Bacteriology of Necrotizing Fasciitis

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Meleney presented the first major series of patients with necrotizing fasciitis in 1924 [*1*]. His careful bacteriologic studies disclosed a beta-hemolytic streptococcus cultured from each of his patients, and he named the disease "acute hemolytic streptococcal gangrene." Meleney's studies were limited by the bacteriologic technic of his time; some groups of bacteria may not have been cultured and others were incompletely identified. Gram-stained lesion smears, which allow observation of the kinds and proportions of bacteria originally present, were not reported.

Since Meleney, few authors have focused on the bacteria involved in the pathogenesis of necrotizing fasciitis; most studies have concentrated on the clinical presentation and therapeutic implications. Bacteriology and culture reports often have been incomplete, and rarely have careful anaerobic culture technics been employed. The present study investigates the bacteriology of necrotizing fasciitis in patients by the examination of gram-stained smears as well as by both aerobic and anaerobic collection and culture methods.

Clinical Material and Methods

Sixteen patients undergoing operative debridement for necrotizing fasciitis at San Francisco General Hospital during the last five years were reviewed. Only intraoperative culture results were analyzed. Aerobic and anaerobic cultures and gram-stained smears were made immediately of the fluid aspirated and tissue debrided from the center of the lesion. Exudates were aspirated by syringe and needle, freed of air bubbles, and transferred to anaerobic transport tubes containing oxygen-free CO_2. At least 5 cc of tissue was packed into an opened anaerobic transport tube. The tube was held upright in order to retain the CO_2 and then restoppered. Specimens were usually cultured within 1 hour after they were removed from the patient. Brucella agar (Pfizer) supplemented with 6 per cent sheep blood, 10 μg/ml vitamin K, and 5 μg/ml hemin with and without 100 μg/ml kanamycin and chopped meat broth were used for primary culture of anaerobes. Tryptic digest casein-soy blood agar, chocolate agar, and MacConkey medium were used for culture of aerobes. The specimens were incubated at 36°C both aerobically and anaerobically in a glove box containing 10 per cent CO_2, 5 per cent H_2, and 85 per cent N_2 or in Gas Pak® jars (Bioquest, Cockeysville, MD 21030). Identification of anaerobes was made by means of gas chromatography for short chain fatty acids and dicarboxylic acids, and with prereduced anaerobically sterilized test media defined in the Virginia Polytechnic Anaerobe Manual [*3*]. Other bacteria were identified according to the American Society for Microbiology Manual of Clinical Microbiology [*4*].

The diagnosis of necrotizing fasciitis in these cases was based on the presence of edema and necrosis, often with partial liquefaction of subcutaneous fat and adjacent deep fascia. These tissues were usually gray or gray-green. Underlying muscle had normal vascularity and was usually completely spared or showed slight edema. Only if the fascial envelope of the muscle was completely involved did muscle necrosis develop. Overlying skin was usually only slightly erythematous and edematous. When the underlying fat and fascial necrosis was extensive, cyanosis and gangrene of the skin occurred. All patients demonstrated severe systemic toxicity with fever ranging 102 to 105°F. Alterations in mentation, ranging from disorientation to obtundation, were evident. Microscopic examination of debrided tissue showed intense polymorphonuclear cell infiltrate, focal necrosis, and microabscesses in the fascia and subcutaneous tissue. Often, complete thrombosis of the small arterioles and venules of the subcutaneous fat was present. Adjoining muscle and skin showed comparatively little inflammation. Figures 1, 2, 3, and 4 show the clinical appearance of the lesion in one patient.

Of the sixteen patients with severe necrotizing fasciitis reviewed, eleven had infection primarily in the upper extremity and/or shoulder girdle, one each had infections involving the chest and back, and three had involvement of the abdominal wall. Five patients were in septic shock. The source of infection was subcutaneous or intravenous injection of illicit drugs, most commonly heroin, in nine patients, minimal external trauma with skin disruption in three, and postoperative wounds in three. One patient was comatose and could give no history.

