LECTURE 7

SURGICAL INFECTION. CLASSIFICATION. ACUTE PURULENT INFECTION. OSTEOMYELITIS

I.Actuality of theme. A surgical infection is an infection that (1) is unlikely to respond to nonsurgical treatment (it usually must be excised or drained) and occupies an unvascularized space in tissue or (2) occurs in operated site. Surgeons are regrettably familiar with vicious circle of operation or injury, infection, malnutrition, immunosuppression, organ failure, reoperation, futher malnutrition, and futher infection. One of the fine arts of surgery is to know when to intervene with excision, drainage, physiologic support, anthibiotic therapy, and nutritional therapy.

From ancient times to the 19th century, infection served as a great impediment to surgical progress. It would take many thousands of years before the system of thought in surgery would be transformed from orthodoxy to our modern dynamic scientific method. Within this philosophical framework, Louis Pasteur shook the mythical underpinnings of wound putrefaction and provided a framework on which Lister built the system of antisepsis.

With the introduction of antibiotic therapy, it was hoped that serious infection complicating surgical practice would be eliminated. Unfortunately, this has not occured. Not only has the problem of postoperative wound and hospital-aquaired infection continued, but widespread antibiotic therapy has often made prevention and control of surgical infection more difficult. In this regard has been the emergance of increasing numbers of serious infections related to a complex combination of factors, including the performence of more complicated and longer operation, an increase in the number of heriatric patients with accompanying chronic or debilitating disesaes, many new surgical procedures with implants of foreign materials, including artifitial hearts, joints and vessels, a rapidly expanding number of organ transplantsrequaring the use of immunisuppressive agents, and increased utilization of diagnostic and treatment modalities that cause greater bacterial exposures or the suppression of normal host resistance. Unfortunately, many infections continue to follow laxity in aseptic techique, disregard for esthablished surgical principles, and unwarrented reliance upon prophylactic antibiotic therapy. Not only are the infections of medical significance, but they represent medicolegal problems. Todaty surgeons must accept the responsibility of coping with infections and realize that knowledge of many aspects of microbiology, immunjlogy, and pharmacology is essential to complement the surgical skills. Basic understanding of the body's defenseagainst infection is essential to a rational application of surgical and other therapeutic principles to the control of infection.

II. Aims of lecture :
Educational:

- To expound the classification of surgical infection(β= II);
- To elucidate the ethiology and pathogenesis of surgical infection (β= II);
- To elucidate the ethiology and pathogenesis of surgical site infection (β= II);

- To describe the classification, diagnosis, treatment of acute purulent soft tissue diseases (β= II);
- To describe the ethiology, pathogenesis, diagnosis and treatment of hematogenous osteomyelitis and chronic osteomyelitis (β= II);
- To study the students the main principles of evidence-based medicine according the subject of lecture (β= IV).

Educative:

1. To educate for students sense of responsibility for every prescription, research, procedure, manipulation or surgery, for a health and renewal of capacity of patient, for the rightness of adequate estimation of the common state of patients and grant of timely effective treatment.
2. To develop deontological notion in the students, to study the students carry our deontological approach to patients with surgical infection.

III. Plan and organization of structure of lecture

№	Basic stages of lecture and their maintenance	Aims are in the levels of abstraction	Type of lecture, methods and facilities of activation of students, equipment	Division of time
1	Preliminary stage. Determination of educational aims and motivation.		Items I,II	5%

2	**Basic stage. Teaching of lecture's material** • Classification of surgical infection; • Ethiology and pathogenesis of surgical site infection; • Ethiology and pathogenesis of surgical infection; • Classification, diagnosis, treatment of soft tissue infections; • Ethiology, pathogenesis, diagnosis and teatment of hematogenous osteomyelitis and chronic osteomyelitis	II II II II II	Type of lecture – thematic (with controversial elements – critical analysis of results of meta-analyses, randomized controlled, trials, guidlines which are devoted for the problem of surgical infection). Facilities of activation of students are a questions, controversial situations, illustrative material	85%
3	Final stage (resume of lecture, general conclusions, answers to the possible questions, task for students for preparation for practical classes)		List of literature, question, task for students	10%

IV. Subject of a lecture

Surgical infection is a term that, though frequently used, is not clearly defined. In the strictest sense, it implies infection amenable to operative management through surgical source control, as in the case of complicated diverticulitis or necrotizing soft tissue infection. More generally, however, the term can refer to any infection commonly seen in surgical patients (e.g., central line infection or postoperative pneumonia). Both definitions are pertinent, in that the same diagnostic principles apply in each situation.

Surgical Site Infections

The Centers for Disease Control and Prevention term for infections associated with surgical procedures was changed from surgical wound infection to surgical site infection in 1992 . These infections are classified into incisional, organ, or other organs and spaces manipulated during an operation; incisional infections are further divided into superficial (skin and subcutaneous tissue) and deep (deep soft tissue-muscle and fascia). These definitions should be followed universally for surveillance, prevention, and control of surgical site infections.

The presence of surgical infectious disease is usually determined clinically and confirmed microbiologically. Identification of an infection is rarely incidental: most often it is sought in response to a clinical signal. This signal is frequently fever but may be one or more of a number of other symptoms and signs.
Most surgical infections are outpatient conditions that are easily diagnosed and treated. Infections in hospitalized patients, whether related to the primary surgical disease or resulting from surgical therapy, are less easily managed. The greatest challenges in diagnosis and treatment of surgical infections arise in the perioperative and postoperative periods.

General Approach to Diagnosis of Infection

The search for an infection is prompted by a clinical signal indicating a problem in need of resolution. The signal denotes the response of a patient to an infectious stimulus and is a function of the patient's physiologic ability to react to the endogenous and exogenous mediators liberated through the infectious process. A thorough history and physical examination are imperative and should be followed by selected laboratory tests. Normally responsive patients tend to show the classic signals, whereas compromised or complex patients often show more subtle signs that may only be noted as abnormalities on routine bloodwork.

The diagnostic approach to suspected infection must be modified according to patient characteristics and the circumstances of presentation; for example, the specific differential diagnosis for infection appearing on postoperative day 1 will clearly be different from that for infection appearing after 1 week in the ICU.

There are three important elements at play when a surgical patient experiences an infection: (1) the health of the host, (2) the intensity of the physiologic response to infection, and (3) the nature of the pathogen. All three factors must be considered carefully in the diagnosis of surgical infection.

Host Status: Normally Responsive versus Compromised or Complex

That signs and symptoms of infection in compromised or complex patients differ from those in normally responsive hosts has important diagnostic implications. Normally responsive patients, for whom the physician can obtain a history and

perform a physical examination, respond to infection in the classic manner—typically, with fever, tachycardia, leukocytosis, malaise, and other appropriate symptoms. For many reasons—even simply if the infection is severe—normally responsive patients may become compromised. Compromised or complex patients are unable to meet inflammatory or infectious challenges in the normal manner. Hence, the clinical signals of infection in such patients differ from those in normally responsive patients, often being absent or developing at a later stage of infection. Indeed, a multitude of clinical conditions or physiologic states define compromised or complex patients. These include the extremes of age, immunosuppression as a result of either disease (e.g., HIV infection or lymphoma) or medication (e.g., chemotherapy), thermal injury, major trauma, acute end-organ failure in the ICU, and the presence of more than one chronic disease. The prevalence of such patients in modern hospital surgical practice is increasing steadily.

