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# Cefoxitin: Clinical Evaluation in Thirty-Eight Patients

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Clinical and bacteriological efficacy, patient tolerance, and toxicity of cefoxitin, a beta-lactamase-resistant cephamycin, were evaluated in 38 patients; 13 had soft tissue infection, 12 had pneumonia, 3 had urinary tract infection, 2 had peritonitis, and 4 had miscellaneous infections. In five patients, infection was clinically evident, though not bacteriologically proven. The latter patients were evaluated with regard to tolerance and toxicity only. Among the 34 infections in 33 patients, 71% were considered clinically cured; 86% of those patients who could be recultured were bacteriologically cured. Phlebitis was noted in 32% of the total group, and eosinophilia was observed in 16%. Unexplained deterioration in renal function occurred in two patients. Mean peak cefoxitin levels in serum were 72 μg/ml 30 min after a 2-g infusion and 32 μg/ml 30 min after a 1-g infusion. Cefoxitin was more active against facultatively and obligately anaerobic gram-negative organisms isolated from these patients than was cephalothin.

The search for cephalosporin derivatives that are resistant to cephalosporinases and nontoxic to humans has led to the development of cefoxitin, a new beta-lactamase-resistant cephamycin (16, 12). The present study was undertaken to determine the clinical efficacy, patient tolerance, and safety of cefoxitin in the therapy of infections caused by susceptible organisms.

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## MATERIALS AND METHODS

**Patients.** Patients studied were hospitalized at the Wadsworth Veterans Administration Hospital. In each case, clinical findings and either prior culture or Gram-stained material suggested infection with bacteria likely to be susceptible to the cephalosporins or shown to be susceptible to cefoxitin itself. Informed written consent was obtained from the patients or their relatives.

Cefoxitin was administered via scalp vein infusion set or short catheter as 1 or 2 g, diluted in 100 ml of either normal saline or 5% dextrose in water, given over 20 to 40 min. Doses were administered either every 4 or 6 h. Infusion sites were changed at least every 72 h or at the first sign of inflammation.

Possible toxicity was monitored by daily examination of patients; determinations of complete blood cell count, Coombs test, urinalysis, serum glutamic oxalacetic and glutamic pyruvic transminases, bilirubin, alkaline phosphatase, electrolytes, glucose, creatinine, and blood urea nitrogen determinations were performed at frequent intervals. Appropriate cultures were obtained from all patients before and, when possible, during and after termination of therapy. In all but three suspected cases of pneumonia, transtracheal aspiration was performed to obtain specimens free of oropharyngeal flora.

Efficacy of therapy was evaluated in two principal ways: clinical outcome and bacteriological outcome. Follow-up cultures were not feasible in patients with soft tissue infections who showed resolution, and patients with pneumonia were not subjected to repeat transtracheal aspiration. Clinical cure was defined as recovery from infection achieved either by cefoxitin therapy alone or by local measures, including surgery, in addition to cefoxitin. Eradication of the susceptible initially isolated pathogens without ingrowth of clinically significant organisms on subsequent culture was regarded as evidence of bacteriological cure.

**Cefoxitin assays.** Blood specimens were collected, and the serum was separated aseptically and frozen for subsequent assay. Assays were performed on the sera of 19 of the 38 patients reported here and from an additional 11 patients subsequently treated. Specimens were obtained immediately at the end of infusion, at 30, 60, and 90 min, and at 2, 4, and 6 h after the end of infusion. Eighty-three serum specimens, three ascitic fluid samples, and two cerebrospinal fluid samples were assayed. Assays were performed by the Infectious Disease Department, UCLA Center for the Health Sciences, Los Angeles, Calif., using a modification of the cup-plate technique of Goodwin et al. (6) with *Micrococcus lysodeikticus* as the test organism. Forty duplicate specimens were assayed by Merck, Sharp & Dohme, West Point, Penn., with comparable results.

**Susceptibility testing.** Testing of susceptibility to cefoxitin and to cephalothin was performed on 67

clinical isolates by agar or broth dilution technique (5, 15).

## RESULTS

Thirty-eight patients (one female, the others male) were treated with cefoxitin between June 1975 and January 1976. The age range was from 21 to 79 years, with a median of 52. Duration of therapy ranged from 2 to 31 days with a median of 8 days. Thirty-three patients received either 4 or 6 g per day. Another 11, considered here for pharmacological evaluation, received 8 to 12 g per day.

Twelve patients had pneumonia, 13 had soft tissue infection, 2 had urinary tract infection, 2 had peritonitis (one with a concurrent urinary tract infection), and 4 had miscellaneous infections. Seven patients were bacteremic. No pathogenic organisms were cultured in five patients despite clinical signs of infection. Bacteriological and clinical outcomes are summarized in Table 1.