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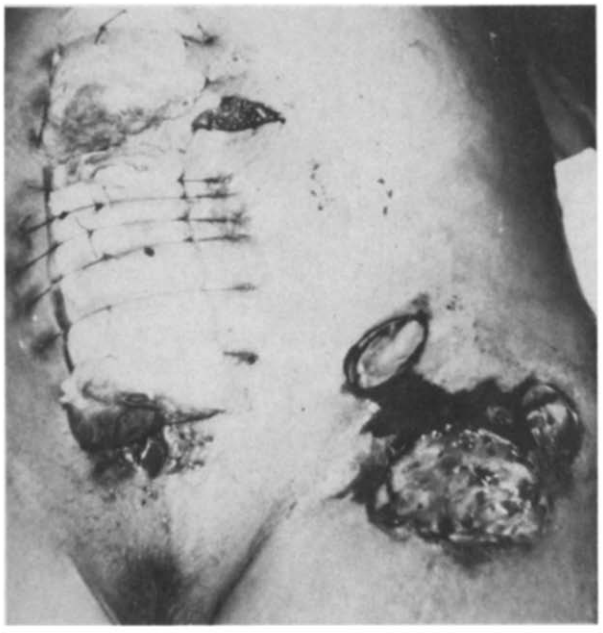

Figure 1. Appearance of lower abdomen and left groin fourteen days after gynecologic surgery. Ulcerative lesion in groin developed spontaneously. Note gangrene of skin edges. Abdominal wall and thigh are markedly edematous and erythematous.

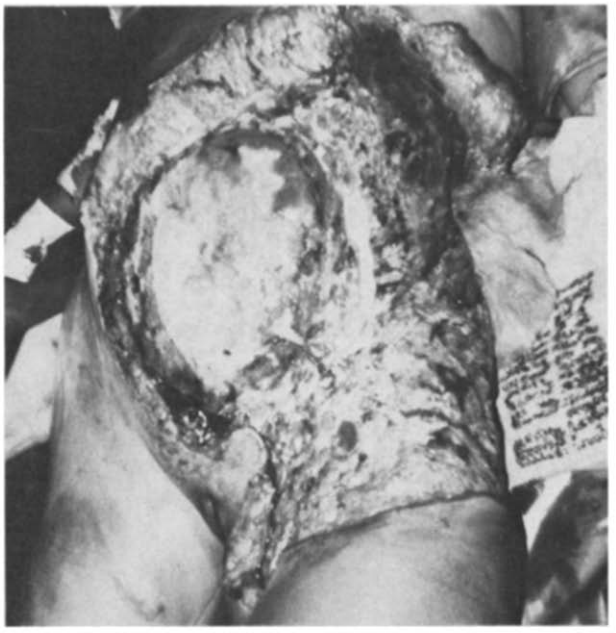

Figure 2. Extensive debridement necessary to remove devitalized subcutaneous tissue and fascia. Full thickness loss of abdominal wall in midline has been replaced with Marlex® mesh.

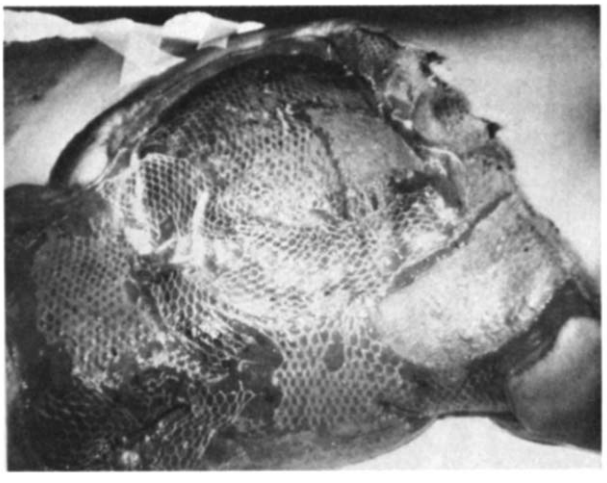

Figure 3. Skin grafting of debrided area three weeks postoperatively. Note grafting directly over Marlex® mesh.