It must be kept in mind, however, that the normally responsive patient and the compromised or complex patient are merely extreme points on the clinical spectrum rather than categorically distinct populations.

Physiologic Response to Infection

Normally Responsive Patients

Cardinal Signs of Inflammation. Rubor, calor, dolor, tumor, and functio laesa—that is, redness, heat, pain, swelling, and loss of function—have been considered the cardinal signs of localized inflammation since the times of Hippocrates and Galen. They remain the primary signals leading to medical consultation for outpatient surgical infections and for many of the infectious complications of operation. They are emblematic of the host's effort to contain infection locally and may signal the presence of infection even in cases where the primary site of infection is a deeply situated organ or tissue. Infections amenable to surgical intervention may present in this way; however, nosocomial infections complicating the course of surgical patients are generally signaled in more subtle ways.

Fever. Fever is perhaps the most common signal that an infectious process is present. Postoperative fever is a normal part of the recovery process; understanding the typical febrile course is important in differentiating normal from pathologic fever. It is unusual for a sudden, very high fever to be the first signal of an infection. Infection usually begins to manifest itself with a prodrome, recognition of which speeds diagnosis and therapy. Investigation should be started when the patient's temperature reaches 38° C (100.4° F) rather than 40° C (104° F). Although this point may seem obvious, many of the crisis intervention measures required in managing fevers could be avoided if the significance of more modest temperature elevations were recognized more often.

A fever that appears after the normal postoperative temperature elevation has resolved must not be ignored. To simply wait for such a fever to dissipate is to

court disaster. In the absence of a clear diagnosis, a thorough physical examination of the patient, directed by laboratory tests and followed by reexamination as necessary, is required to identify occult infection.

Miscellaneous Signals. It is common wisdom that the signals communicating underlying infection in the compromised or complex host may be subtle. The astute clinician will be in tune with these and, with experience, will recognize when to undertake a diligent search for infection.

Altered heart rate. A heart rate that is either too high or too low may signal an infection. On rare occasions, a change in rhythm in the elderly (e.g., paroxysmal atrial tachycardia, flutter, or atrial fibrillation) indicates an infectious process. Gram-negative sepsis may produce a so-called relative bradycardia, meaning that the resulting tachycardia is not as pronounced as one might expect. An unexplained sustained increase in heart rate should not be ignored.

Tachypnea. Whereas tachypnea occurs commonly after operation in response to pain or poor pulmonary toilet, it may also signal either the prodrome of infection or the onset of SIRS. Because tachypnea may herald not only infection but also other important diagnoses (e.g., pulmonary embolism), it must be thoughtfully and methodically evaluated.

Pain. Pain that persists or is out of proportion to the expected response deserves attention. Whenever a surgical wound that was healing favorably for the first 5 to 7 days becomes more painful, a deep SSI must be suspected and ruled out, even if other signs are absent. Unexplained muscular pain is often the first harbinger of deadly necrotizing soft tissue infection caused by gram-positive bacteria (e.g., group A streptococci), the early recognition of which may be lifesaving and limb-preserving. Sometimes pain is referred, and the painful area appears normal on examination. Pneumonia that presents with abdominal findings is a classic example, as is the shoulder-tip pain with a normal range of motion seen in patients with a subphrenic abscess.

Confusion. Confusion is a common symptom of infection in the elderly; it is also an important signal in patients who had been well and fit. The physician's first response to confusion in an elderly patient in the postoperative period must be to seek a cause, not to order sedation.

Ileus. Ileus has many causes, some of which are not well understood. Prolonged ileus after abdominal operation—as well as almost any ileus after other operations—requires explanation. Infections at remote sites (e.g., SSI and pneumonia) can produce ileus, as if the bowel were a target organ such as the kidney, the liver, or the lung.

Compromised or Complex Patients

The presence of fever remains a common signal of infection in the compromised or complex patient; however, it may be absent, or the patient's temperature may already be elevated as a result of other causes. Cardinal signs of infection may be present. More often, however, the infection is occult, and classic signals are

unrelated to the infectious focus. In some very ill immunocompromised patients, findings that usually signal an infection may already be present. Slight changes in clinical status (e.g., minor temperature elevations, increased fluid requirements, confusion, and ileus) or changes in laboratory findings (e.g., an elevated WBC count, glucosuria, and hyperglycemia) should trigger investigation.

Patients in whom the first signal of an infectious process is organ dysfunction or failure, rather than fever and tachycardia, are likely to be physiologically compromised and seriously ill; perhaps more important, however, is that they are a group whose diagnosis and management require expert clinical skills. Because the classic septic response may not be present, it is essential to be alert to the signs and symptoms of occult infection. In these patients, the laboratory and the radiology suite become increasingly important in diagnosing and documenting the evolution of the infection.

Clinical Signs and Symptoms of Occult Infection. Subtle changes in temperature, mental status, pulse rate, or respiratory rate may signal occult infection, as may the development of pain or ileus.

Intermittent hypotension and septic shock. Septic shock, an important manifestation of an unrecognized focus of infection, is the original expression of multisystem failure. Fortunately, it rarely occurs without warning. The prodrome often includes fever and sometimes other signals. Recurring hypotension is the most characteristic signal; it usually is not catastrophic and responds quickly to fluid resuscitation. Oliguria may accompany the hypotension. If this clinical state is allowed to progress, the hypotension will lead to renal failure, with substantial water retention. Septic shock will result if the infection is not identified and treated with appropriate antibiotics and source control as necessary.

Both clinical assessment and laboratory studies are necessary to confirm the presence of septic shock, although florid septic shock is easily recognized on clinical examination alone. Any or all of the following findings may be present in varying degrees: tachycardia; tachypnea; hypotension; warm, dry extremities; generalized flushing; and other signs suggesting a hyperdynamic, hypermetabolic state. Swan-Ganz catheter measurements confirm high cardiac output and low peripheral vascular resistance.

Gastric hemorrhage. Gastric hemorrhage may be the presenting symptom of serious infection even if prophylactic measures against such hemorrhage have been taken. It is particularly suggestive of perigastric abscess resulting from anastomotic leakage after upper abdominal surgery. Gastric hyperacidity and bleeding generally respond to drainage of an abscess. Hemorrhagic gastritis must always be considered a signal of occult infection, which demands prompt diagnosis and treatment.

Delayed wound healing. The absence of wound healing can indicate the presence of a significant infection. Typically in such a case, wounds left for delayed primary closure or secondary closure do not exhibit the appropriate granulation tissue and

appear pale, dry, and unhealthy. The development of good granulation tissue is a sign that infection is controlled.

