	3	14
<i>S. epidermidis</i>	11	4	5
<i>Streptococcus pyogenes</i>	9	5	4
<i>Proteus species</i>	8	6	0
<i>Bacillus</i>	4		
<i>Klebsiella</i>	4		
<i>Serratia</i>	3		
<i>Citrobacter</i>	2		
<i>E. coli</i>	2		



**Fig. 14** Representative sedimentation rate profiles for Stage IA/B, IIA, IIIA, and IVA. Most Type I lesions are in B-hosts. Sedimentation rate is an inadequate follow-up index in these patients, secondary to frequent illnesses (A-arrows). The response of the only A-host in the Stage IA/B group (patient I.A.) mimicked a patient with a Stage IIIA osteomyelitis.

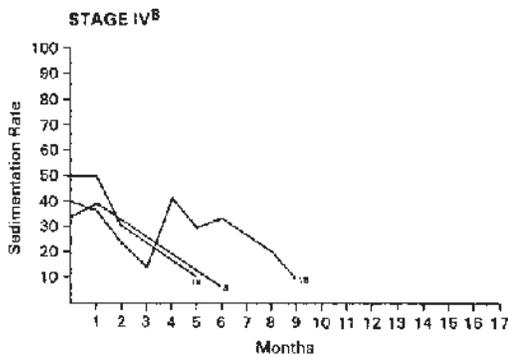


**Fig. 15** Cavitation and reclassification to Stage III or IV will occur whenever Stages I and II appear simultaneously.

ablation is improved when a downstaging of the process can be accomplished by therapy.

Patients with Stage IV lesions suffer the greatest number of complications and represent the majority of treatment failures. The Stage IVB sedimentation rate profile (Fig. 16) represents a composite of B-hosts with type IV lesions whose deficiencies were corrected with successful host alteration (Table II). The sedimentation rates and arrest rate in these patients have mimicked the Stage IVA responses. These encouraging results have strengthened our commitment to the diagnosis and treatment of host deficiencies. The B-host challenges the frontiers of modern medicine and brings the search for effective adjunctive therapies to the forefront.<sup>6-9,28-33</sup>

In our experience, indium-111 chloride imaging has been accurate in localizing per-



**Fig. 16** The sedimentation rate profile of three B-hosts with Type IV lesions who underwent physiological downstaging to Class A by host alteration.

sistent osteomyelitis in adults.<sup>34</sup> The cytokinetics of this imaging technique have not been established. Detailed anatomic mappings of specimens amputated 24 hours after indium chloride injection have demonstrated heavy indium concentration in tissues laden with histiocytes, macrophages, plasma cells, and lymphocytes as well as those with leukocytic infiltrates. This scan is not limited to acute and subacute processes<sup>35</sup> and appears to have a limited affinity for noninfected, reactive bone.<sup>36</sup>

The methods used in the management of osteomyelitic wounds have influenced our long-term results. All of the amputation stumps reverted to normal indium studies by 12 months. Similarly, 94% of the wounds managed with primary closure (Fig. 7) in Stages II and III displayed normal indium concentrations at one year. The biology of wound healing in complex reconstructions lends support to the gradual resolution in the indium scan sequence depicted in the follow-up protocol. Eighty-seven percent of the Stage I, III, and IV lesions were indium-positive at 12 months if dead space management included cancellous bone grafts and/or an osteosynthesis. However, this percentage decreased to 12% and 6% at 18 and 24 months, respectively. Two of our overall treatment failures had at least one normal sedimentation rate prior to a recurrence of disease at seven and 12 months. No patient with a positive-

turned-negative indium scan has experienced recurrence. Our prospective study should help define the role of sequential indium-111 chloride imaging in following treated cases of adult osteomyelitis.

## CONCLUSIONS

1. The treatment and prognosis of adult osteomyelitis correlate with the clinical stage of the disease.
2. The UTMB staging system provides guidelines for the use of adjunctive therapies and a basis for comparing treatment protocols from institution to institution.
3. Serial indium-111 chloride scans may prove a sensitive index for following patients with treated osteomyelitis.

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