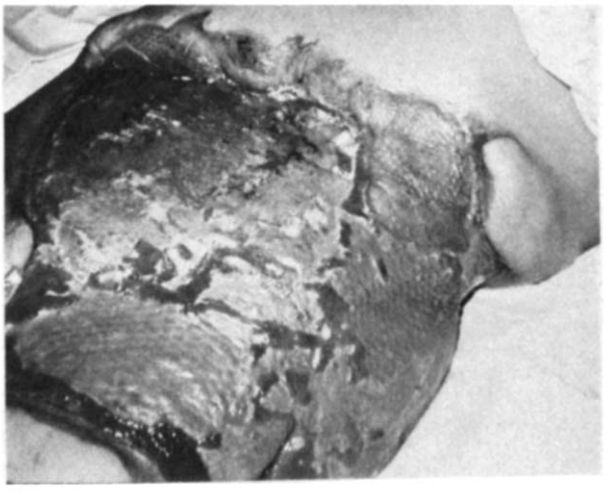

Figure 4. Appearance of wound six weeks postoperatively. Note amount of wound contracture in comparison with Figure 2.

Results

Two to eleven organisms were identified in fifteen of the sixteen patients; in one patient, only a single organism, group A streptococcus, was isolated. A total of seventy-five bacterial species were identified in the sixteen patients. (Table I.)

The organisms cultured conformed with those seen on the gram stain. At least one facultative streptococcus was recovered from all but one patient. One or more bacteroides were isolated from ten patients and a peptostreptococcus from eight patients; both occurred together with facultative streptococci. The results of the culture distinguished two distinct groups of patients:

Type I. Thirteen patients. Anaerobic bacteria and facultative anaerobic bacteria, such as enterobacteriaceae and streptococci other than group A, were isolated in combination. In each of these patients, at least one anaerobic organism was cultured in combination with a facultative anaerobe. In none of the patients were anaerobic bacteria isolated alone.

TABLE I Bacteria Cultured from Necrotizing Fasciitis

	Total No. of Isolates
Anaerobic Bacteria (gram-positive)	
Peptostreptococcus species	8
Peptococcus species	4
Eubacterium species	1
Propionibacterium species	4
Clostridium perfringens	3
Clostridium (not perfringens)	1
Anaerobic Bacteria (gram-negative)	
Bacteroides (total)	15
Bacteroides melaninogenicus	5
Bacteroides fragilis	2
Bacteroides corrodens	2
Bacteroides species	6
Fusobacterium necrophorum	1
Facultative Bacteria	
Streptococcus (total)	22
Streptococcus pyogenes (group A)	3
Streptococcus agalactiae (group B)	1
Streptococcus (group D)	
Enterococcus	4
Streptococcus bovis	2
Streptococcus angiosus (group F)	1
Streptococcus, beta-hemolytic (not group A, B, or F)	3
Streptococcus intermedius	1
Streptococcus, alpha-hemolytic (not group D)	7
Staphylococcus aureus	1
Staphylococcus epidermidis	1
Enterobacteriaceae (total)	12
Escherichia coli	4
Citrobacter freundii	1
Klebsiella pneumoniae	2
Enterobacter cloacae	2
Serratia marcescens	2
Proteus mirabilis	1
Aerobic Bacteria	
Pseudomonas aeruginosa	2

Note: All bacteria isolated from each specimen are counted in this table. Individual patient's cultures may contain more than one bacteroides, enterobacteriaceae, or other group.

Type II. Three patients. Group A streptococcus (Streptococcus pyogenes) was isolated alone in one patient, occurred in combination with Staphylococcus aureus in one, and with epidermidis in one. In none of these three cases were anaerobic bacteria or enterobacteriaceae cultured.

Group A streptococcus and staphylococcus, characteristic isolates in type II patients, were not isolated from any of the type I patients. Enterobacteriaceae, which were cultured from nine of the thirteen type I patients, included Escherichia coli (4 patients), Citrobacter freundii (1), Klebsiella pneumoniae (2), Enterobacter cloacae (2), Serratia marcescens (2), and Proteus mirabilis (1). Facultative streptococci other than group A streptococcus were cultured from twelve of thirteen patients. These included Streptococcus agalactae (group B, 1 patient), enterococcus (group D, 4), Streptococcus bovis (group D, 2), S angiosus (group F, 1), S intermedius (1), streptococcus (beta-hemolytic, not group A, B, or F; 3), and streptococcus (alpha-hemolytic, not group D; 6). Clostridia were recovered in four patients but were not the predominant bacterium. In none of these patients was muscle involvement significant.

Combinations of bacteria that were observed in this study are shown in Table II. The associations that occurred most often are bacteroides + streptococcus (not group A) in 10 instances, bacteroides + peptostreptococcus in 7, bacteroides + streptococcus (alpha-hemolytic, not group D) in 6, bacteroides + enterobacteriaceae in 6, and peptostreptococcus + enterobacteriaceae in 5.