Renal failure. Renal failure is identified by elevations in serum creatinine and blood urea nitrogen (BUN) levels, which can be highly sensitive signals of developing infection. A still more sensitive indicator is an alteration in creatinine clearance, a laboratory test underutilized in the ICU. Such alterations are generally evident before changes in serum levels. Creatinine clearance should be measured at an early stage in high-risk patients. In the presence of shock, renal failure can develop suddenly. Otherwise, loss of renal function is insidious, but it can usually be identified if sought before oliguria or anuria develops. Resolution of infection is associated with return of function.

Hepatic and respiratory failure. Hepatic failure or by a change in the fraction of inspired oxygen (F_IO_2) requirement, can behave in the same way as renal failure (i.e., with drainage and control of infection leading to restoration of function).

Abnormal platelet count. Thrombocytosis is often seen in association with infection, particularly with compromised hosts, in whom the infection may be occult. Thrombocytopenia may also indicate serious infection, though it is not a common signal. When this occurs in the context of sepsis, it is either because disseminated intravascular coagulation (DIC) has developed or because there is a diminution of all blood lines indicating marrow dysfunction. The cause of any abnormal platelet count should be identified promptly if possible.

Hyperglycemia and insulin resistance. Hyperglycemia and insulin resistance are often reliable signals of the presence of infection in diabetic patients as well as in nondiabetic patients. The degree of insulin resistance can reflect the severity of the infection as well as the effectiveness of infection control.

Immune failure. Severe infection is immunosuppressive. The most clinically applicable measure of immune failure at present is probably delayed wound healing.

Evaluation for Presence of Infection

Once the surgeon has evaluated in which ways the patient may be compromised or susceptible to infection and to which degree the physiologic response is inappropriate or harmful (i.e., provokes multiple organ dysfunction or shock), then it is important to consider which microorganisms might be responsible for the presenting clinical picture. This obviously varies with the clinical situation and requires knowledge of specific condition-associated pathogens, as well as the prevalence of certain pathogens in a particular hospital or ICU.

The prevalence of resistant bacterial strains should be monitored in clinical settings in order to guide empirical therapy. MRSA, once an occasional finding, has become so common in some ICUs and chronic wards that surveillance and isolation programs are no longer employed. It is now the rule for extensive invasive monitoring and access devices to be used in the care of critically ill patients, who thereby become particularly predisposed to gram-positive infection;

accordingly, it is important to appreciate the likelihood that MRSA will be encountered. The same is true of Pseudomonas aeruginosa, a ubiquitous commensal and a common gram-negative pathogen in hospitals. In compromised patients exposed to multiple antibiotics, P. aeruginosa readily acquires antibiotic resistance that usually necessitates the use of double-agent or broad-spectrum coverage for effective management.

Recognizing the virulence of certain pathogens is as important as appreciating their antibiotic susceptibilities. Enteroinvasive Escherichia coli, for example, may cause a rapidly progressive hemorrhagic enteritis and provoke a fatal septic syndrome marked by acute renal failure, bleeding, and coma. Necrotizing soft tissue infections, particularly when caused by gram-positive organisms, may be precipitously fatal. Early Gram stain microscopy to identify the specific pathogen is a critical step in the management of this condition.

Essentially the same clinical and laboratory assessments are used to evaluate normally responsive and compromised or complex patients for the presence of infection. There is, however, a significant difference in emphasis. In normally responsive patients, the diagnosis of infection is usually made on clinical grounds with laboratory support, whereas in compromised or complex patients, the diagnosis is usually made on the basis of laboratory findings with clinical support.

The ICU patient presents a particular conundrum. Nosocomial infection is identified in an estimated 20% of such patients. Despite the frequency with which it is suspected and reported, it is difficult to prove unequivocally. The perceived prevalence of nosocomial infection has created a strong predisposition toward instituting empirical antibiotic therapy in ICU patients; however, the global value of this action in terms of both patient outcome and the impact on the ecology of the ICU is unconfirmed and requires validation. The enormous inconsistencies in how infections are diagnosed have a tremendous effect on our ability to assess the efficacy of therapy for infection. The current approach to diagnosing infection in surgical patients, particularly those who are critically ill or compromised, is still in great need of clarification and standardization. Until these issues are resolved, the clinician must be familiar with the strengths and limitations of a variety of current diagnostic methodologies and then exercise thoughtfulness and disciplined diligence. The likelihood that a particular patient is infected (i.e., the pretest probability) is as important in the decision to treat as the fulfillment of any particular constellation of diagnostic criteria.

History and Physical Examination

The history should include all comorbid conditions (e.g., diabetes, lung disease, cirrhosis, hepatitis, kidney disease necessitating dialysis, and previous important infections) as well as a hospitalization history that covers health status, surgical diagnosis and therapy, additional therapeutic interventions (including interventional radiology, monitoring devices, drains, and drugs), and other related variables.

In the early postoperative period (3 to 6 days after operation), the traditional causes of the signals of infection have their origin in the wound, intravascular lines, the urinary tract, and the lungs. Deep thrombophlebitis, with or without pulmonary embolism, may also initiate a systemic inflammatory response that mimics sepsis. The general physical examination is often unrewarding, but a number of specific examinations should be carefully performed, with emphasis given to (1) all wounds and surgical sites, (2) all invasive monitoring or therapeutic devices and surrounding areas (notably central and peripheral I.V. lines), (3) all drainage systems and surrounding tissue, with particular attention paid to the nature of the drainage and whether it has recently changed in character or volume (particularly if it has stopped), (4) the rectal examination (for pelvic or prostatic infection), (5) areas of potential decubitus ulcers, (6) the neck (for CNS infection), (7) intravascular lines, surrounding tissue, and proximal vessels, (8) the lungs, and (9) the legs. The physical examination is important as a guide for selecting specimens for microbiologic analysis, particularly when there have recently been significant changes in wounds or drainage. The decision to seek radiologic consultation may depend on the findings on physical examination.

Diagnostic Tests

Hematologic and Biochemical Tests. After physical assessment, laboratory blood tests are routinely relied on to orient the surgeon toward or away from a clinical diagnosis of infection. Leukocytosis, particularly with an increase in band forms, is a usual but inconsistent marker for infection. The WBC count is widely used to follow the response of infection to therapy and thus has been adopted as a surrogate indicator of the success or failure of therapy. Surprisingly, however, the documentation supporting this ubiquitous practice is sparse. Not only is the daily series of complete blood counts often ordered in conjunction with the initiation of antibiotic therapy wasteful and unpleasant for the patient, but it also typically tells the clinician little about the clinical course that cannot be gleaned at the bedside.

In more complex surgical patients, other biochemical cues are used to varying degrees as means of assessing the likelihood of infection. In addition to thrombocytosis, thrombocytopenia, hyperglycemia, and metabolic acidosis, which commonly reflect the stress of severe infection, changes in the erythrocyte sedimentation rate (ESR) and in blood levels of C-reactive protein (CRP), procalcitonin (PCT), interleukin-6 (IL-6), and tumor necrosis factor (TNF) have a significant association with the presence of clinical infection. As yet, however, no single mediator of systemic inflammation has been validated as a reliable clinical tool for surveillance of the progression of infection or the response of infection to treatment.

Microbiologic Studies. As a rule, Gram stains and cultures of wound tissue, sputum, urine, and drainage effluent are useful studies. In some cases, a battery of cultures of potential sites of infection may be the only feasible approach
The use of polymerase chain reaction (PCR) technology to detect bacterial DNA is emerging as a useful alternative to microbiologic culture for determining the presence of infection. PCR identifies and amplifies a specific bacterial DNA sequence by means of a chemical proliferation process that may take no longer than a few hours.
Radiology. A chest x-ray is mandatory. Ultrasonographic examination of the operative site may be useful to evaluate the possibility of a deep abscess. Computed tomography of the operative site is often more useful than ultrasonography because the presence of wounds, dressings, and drainage tubes may obscure ultrasonographic findings. In compromised or complex patients who have not recently undergone an operation, the medical and surgical history combined with the radiologic examination may be the only guide to the potential infectious focus (e.g., ulcer, diverticulitis, cholecystitis or cholangitis, or obstructed ureter). Percutaneous aspiration of potentially infected fluid should be considered, and this fluid should be microbiologically examined if possible.

Approaches to Specific Infections in Complex Surgical Patients

The following infections may occur in all surgical patients. They are, however, much more difficult to identify in complex surgical patients, such as those admitted to the ICU. Because the signals of infection are less specific and extremely difficult to interpret after injury or operation, the approach to diagnosing and treating infection must be cautious and disciplined.

Surgical Site Infection

SSIs include all infections occurring within the operative field, from the skin to the actual area of surgery. The patient history should address previous diseases as well as issues concerning the operation itself, such as wound risk classification, duration, difficulty, urgency, use of drains, other details of the procedure, time elapsed since the operation, and whether the patient was immunosuppressed, experienced trauma, or received chemotherapy. Physical examination should focus on the cardinal signs of infection and the absence or presence of a healing ridge.
Deeper infections tend to become apparent later in the postoperative course, often after a period in which the patient appears to be recovering, and are associated with a variety of signals, some of which can appear suddenly. The physical examination is often useless or misleading because of discomfort associated with the operation. Surgical site pain that increases or fails to resolve in the 7 to 10 days following surgery is an important yet subtle marker for occult infection that calls for investigation. Rectal examination is important because it may detect abscess formation or bleeding. Return of ileus after an abdominal operation is a significant clue to the presence of abdominal infection.

Culture is essential because use of the correct antibiotics is particularly vital in treatment of compromised or complex patients. Knowledge of the organism and its sensitivities is the key to identifying epidemic or multiresistant strains.

Pulmonay infection. The most frequent sites of infection postoperatively are lungs. Postoperative pulmonary complications play a significant role in the risk for surgery and anesthesia. The most important and morbid postoperative pulmonary complications is pneumonia. While clinicians are very conscious of the importance of, and risk factors for, cardiac complications, clinicians who care for patients in the perioperative period may be surprised to learn that postoperative pulmonary complications are equally prevalent and contribute similarly to morbidity, mortality, and length of stay. Pulmonary complications may also be more likely than cardiac complications to predict long-term mortality after surgery, particularly among older patients

Urinary Tract Infection

Nearly all patients admitted to the ICU have a urinary catheter in place; of these, it is estimated that about 20% progress to urinary tract infection (UTI). Bacteria adhere to urinary catheter surfaces, where they promote growth of a so-called biofilm composed of microorganisms, bacterial glycocalices, Tamm-Horsfall protein, and urinary salts. Eradication of this infectious nidus is essentially impossible without catheter removal. The standard criterion for the diagnosis of UTI (10^5 bacteria/ml) is difficult to apply in catheterized patients because antibiotic therapy without removal of the catheter and the source of bacteriuria would be ineffective. Furthermore, it is well established that urine cultures demonstrating as few as 10^1 or 10^2 bacteria/ml increase 1,000-fold within 1 to 2 days; effectively, therefore, any bacterial growth on a urine culture from a catheterized patient signals heavy colonization, if not infection.

More important than quantifying the degree of bacteriuria is determining whether there is any evidence of tissue invasion by urinary bacteria, which would present as pyelonephritis, cystitis, prostatitis, epididymitis, bacteremia, or sepsis. The patient history should determine whether a Foley catheter was used and for how long; how, when, and why it was inserted; instrumentation (e.g., a so-called in-and-out catheter, cystoscopy, or transurethral resection of the prostate); and whether the patient has had any previous UTIs. The physical examination should ascertain whether there is any costovertebral angle tenderness or evidence of prostatic or epididymal tenderness.

Laboratory tests should include gross and microscopic urinalysis, urine culture, and sensitivity tests. Blood culture is important because it may substantiate the diagnosis, identify the bacteria present, and determine the degree of invasiveness of the infectious process.

Vascular Catheter Infection

The most frequent sites of infection postoperatively are I.V. catheters, particularly peripheral ones. Diagnosis of peripheral catheter infection is simple and is made on clinical grounds. Diagnosis of central venous catheter (CVC) infection is more difficult. Because hospitalized patients are increasingly being managed with monitoring or therapeutic modalities that depend on vascular access (e.g., total parenteral nutrition and dialysis), line infections have become more common, with an incidence ranging from two to 30 infections per 1,000 CVC days. The combined pressures imposed by (1) the need to maintain vascular access in sicker and more complex patients and (2) the increasing predominance of gram-positive CVC infections observed since the late 1970s have led clinicians in many centers to administer empirical therapy without line removal to complex patients as a matter of course; some even advocate 10 to 21 days of vancomycin-based therapy. The problems associated with the latter approach—emerging vancomycin resistance, nephrotoxicity, and rash—are serious and relate specifically to the diagnostic strategy used to manage potential CVC infections. Distinguishing between contamination, colonization, and true infection is problematic; as a result, a number of diagnostic strategies have been advocated that are predicated more on practicality and cost-effectiveness than on microbiologic reality.

It is believed that CVC infection most commonly arises from invasion by skin microorganisms (S. aureus or S. epidermidis in about 80% of cases), which may manifest itself as exit-site purulence with or without local cellulitis, as a tunnel infection that may be clinically difficult to detect, or as catheter-related bloodstream infection (CR-BSI). Of these, CR-BSI, which complicates as many as 5% of line placements, is the most clinically important entity. It is strictly diagnosed by identification of the same microorganism (identical species and antibiogram) grown from both the catheter and a peripheral blood culture.