Two cases of bacteremia occurred, one caused by Citrobacter freundii and the other by Streptococcus angiosus, organisms also isolated from the site of the necrotizing fasciitis. Citrobacter also was isolated with a bacteroides species from an epidural abscess occurring concomitantly with necrotizing fasciitis and bacteremia.

Comments

Each of the patients in this series had extensive necrotizing fasciitis with fascial and subcutaneous necrosis and thrombosed microvasculature. The clinical pictures of type I or type II patients were indistinguishable on gross or microscopic anatomic appearance. Still, two entirely different types of bacteria were isolated from the lesions.

In Meleney's original report of patients with necrotizing fasciitis [*1,5*] a "hemolytic streptococcus" invariably was found, sometimes in pure culture. In our study, group A streptococcus (S pyogenes) was present alone or as the predominant organism in conjunction with a staphylococcus in three patients. No anaerobic bacteria or enterobacteriaceae were isolated from these lesions. The association of hemolytic streptococcus and staphylococcus has been noted in patients reported on by Meleney [*5*] and Wilson [*6*]. The bacterial culture resulting from these three patients in our study resemble those described by Meleney [*1,5*] and in other early reports [*6–9*].

Although the "hemolytic streptococcus" Meleney found in the lesions of necrotizing fasciitis are commonly assumed to be beta-hemolytic group A streptococcus, this remains unknown, since Lancefield [2] had not developed his system for grouping streptococci at the time of Meleney's study. Complete or beta-hemolytic reaction on sheep blood agar can be produced by many different groups of streptococci. Beta-hemolytic streptococci were isolated from the lesions in eight patients in this study: three were group A; one was group B; one was group F; and three were not grouped, but were not A, B, or F.

TABLE II Bacterial Associations Observed in Necrotizing Fasciitis

	Frequency	All Str, not Group D	Group D Str	αH Str not Group D	Group A Str	S	E	B	C	P	PS
Frequency		12	4	6	3	2	9	10	4	4	8
Bacteroides (B)	10	10	4	6	0	0	6	—	2	4	7
Fusobacterium	1	1	1	1	0	0	1	1	1	1	1
Clostridium (C)	4	3	3	1	0	0	4	2	—	0	1
Propionibacterium	4	4	1	3	0	0	2	3	0	2	4
Peptococcus (P)	4	4	1	3	0	0	2	4	0	—	4
Peptostreptococcus (PS)	8	8	2	5	0	0	5	7	1	4	—

Note: When more than 1 organism of a genus or group is isolated from 1 specimen, it is counted only once in this table.
Note: Str = streptococcus; αH = alpha-hemolytic; S = staphylococcus; E = enterobacteriaceae.

Entirely different organisms were isolated from the remaining thirteen patients, and these seemed to represent similar clinical entities but different bacterial etiologies. Neither beta-hemolytic group A streptococcus nor staphylococcus was found in these lesions, but they did contain from three to eleven other bacterial species, including anaerobic bacteria in association with a wide variety of streptococci other than group A (5 of which showed beta-hemolysis) and enterobacteriaceae. Bacteroides and peptostreptococci were the most frequently isolated anaerobes.

Meleney attempted to culture anaerobic bacteria but only recovered Clostridium perfringens from one patient. It is likely that the methods he used for collection and cultivation did not permit the recovery of the more fastidious anaerobes. Since Meleney and nearly all other investigators of necrotizing fasciitis failed to report results of gram-stained smears, it is impossible to know whether anaerobic bacteria were present but not cultured. Crosthwait, Crosthwait, and Jordan [7] reported the isolation of enterobacteriaceae from necrotizing fasciitis but did not cultivate anaerobic bacteria. Rein and Cosman [*10*] reported one case of necrotizing fasciitis in which bacteroides was cultivated. Menda et al [*11*] reported isolation of a wide variety of anaerobic bacteria, including bacteroides, fusobacterium, and peptostreptococcus, isolated from ten patients with necrotizing fasciitis. They invariably found that coliform bacteria (enterobacteriaceae) were isolated from specimens also containing bacteroides and fusobacteria. In our study, the isolation of enterobacteriaceae with bacteroides or fusobacteria was not invariable (Table II), occurring only in type I patients. However, a streptococcus of some kind was isolated from each patient. The invariable presence of facultative bacteria in the cultures of necrotizing fasciitis in *this* study and that of Menda et al [*11*] and the absence of cases in which only anaerobes were present suggests that facultative anaerobic bacteria, such as streptococci and enterobacteriaceae, are an important factor in the development of these lesions.

The facultative bacteria may be the most significant agent, or the causal mechanism may be a synergistic action between the facultative bacteria and anaerobic bacteria, neither of which can produce the lesion alone. A synergistic action of mixtures of bacteria is well known in experimental and natural infections. Hite, Locke, and Hesseltine [*13*] demonstrated a synergistic action of Fusobacterium necrophorum and Streptococcus liquefaciens in producing necrotic abdominal wall lesions in mice. Roberts [*14,15*] showed that infective bulbar necrosis of sheep is produced by the synergistic association of Fusobacterium necrophorum and Corynebacterium pyogenes. The latter organism produces a nutrient necessary for the growth of the fusobacterium, while the fusobacterium produces a leukocidal toxin which protects both organisms from phagocytosis. Recently, Weinstein et al [*16*], working with experimental peritoneal infections in rats, have observed complex interactions among the gut flora introduced intraperitoneally which suggest synergistic action. It is commonly assumed that facultative bacteria also assist the growth of anaerobes by utilizing oxygen, by diminishing the oxidation-reduction potential, or by supplying catalase. The isolation of group A streptococcus (Streptococcus pyogenes), either alone or in the presence of staphylococcus, from lesions of necrotizing fasciitis suggests that the group A streptococcus has full pathogenic capacity to produce this lesion.

It is apparent from the results obtained in this study that a gram-stained smear, as well as careful anaerobic collection and cultivation of tissue or a lesion aspirate, are essential in the diagnosis and management of necrotizing fasciitis. Surgical therapy

with radical excision and full debridement of tissue to the limits of the involved fascial and subcutaneous tissue is the most important element of therapy. Antimicrobial therapy should be intravenous or parenteral. Selection of antimicrobials can be guided by the gram stain: penicillin G remains the treatment of choice for group A streptococcal infections; methicillin or other penicillinase-resistant penicillin may be used when staphylococcus is isolated. For those patients in whom the smear shows mixed bacterial flora, the initial treatment should include not only penicillin but also an aminoglycoside with broad spectrum activity, such as gentamicin or tobramycin, to inhibit enterobacteriaceae, enterococcus, or other streptococci with similar resistance patterns. Additional antimicrobial coverage of the anaerobic bacteria should be provided by large doses of intravenous penicillin and clindamycin or chloramphenicol in combination therapy with the aminoglycoside. Many of the anaerobic bacteria other than Bacteroides fragilis are susceptible to penicillin G. Broad coverage of the anaerobic bacteria, including B fragilis, is best provided by chloramphenicol or clindamycin. Metronidazole, which both inhibits and destroys most anaerobic bacteria, is under investigation in the treatment of anaerobic infections.

Summary

Sixteen patients with necrotizing fasciitis were observed under clinical and laboratory conditions for collection, preservation, and culture that permitted optimal retrieval of anaerobes. The clinical observations of necrosis of fascia, subcutaneous fat and skin with thrombosis of the microvasculature, and absence of myonecrosis were clearly apparent in these patients. Two clear-cut groups of culture and gram stain results were found, suggesting that the clinical entity of necrotizing fasciitis can occur after infection by different infecting organisms. The cultivation of Streptococcus pyogenes (group A), either alone or in combination with staphylococcus, in three patients conforms to the culture results found by Meleney [1] in his original description.