The catheter may be cultured in one of several ways, the most common of which is the roll-plate method . Because it is theoretically possible that this technique may fail to detect bacteria harbored within the catheter lumen, some authorities advocate the more sensitive sonication method, in which the catheter segment is immersed and agitated in a medium to produce a broth that contains bacteria from both the internal and the external surfaces of the line. This technique is both more costly and more time consuming, in that it requires quantitative cultures that are deemed positive only when more than 10^3 colony-forming units are detected. More often, blood drawn through the CVC or cultured from an exit-site exudate is compared with peripheral cultures, and thus there is no need to remove the line. If quantitative cultures are done, a line blood culture showing five to 10 times more growth than the peripheral sample strongly suggests that the catheter is the source of the bacteremia. A less costly method that renders quantitative cultures unnecessary relies on the speed of bacterial growth: if growth in catheter-drawn

blood is faster than that in peripherally drawn blood, a primary line infection is likely. On its own, a line blood culture is not sensitive or specific enough to be diagnostically useful.

Soft tissue infection

Soft tissue infection commonly results from inoculation of bacteria through a defect in the epidermal layer of the skin, such as may occur with injury, preexisting skin disease, or vascular compromise. Less commonly, soft tissue infection may be a consequence of extension from a subjacent site of infection (e.g., osteomyelitis) or of hematogenous spread from a distant site (e.g., diverticulitis or *C. septicum* infection in patients with colonic carcinoma). It may also occur de novo in healthy patients with normal-appearing skin, often as a result of virulent pathogenic organisms.

Conditions that disrupt the skin and alter its normal barrier function predispose patients to bacterial contamination. Host factors may increase susceptibility to infection and limit the patient's ability to contain the bacterial inoculum. Clinically occult infection or inadequate treatment of other conditions may also lead to secondary development of soft tissue infection (as is sometimes seen in patients with diverticulitis; perirectal, pilonidal, or Bartholin's cyst abscesses; strangulated hernias; or panniculitis). Delayed or inadequate treatment of superficial infections (e.g., folliculitis, furuncles, carbuncles, cellulitis, and surgical site infections) may lead to more severe necrotizing infections.

Soft tissue infections may be classified as

- ✓ superficial or deep;
- ✓ nonnecrotizing or necrotizing;
- ✓ primary (idiopathic) or secondary;
- ✓ monomicrobial or polymicrobial.

Superficial infections involve the epidermis, dermis, superficial fascia, or subcutaneous tissue, whereas deep infections involve the deep fascia or muscle. Necrotizing soft tissue infections are distinguished by the presence of extensive, rapidly progressing necrosis and high mortality. Such infections are termed necrotizing cellulitis, necrotizing fasciitis, or myonecrosis according to whether the deepest tissue layer affected by necrosis is subcutaneous tissue, deep fascia, or muscle, respectively.

Primary (idiopathic) soft tissue infections occur in the absence of a known causative factor or portal of entry for bacteria. Such infections are uncommon and are believed to result from hematogenous spread or bacterial invasion through small unrecognized breaks in the epidermis. Soft tissue infection caused by *V. vulnificus* is an example of a primary soft tissue infection: it is attributed to

bacteremia developing after the ingestion of contaminated raw seafood. Only 10% to 15% of all necrotizing soft tissue infections are idiopathic; the remaining 85% to 90% are secondary infections, developing as a consequence of some insult to the skin that predisposes to infection. Secondary soft tissue infections may be further categorized as posttraumatic, postoperative, or complications of preexisting skin conditions.

Soft tissue infections are classified as monomicrobial when they are caused by a single organism and as polymicrobial when they are caused by multiple organisms. Most superficial soft tissue infections are caused by a single aerobe, usually *S. pyogenes* or *S. aureus*. Exceptions to this general rule include infections associated with skin damaged by animal or human bites, cellulitis associated with decubitus or other nonhealing ulcers, and infections in immunocompromised patients. These infections are typically polymicrobial, often involving aerobic or facultative gram-negative organisms and anaerobes in addition to aerobic gram-positive bacteria.

Deep necrotizing soft tissue infections are polymicrobial 70% to 75% of the time. They are caused by the synergistic activity of facultative aerobes and anaerobes. *S. aureus, S. pyogenes*, and enterococci are the most common gram-positive aerobes. *Escherichia coli* is the most common gram-negative enteric organism. *Bacteroides* species and peptostreptococci are the most common anaerobes. The remaining 25% to 30% of deep necrotizing infections are monomicrobial. Most primary necrotizing soft tissue infections are monomicrobial. These infections are more fulminant and are notable for their acute onset, rapid progression, and systemic toxicity. *S. pyogenes* is the pathogen in more than half of monomicrobial infections; *S. aureus, C. perfringens, V. vulnificus*, and *P. aeruginosa* are less common.

Osteomyelitis

Osteomyelitis is an acute or chronic inflammatory process of the bone and its structures secondary to infection with pyogenic organisms.The infection associated with osteomyelitis may be localized or it may spread through the periosteum, cortex, marrow, and cancellous tissue. The bacterial pathogen varies on the basis of the patient's age and the mechanism of infection. The following are the 2 primary categories of acute osteomyelitis:

Hematogenous osteomyelitis is an infection caused by bacterial seeding from the blood. Acute hematogenous osteomyelitis is characterized by an acute infection of the bone caused by the seeding of the bacteria within the bone from a remote source. This condition occurs primarily in children. The most common site is the rapidly growing and highly vascular metaphysis of growing bones. The apparent slowing or sludging of blood flow as the vessels make sharp angles at the distal metaphysis predisposes the vessels to thrombosis and the bone itself to localized necrosis and bacterial seeding.

Direct or contiguous inoculation osteomyelitis is caused by direct contact of the tissue and bacteria during trauma or surgery. Direct inoculation (contiguous-focus) osteomyelitis is an infection in the bone secondary to the inoculation of organisms from direct trauma, spread from a contiguous focus of infection, or sepsis after a surgical procedure. Clinical manifestations of direct inoculation osteomyelitis are more localized than those of hematogenous osteomyelitis and tend to involve multiple organisms.

Additional categories include **chronic osteomyelitis and osteomyelitis secondary to peripheral vascular disease.** Chronic osteomyelitis persists or recurs, regardless of its initial cause and/or mechanism and despite aggressive intervention. Although listed as an etiology, peripheral vascular disease is actually a predisposing factor rather than a true cause of infection.
Classification of osteomyelitis represented in table 1.

Table 1. Classification of osteomyelitis

Classification
(1) Acute hematogenous osteomyelitis
(2) Sub acute osteomyelitis. Brodie's abscess is classified under this heading by some authors.
(3) Subacute epiphyseal osteomyelitis
(4) Chronic osteomyelitis
(5) Chronic-sclerosing osteomyelitis
(6) Osteomyelitis of miscellaneous group caused by rare organisms like salmonella, brucella, tuberculosis etc.