References

1. Meleney FL: Hemolytic streptococcus gangrene. *Arch Surg* 9: 317, 1924.
2. Lancefield RC: A serological differentiation of human and other groups of hemolytic streptococci. *J Exp Med* 57: 571, 1933.
3. Holdeman LV, Moore WEC: Anaerobe Laboratory Manual, 3rd ed. Anaerobe Laboratory, Virginia Polytechnic Institute, Blacksburg, Virginia, 1975.
4. Lennette EH, Spaulding EH, Truant JP: Manual of Clinical Microbiology, 2nd ed. American Society for Microbiology, Washington, DC, 1974.
5. Meleney FL: Differential diagnosis between certain types of infectious gangrene of the skin. *Surg Gynecol Obstet* 56: 847, 1933.
6. Wilson B: Necrotizing fasciitis. *Am Surg* 18: 416, 1952.
7. Crosthwait RW Jr, Crosthwait RW, Jordan GL Jr: Necrotizing fasciitis. *J Trauma* 4: 149, 1964.
8. Rea WJ, Wyrick WJ: Necrotizing fasciitis. *Ann Surg* 172: 957, 1970.
9. Meade JW, Mueller CB: Necrotizing infections of subcutaneous tissue and fascia. *Ann Surg* 168: 274, 1968.
10. Rein JM, Cosman B: Bacteroides necrotizing fasciitis of the upper extremity. *Plast Reconstr Surg* 48: 592, 1971.
11. Menda KB, Norsen J, Kallick C, Levin S: Necrotizing fasciitis: bacteriology and clinical features. Abstract #275, Abstracts of 14th Interscience Conference on Antimicrobial Agents and Chemotherapy, September 1974.
12. Finegold SM, Bartlett JG, Chow AW, et al: Management of anaerobic infections. *Ann Intern Med* 83: 375, 1975.
13. Hite KH, Locke M, Hesseltine HC: Synergism in experimental infections with nonsporulating anaerobic bacteria. *J Infect Dis* 84: 1, 1949.
14. Roberts DS: The pathogenic synergy of Fusiformis necrophorus and Corynebacterium pyogenes. I. Influence of the leukocidal exotoxin of F necrophorus. *Br J Exp Pathol* 48: 665, 1967.
15. Roberts DS: Synergic mechanisms in certain mixed infections. *J Infect Dis* 120: 720, 1969.
16. Weinstein WM, Onderdonk AB, Bartlett JG, Louie TJ, Gorbach SL: Antimicrobial therapy of experimental intraabdominal sepsis. *J Infect Dis* 132: 282, 1975.

Discussion

William P. Mikkelsen (Los Angeles, CA): Today, most of us ascribe synergism between an anaerobic streptococcus and a hemolytic staphylococcus as Meleney's cutaneous and subcutaneous infection, although he actually described a variety of streptococcal and staphylococcal infections, both aerobic and anaerobic. The aggressiveness, sensitivity to antibiotics, and host resistance of the organisms determine the extent and location of the infection, regardless of their synergism or anaerobic or aerobic nature. Thus, it might be more appropriate to describe the tissues involved in the infection and the causative organisms, thereby including gas gangrene and streptococcal cellulitis as well as the more indolent infections caused frequently by multiple organisms.

In all cases of extensive soft tissue infection, treatment must be aggressive, involving extensive drainage and debridement and administration of appropriate antibiotics in massive doses. Even then, lethality can be excessive, ranging from approximately 30 per cent in typical Meleney's disease to as high as 75 per cent in the deeper infections. Diabetes and old age especially contribute to a high mortality. Therefore, the authors' mortality rate, two of sixteen patients, is probably the best in the literature.

That aerobic organisms are reported to cause only so-called necrotizing fasciitis in the literature is probably due to faulty anaerobic culture technics, as stated by the authors. The large number of separate species that may be involved in a given case (as many as 11 in 1 patient) may make antibiotic management even more difficult. Since so many of these desperately ill patients harbor anaerobic bacteroides, it is probably justifiable to employ chloramphenicol as the antibiotic of first choice.

Samuel E. Wilson (Los Angeles, CA): In three of the authors' patients, necrotizing cellulitis and fasciitis occurred in postoperative wounds. The clinical setting for necrotizing cellulitis of the anterior abdominal wall begins with laparotomy for intraabdominal sepsis, after which the surgeon closes all layers of the skin and subcutaneous tissue. The prospective study by Cruse (*Arch Surg* 107: 206, 1973) clearly shows that closure of a grossly contaminated wound results in a delayed abscess rate in excess of 38 per cent. The organisms contaminating the subcutaneous space are of enteric origin and are composed of polymicrobial, mixed aerobic, and anaerobic flora. Closure of the wound provides a dead space with the anaerobic conditions necessary for this mixed bacterial growth. Two enzymatic mechanisms are responsible for the invasive nature of the process: heparinase, elaborated by bacteroides species and Fusobacterium mortiferum, results in intravascular coagulation (*Infect Immun* 8: 911, 1973) and collagenase, produced by clostridium and melaninogenicus, causes tissue destruction (*J Bacteriol* 81: 614, 1961). In a small number of patients, this process continues to necrotize fasciitis; however, it will be prevented by the simple technic of delayed wound closure.

Norman McK. Christensen (Eureka, CA): We have seen two patients with necrotizing fasciitis in our small community hospital within the past six months. The first patient was a twenty-eight year old man who, four days after appendectomy, became febrile and developed rapidly increasing erythema and edema about the wound. He underwent reoperation, and a 30 × 20 cm area of skin, subcutaneous tissue, and fat was debrided. Alpha streptococcus, Bacteroides fragilis, and Escherichia coli (type I) were cultured. The second patient was a fifty-six year old man who underwent repair of a recurrent hernia. On the first postoperative day he was febrile but asymptomatic. On the sixth postoperative day, approximately 6 cm in diameter of skin, subcutaneous fat, and fascia were debrided and coagulase-positive staphylococcus and peptostreptococcus (type I) were cultured from the wound.

This process, therefore, can have contrasting presentations. Edema and erythema may increase manifestly or the wound may look almost normal and the infection become apparent only when the subcutaneous tissue and fascia are inspected.

Arthur J. Donovan (Mobile, AL): Wide debridement and drainage are essential in the treatment of necrotizing fasciitis. This emphasis is not intended to minimize the importance of appropriate antibiotic therapy. The dissection must be carried into normal tissue, and all of the necrotic tissue must be excised. In some instances, when the infection is in the distal extremity, this may best be accomplished by amputation.

These infections are frequently encountered in patients with diabetes and may arise from perianal sepsis. Where extensive perianal debridement has been necessary, proximal diverting colostomy is performed.

The wound should be reexamined in the operating room, usually under anesthesia, within 24 hours of initial debridement. Further drainage and debridement are often necessary and must be repeated as frequently as necessary thereafter until progression has ceased and all necrotic tissue has been debrided. The surgeon must not be concerned with the extent of the resulting defect. Arrest of the spreading lethal infection must be of first priority.

Once the necrotizing infectious process is controlled, the wound can be treated as an extensive burn. Use of the Hubbard tank is most important in preparing the wound for closure. Nutritional support and all technics available for wound closure must be employed.

F. William Blaisdell (closing): The antibiotic therapy we recommend for the two types of infection are: for type I infection, high dose penicillin combined with either tobramycin or gentamicin, and clindamycin or chloromycetin if there is bacteroides infection; for type II, methicillin and penicillin.

Gram staining should be part of the initial evaluation of these patients. This provides information regarding the relative frequency and significance of the organisms so that one can know which of the bacteria cultured were predominant in the initial infection.

As Doctor Mikkelsen mentioned, only two of sixteen patients died. Most of these patients, however, were critically ill and would have died five to ten years ago.

(Slide) This is an example of the appearance of the infection after debridement. This patient had injected drugs about his shoulder. A preliminary incision laterally in the axilla identified the process there. Another incision was made over the pectoral muscles and demonstrated the greenish appearance of the pectoral fascia. Adequate debridement involved removal of much of the tissue about his shoulder.

(Slide) The second patient was referred to us when, after hysterectomy, she developed a slough of much of the abdominal wall. I suspect one of the retention sutures caught a loop of bowel. At the time of presentation, there was necrosis of the groin around the lower midline abdominal incision. The infection was found to extend from the axilla to the upper thigh and involved all the fascia of the muscles of the anterior abdominal wall. This resulted in muscle necrosis, which usually does not occur until the fascial envelope is completely infected. Marlex mesh was used to close the massive abdominal wall defect. (Slide) Granulations formed through the Marlex, and skin was grafted over this. After a very stormy postoperative course she recovered and is doing well.

I agree with Doctor Donovan's emphasis on the importance of reoperation. The first debridement is often inadequate; the surgeon is reluctant to remove all the tissue required to control the infection. These patients should undergo operation routinely within 12 to 24 hours after initial debridement. The margins of the wound should be inspected and additional debridement carried out as necessary. Even if the second operation discloses a clean wound, three to five inspections may be necessary to ensure that the infection is under control.