Excluding the axial skeleton, lower extremity osteomyelitis accounts for 90% of osteomyelitis cases and is much more common than upper extremity osteomyelitis, which accounts for 10% of extremity cases.
The most common bones involved in osteomyelitis in descending order are as follows:

- Tibia (50%)
- Femur (30%)
- Fibula (12%)
- Humerus (3%)
- Ulna (3%)
- Radius (2%)

The **pathophysiology** of osteomyelitis develops as a result of the particular anatomy and physiology of long bones. The medullary cavity of bones, where hematogenous infection begins, is encased in a rigid structure, which does not allow for the expansion of the inflammatory process. Progression of the infection restricts medullary blood supply. Passage of pus through the cortex elevates the periosteum and the resulting sub-periosteal abscess causes bony infarction as the cortical bone is supplied by end-arteries from the periosteum. The necrotic bone acts as a persistent foreign body. Healing by periosteal new bone formation forms an involucrum, which further isolates the sequestrum from antibiotic therapy. Poor blood supply and necrotic bone account for the need for prolonged antibiotic therapy in hematogenous osteomyelitis and the resistance of chronic osteomyelitis to therapy. Inflammation induces osteoclastic activity, which is partially responsible for the osteoporosis seen in x-rays of infected bone. The pathophysiology of post-trauma osteomyelitis shares some but not all of these characteristics.

The growth and development of long bones from the epiphyseal plate also determines the characteristics of bony infection. Prior to 2 years of age, blood vessels cross the physis allowing the initial metaphyseal infection to spread into the epiphysis or even the joint. After 2 years, the developing plate restricts this and the infection tends to spread into the diaphysis of long bones. A recent study found simultaneous septic joint involvement adjacent to an osteomyelitis in as many as 1/3 of cases and also questioned this age distribution. Since the neck of the femur is essentially intra-articular, osteomyelitis here is always associated with septic arthritis. Untreated septic arthritis, involving particularly the intra-articular proximal femoral and humeral growth plates, can cause profound growth disturbances. With plate closure after puberty, hematogenous osteomyelitis becomes uncommon in adults, only to recur again in the elderly. Particular biochemical features also determine the bacteriology of osteomyelitis. Staphylococcus aureus possesses receptors, which facilitate its adherence to and invasion of bone and joint tissues. These may account for the fact that staph aureus constitutes the majority of endogenous infections. Osteomyelitis from staph aureus carrying the pvi gene appears to result in higher complications. Streptococci are the second most common organisms, while specific vaccines against haemophilus influenzae have restricted this organism in those countries where they are used. While most hematogenous infections are single agent, post-traumatic infections often yield multiple bacteria. Pseudomonas is a common pathogen, after puncture wounds of the foot. Patients with sickle cell anemia have an increased incidence of osteomyelitis caused by more frequently by salmonella agents. Differentiating osteomyelitis from bone infarction, which requires only hydration and analgesia, is challenging.

A single pathogenic organism is almost always recovered from the bone. The most common bone isolates are *Staphylococcus* species, the most common gram-negative organism is *Pseudomonas aeruginosa,* and the most common anaerobes are *Peptostreptococcus* species.

Hematogenous osteomyelitis

In hematogenous osteomyelitis, local symptoms referable to bones are more frequently absent in neonates than in children. In adults, soft tissue findings may be more prominent than bony involvement.

In infants, local findings that may lead the clinician to suspect osteomyelitis are usually absent in neonates. When they develop, local findings can include decreased motion of a limb and edema (pseudoparalysis) and joint effusion adjacent to the bone infection (present in 60-70% of cases).

Systemic symptoms are frequently present in *S aureus* osteomyelitis but may be absent when other pathogens are involved.

Children with hematogenous osteomyelitis, in contrast with neonates, typically have the following systemic symptoms:

- Abrupt fever
- Irritability
- Lethargy
- Refusal to use the affected limb
- Local signs of inflammation present for 3 weeks or less

While this is the classic presentation, signs of systemic toxicity other than minimal temperature elevation are absent in 50% of children with osteomyelitis.

In adults, acute clinical presentations of fever, chills, swelling, and erythema over the involved bones are usually seen in acute hematogenous osteomyelitis.

Vertebral osteomyelitis is usually hematogenous in origin but may be secondary to trauma. A preceding history of urinary tract infection or injection drug use often is present. Other sources of infection include skin and soft tissue, respiratory tract, infected intravascular device site, endocarditis, dental infection, or unknown sources.

The patient usually presents with vague symptoms and signs consisting of dull, constant back pain and spasm of the paravertebral muscles. Localized pain and tenderness of the involved bone segments is present in at least 90% of cases. The pain is usually insidious and slowly progresses over 3 weeks to 3 months.

Chronic osteomyelitis

No exact criteria exist for defining when acute osteomyelitis becomes chronic. Clinically, the first bone infection is considered acute, and relapse of bone infection is labeled chronic. However, this simplistic classification is clearly inadequate. The hallmark of chronic osteomyelitis is the presence of dead bone (the sequestrum). Involucrum (reactive bony encasement of the sequestrum), local bone loss, persistent drainage, and/or sinus tracts are other common features of chronic disease.

The patient with chronic osteomyelitis commonly presents with chronic pain and sinus formation with purulent drainage. Fever is usually low grade or absent. The chronic infection usually does not progress or does so slowly. If a sinus tract becomes obstructed, the patient can present with a localized abscess, soft tissue infection, or both.

DIAGNOSIS

Lab Studies:

Routine laboratory test findings are usually nonspecific. These include CBC count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), renal and hepatic profile, and bone profile.

Blood culture results are positive in 50% percent of cases of acute osteomyelitis. A positive blood culture result in a patient with radiological findings consistent with osteomyelitis obviates the need for biopsy to obtain the specific microbiologic diagnosis.

Imaging Studies:

Plain films: Plain radiographs are relatively inexpensive, may be used to make the diagnosis, help in interpreting and choosing other studies, and allow one to exclude other conditions (eg, gas in the soft tissues). In uncomplicated acute infection, the triad of soft tissue swelling, bone destruction, and periosteal reaction is fairly specific for osteomyelitis and is sufficient to warrant a course of therapy (empiric until the microbiologic diagnosis has been established).

Plain films are generally insensitive for the diagnosis of acute osteomyelitis. This is in part due to the 2-3 weeks required for bone changes to be evident on plain films, although changes may be seen at this time on the other imaging modalities. Furthermore, in complicated situations, bone changes may not be distinguishable from those due to another process, such as a Charcot joint, fractures, or cancer. Thus, the diagnosis of acute osteomyelitis cannot be excluded if the plain film findings are negative. Further testing should be performed because early therapy is essential to reduce the formation of necrotic bone and the development of chronic osteomyelitis.

The primary findings are different in chronic osteomyelitis, which is characterized by bone sclerosis, periosteal new bone formation, and sequestra. It is difficult to distinguish active from inactive infection.

CT scan: This modality is used to evaluate an area in which focal findings are present on examination and plain films findings are negative. The CT scan (with and without contrast) is very accurate for detecting cortical destruction, intraosseous gas, periosteal reaction, and soft tissue extension.

MRI: This study is an alternative to CT scan and is especially useful in evaluating a patient for osteomyelitis in the vertebrae and in the infected foot. MRI provides useful anatomic detail in planning for surgical debridement, since it may show abscesses that need drainage, and can reduce the risk of operating on bland

cellulitis. MRI can also be used to delineate soft tissue/epidural involvement and spinal cord impingement that cannot be seen on nuclear medicine images.

Ultrasonography: Ultrasound findings consistent with osteomyelitis include fluid collection adjacent to the bone without intervening soft tissue, elevation of the periosteum by more than 2 mm, and thickening of the periosteum. Ultrasound may also improve the yield from fine-needle biopsies.

Scintigraphy: Multiple different nuclear medicine imaging procedures are available to evaluate for osteomyelitis, including bone scan, indium-labeled leukocyte scan, and bone marrow scan.

Three-phase bone scan

The 3-phase technetium bone scan is the test of choice in evaluating for acute osteomyelitis if the plain film findings are normal. In this situation, it has an estimated sensitivity and specificity of almost 95% and findings generally are positive in 2-3 days of infection. However, any process that results in increased bone turnover appears as a hot spot that is indistinguishable from osteomyelitis. These false-positive findings can occur with posttraumatic injury, following surgery, diabetic feet, septic arthritis, noninfectious inflammatory bone disease, cancer, healed osteomyelitis, and Paget disease.

Indium-labeled leukocyte scan: Indium-labeled leukocyte scanning uses white blood cells labeled with radioactive indium as the tracer. It accumulates at sites of inflammation or infection and in the bone marrow. It is not specific for bone. Since it accumulates in marrow, it is less sensitive for imaging those areas with red marrow (eg, the axial skeleton). The indium scan can also be used for the diagnosis of osteomyelitis at sites of fracture nonunion.

Bone marrow scan: The bone marrow scan uses technetium Tc 99m–labeled sulfur colloid as the tracer. It is taken up by the reticuloendothelial system, including the bone marrow, spleen, and liver. It allows one to image the marrow instead of the bone. Its main use is in conjunction with the indium-tagged white blood cell scan to evaluate for suspected osteomyelitis in the axial skeleton, where the presence of marrow decreases the accuracy of the indium scan.

Gallium citrate scanning: Gallium citrate scanning uses radioactive gallium citrate as the tracer. It acts as an analog of calcium and iron and attaches to transferrin to accumulate at sites of inflammation. Gallium imaging is the most sensitive and specific radionuclide scanning technique for vertebral osteomyelitis. A typical positive test result reveals intense uptake in 2 adjacent vertebrae with loss of the intervening disk space. In one study of 41 patients with suspected vertebral osteomyelitis, increased gallium uptake was detected in all of the 39 patients with biopsy-proven osteomyelitis.

Dual tracer scans: Dual tracer examinations combine an inflammation imaging tracer (indium or gallium) with an "anatomic" tracer (either technetium bone scan or technetium sulfur colloid marrow scan), with images being collected either sequentially or simultaneously. Sequential studies combine the sensitivity of the

bone scan (if findings are negative, no further imaging is done) with the specificity of the indium scan. Simultaneous studies combine the sensitivity and specificity of the indium scan and the bone scan to provide the anatomic detail needed to localize the infection to bone or soft tissue.
Other: Radiolabeled antigranulocyte antibodies are being investigated in an attempt to find a more accurate tracer for localizing infection.
Bone biopsy: The criterion standard for the diagnosis of osteomyelitis is open bone biopsy with histopathologic examination and cultures. Histopathologic features indicating osteomyelitis include necrotic bone with extensive resorption adjacent to an inflammatory exudate. Needle biopsy is commonly used to obtain bone for histopathologic analysis. If the clinical suspicion is strong and blood culture results and the needle biopsy findings are negative, the needle biopsy should be repeated or an open biopsy should be performed. In patients with compromised vasculature, a bone sample can be obtained at debridement for the histopathologic diagnosis of osteomyelitis.

Treatment
A single pathogenic organism is almost always recovered from the bone. The most common bone isolates are staphylococci, the most common gram-negative organism is P aeruginosa, and the most common anaerobe is Peptostreptococcus species. However, in immunocompromised patients, other organisms must also be considered, including fungi and mycobacteria.
Hematogenous osteomyelitis in children can usually be treated with antibiotic therapy alone. However, it is vital to identify a pathogen in order to select the optimal antibiotic therapy. Mismanagement with inappropriate antibiotic(s) encourages disease extension, necrosis, and sequestra formation. A bone biopsy for culture is necessary unless the patient has positive blood cultures along with radiographic findings consistent with osteomyelitis. After cultures are obtained, empiric antibiotics should be selected to cover the most probable pathogens.
Once the etiologic organism is identified, the antibiotic regimen should be modified, if needed, based upon susceptibility patterns. The patient should be treated for 4-6 weeks with appropriate antimicrobial therapy, dating from the initiation of therapy or following the last major debridement surgery. If the initial medical management fails and the patient is clinically compromised by a recurrent infection, medullary and/or soft tissue debridement is necessary in conjunction with another 4- to 6-week course of antibiotics.

Oral antibiotics can be used in pediatric hematogenous osteomyelitis. However, the child should receive 7-14 days of IV antibiotics or continue to receive IV antibiotics until systemic improvement occurs prior to changing to an oral regimen. The latest reports suggest the use of IV antibiotics for 3-4 days; this usually is

enough for the systemic manifestations to improve. In order to undergo oral therapy, the patient must be compliant and have close outpatient follow-up care. Absorption and activity of the orally administered antibiotic should be monitored using serum cidal levels. High doses of the quinolone class of antibiotics can cause articular cartilage damage in young animals, generating some concern about the long-term use of these agents in infants and children. Thus, under most circumstances, children with osteomyelitis should not be treated with a quinolone.

Surgical therapy: Surgical management of contiguous focus osteomyelitis can be very challenging. The principles of treating any infection are equally applicable to the treatment of infection in bone. These include adequate drainage, extensive debridement of all necrotic tissue, obliteration of dead spaces, stabilization, adequate soft tissue coverage, and restoration of an effective blood supply.

Surgical intervention is indicated in the following situations:

- ✓ The patient has not responded to specific antimicrobial therapy within 48 hours;
- ✓ Evidence exists of a persistent soft tissue abscess;
- ✓ Concomitant joint infection is suspected or diagnosed.

In adults with hematogenous osteomyelitis, a thorough intramedullary reaming and unroofing is usually performed with or without bone grafting. Soft tissues are reapproximated, and the limb is protected by external means (brace or cast) until the structural integrity of the bone is reestablished by normal remodeling.

Contraindications: Contraindications to debridement of infected bone are limited and include the following:

- ✓ In acute osteomyelitis in infants, the infection usually responds well to medical therapy alone. Surgery should be reserved for nonresponsive cases;
- ✓ Suspicion of malignancy or presence of secondary bone infection with malignancy should not be treated with debridement alone. Tumor workup and biopsy should be performed first. If infection cannot be eradicated or tumor is not resectable, amputation is indicated;
- ✓ Massive debridement is not recommended in the presence of sickle cell disease; usually a mixture of infection and avascular necrosis is present that may improve after good reperfusion. Massive debridement may produce very large defects that may not be easily amenable to reconstruction surgeries.

Local anesthesia is generally ineffective and should be avoided due to the change in the local Ph of the tissue that prevents the metabolism of the local anesthetics to the active ingredients.

However, this technique is labor intensive and requires an extended period of treatment, averaging 9 months in the device. The Ilizarov pins usually become infected, and the device is painful. Infected pseudoarthrosis with segmental osseous defects can be treated by debridement and microvascular bone transfers.

Vascularized bone transfer is also useful for the treatment of infected segmental osseous defects of long bones that are more than 3 cm in length. Vascularized bone transfers can be placed after 1 month without clinical evidence of infection.

Loss of bone stability, bone necrosis, and soft tissue damage frequently occur in contiguous focus osteomyelitis. Surgical debridement of infected bone and soft tissue provides specimens for culture and hastens eradication of the infection. Other steps in the surgical management of contiguous focus osteomyelitis should be tailored to the specific anatomy of the bone infection. When osteomyelitis is characterized by a full thickness cortical sequestration, patients usually can be treated with removal of the dead infected bone (bone saucerization). Bone grafting may be necessary to augment structural support. These patients may require external fixation for structural support while the bone graft incorporates. Complex reconstruction of both bone and soft tissue is frequently necessary.

In some cases, osteomyelitis progresses to an infection involving the entire diameter of the bone. These patients often require an intercalary resection of the bone in order to arrest the disease process. Since this advanced stage of osteomyelitis involves an entire through-and-through section of bone, a loss of bony stability occurs either before or after debridement surgery. As a result, treatment often must be directed toward establishing structural stability and obliterating debridement gaps by means of cancellous bone grafts or the Ilizarov technique. Vascularized bone grafting is the other possible treatment modality.

Complete debridement of all the devitalized bone and soft tissue, regardless of the size of the wound created, is essential for cure. Immobilization of the bone by cast or external fixations is a must.

The patient with extensive osteomyelitis and poor tissue oxygen perfusion usually requires some type of ablative surgery. Digital and ray resections (toe and corresponding metatarsal), transmetatarsal amputations, midfoot disarticulations, and Syme amputations (amputation of the foot with retention of the heel pad) permit the patient to ambulate without a prosthesis. The amputation level is determined by the vascularity of the tissues proximal to the site of infection and the requirements of a thorough debridement. The patient should be given 4 weeks of antibiotics when infected bone is surgically transected. Two weeks of antibiotics are prescribed when the infected bone is completely excised, but some residual soft tissue infection remains. However, when the amputation is performed proximal to the bone and soft tissue infection, the patient is only prescribed a 1- to 3-day antibiotic regimen.

V. Materials of activation of students
(questions, tasks, controversial situations, illustrative materials and other).

VI. Materials of selftraining of students on the topic of lecture: literature, questions, tasks.

Literature

1.Черенько М. П., Ваврик Ж. М. Загальна хірургія з анестезіологією, основами реаніматології та догляду за хворими. –К.: Здоров'я, 2004. –616 с.
2. Гостищев В. К. Общая хирургия Учебник для медицинских вузов. 4-е изд.,перераб., доп. и испр.- М.: ГЭОТАР-Медиа,2006.- 832 с.
3.Петров С.В. Общая хирургия Учебное пособие. 3-е изд.,перераб. и доп. – М.: ГЭОТАР-Медиа, 2007 – 768 с.
4.Anaya D., Dellinger E. Necrotizing soft-tissue infection: diagnosis and management //Clin Infect Dis.- 2007.- Vol.44, N5.- P.705-710.
5.Anderson D., Kaye K. Skin and soft tissue infections in older adults //Clin Geriatr Med.- 2007.- Vol.23, N3.- P.595-613.
6.Critical Care Infectious Diseases Textbook // by Jordi Rello (Editor), Jordi Vallés (Editor), Marin Kollef (Editor).- Springer; 1st edition (October 1, 2001).- 944 p.
7.Daum R.Clinical practice. Skin and soft-tissue infections caused by methicillin-resistant Staphylococcus aureus //N Engl J Med.- 2007.- Vol.26, N357(4).- P.380-90.
8.Frykberg R., Wittmayer B., Zgonis T. Surgical management of diabetic foot infections and osteomyelitis //Clin Podiatr Med Surg.-2007.- Vol.24, N3.- P.469-482.
9.Gabillot-Carré M, Roujeau J.Acute bacterial skin infections and cellulitis //Curr Opin Infect Dis.- 2007.- Vol.20, N2.- P.118-123.
10.Harwood P., Talbot C., Dimoutsos M. Early experience with linezolid for infections in orthopaedics// Injury.- 2006.-Vol.16, N4.
11.Hefny A., Eid H., Al-Hussona M. et al. Necrotizing fasciitis: a challenging diagnosis //Eur J Emerg Med.- 2007.- Vol.14, N1.- P.50-52.
12.Homer-Vanniasinkam S. Surgical site and vascular infections: treatment and prophylaxis// Int J Infect Dis. –2007.- Vol.11, Suppl 1.- S.17-22.
13.Plodr M., Cermak P., Ferko A. Soft tissue infection classification issues //Rozhl Chir.- 2006.- Vol.85, N11.- P.560-565.
14.Sabiston Textbook of Surgery //by Courtney M. Townsend, R. Daniel Beauchamp , B. Mark Evers, Kenneth Mattox.-Saunders; 17 edition (June 11, 2004).- 2416 p.
15.Salcido R. Necrotizing fasciitis: reviewing the causes and treatment strategies //Adv Skin Wound Care.- 2007.- Vol.20, N5.- P.288-293.
16. Schwartz's Principles of Surgery, 8/e (Schwartz's Principles of Surgery)// by F. Charles Brunicardi , Dana K. Andersen, Timothy R. Billiar, David L. Dunn,

John G. Hunter , Raphael E. Pollock .- McGraw-Hill Professional; 8 edition (October 14, 2004).- 2000 p.
17.Steer A., Carapetis J. Acute hematogenous osteomyelitis in children: recognition and management //Paediatr Drugs.- 2004.- Vol.6, N6.- P.333-346.
18.Taviloglu K., Yanar H. Necrotizing fasciitis: strategies for diagnosis and management //World J Emerg Surg.- 2007.- Vol.7, N2.- P.1-19.
19.Termaat M., Raijmakers P., Scholten H. et al. The Accuracy of Diagnostic Imaging for the Assessment of Chronic Osteomyelitis: A Systematic Review and Meta-Analysis//The Journal of Bone and Joint Surgery (American).- 2005.- Vol.87.- P.2464-2471.
20. Yilmazlar T., Ozturk E., Alsoy A., Ozguc H. Necrotizing Soft Tissue Infections: APACHE II Score, Dissemination, and Survival //World J Surg. – 2007.- N7.

A lecture is prepared on materials of meeting of cyclic methodical commission of Bogomolets National Medical University from 21 June 1998 in conformity with recommendations of Department of pedagogics and pedagogical psychology (associate professor Mileryan V.E.)