**Pneumonia.** Bacterial isolates from the 12 patients with pneumonia are listed in Table 2.

Of the 12 patients, 10 were clinically cured. Two patients died during therapy. Patient 2, admitted because of pneumonia and alcohol withdrawal, became acutely oliguric 6 h after initiation of therapy; urine output resumed after fluid challenge, and cefoxitin treatment was continued at reduced dosage. The patient suffered a cardiorespiratory arrest 30 h later. At autopsy no organisms were cultured, and the bronchopneumonia, though present, was resolving. Patient 18, with metastatic bronchogenic carcinoma and aspiration pneumonia, had become afebrile and appeared improved after 2 days of therapy. On day 3 of therapy, he had a sudden cardiorespiratory arrest and died.

Follow-up sputum cultures were obtained in seven patients, and, in five, susceptible organisms were eradicated. Despite clinical resolution, *S. pneumoniae* was recultured from the sputum of one, patient 24, on day 6 of therapy, and reemergence of *S. aureus* and *K. pneumoniae* occurred after termination of therapy in the second, patient 16. There was, however, no recurrence of clinical symptoms.

**Soft tissue infection.** Of 13 patients with soft tissue infection, 11 were considered clinically cured. Nine required drainage of abscesses, two had diagnostic needle aspiration, and another had drainage of an abscess followed by amputation. Bacterial isolates are listed in Table 3. One of the two treatment failures, patient 29, who had an extensive iliofemoral abscess, showed some resolution of the primary process but died of noninfective pulmonary complications after termination of therapy. The second, patient 26, who had a femoral abscess, had eradication of susceptible organisms but showed only moderate clinical improvement.

Follow-up cultures available from seven patients showed six to be bacteriologically cured. In the seventh, patient 25, the initial culture had been sterile, although pus cells and many gram-positive cocci in chains had been seen on initial Gram stain. Reaspiration of the cellulitis after 2 days of therapy yielded moderate growth of group A streptococci. The Gram stain showed a decrease in the number of pus cells and organisms, and this patient progressed to complete clinical resolution on therapy.

**Urinary tract infection.** All three patients with urinary tract infection (one with concomitant bacterial peritonitis) were clinically and bacteriologically cured (Table 4). *Serratia marcescens*, resistant to cephalothin, was recovered from patient 7 while he was receiving cephalothin. Urine cultures immediately and 5 months after therapy were sterile in this patient; at 6 weeks after therapy, urine cultures were sterile in the other two.

**Peritonitis.** Two patients with chronic liver disease and ascites had bacterial peritonitis (Table 4). Patient 15 was clinically cured and both patients were bacteriologically cured. Patient 14 required a subsequent course of ampicillin to eradicate persistent *Streptococcus faecalis*, resistant to cefoxitin, from the peritoneal fluid.

**Miscellaneous infections.** An infected aortofemoral graft, cervical osteomyelitis with associated epidural abscess, necrotizing pharyngitis, and postoperative cholangitis were diagnosed in one patient each (Table 4).

Two patients were clinically cured. One, patient 4, required removal of an infected vascular graft. The second, patient 34, from whom *Staphylococcus aureus* was cultured and anaerobes were clinically implicated by Gram stain and odor, improved markedly on therapy.

In contrast, patient 36 had *Enterobacter aerogenes*, resistant to cefoxitin, recovered repeatedly from biliary drainage and manifested clinical signs of infection, necessitating aminoglycoside therapy. The last, patient 21, had *S. aureus* bacteremia and a spinal epidural abscess demonstrated by radiological and neurological findings. When cerebrospinal fluid pleocytosis was noted, therapy was changed to high-dose oxacillin. However, cerebrospinal fluid and blood cultures obtained during cefoxitin therapy yielded no growth. Granulation tissue obtained at surgery after 24 h of oxacillin therapy also was sterile.

TABLE 1. *Outcome of 34 infections in 33 patients*

| Diagnosis | No. of patients | Bacteriological | | | Clinical | |
|---|---|---|---|---|---|---|
| | | Cure | Failure | Unevaluable | Cure | Failure |
| Pneumonia | 12 | 5 | 2 | 5 | 10 | 2 |
| Soft tissue infection | 13 | 6 | 1 | 6 | 11 | 2 |
| Urinary tract infection[a] | 3 | 3 | | | 3 | |
| Peritonitis | 2 | 2 | | | 1 | 1 |
| Miscellaneous | 4 | 3 | | 1 | 2 | 2 |

[a] Includes one patient with simultaneous peritonitis.

TABLE 2. *Pneumonias*

| Patient no. | Age | Underlying disease | Organisms isolated | Days of treatment | Results | |
|---|---|---|---|---|---|---|
| | | | | | Bacteriological | Clinical |
| 2 | 49 | Alcoholism | *Haemophilus influenzae* (S)[a] | 3 | N/A[b] | Died |
| 3 | 24 | Hypogammaglobulinemia | *H. influenzae* (T)[c]<br>*Neisseria catarrhalis* (T)<br>*Veillonella parvula* (T) | 6 | Cure | Cure |
| 12 | 33 | Narcotic abuse | *V. alcalescens* (T)<br>*Neisseria* species (T)<br>Diptheroids (T)<br>α-Hemolytic streptococci (T)<br>*H. parainfluenzae* (T) | 6 | N/A | Cure |
| 16 | 56 | Systemic lupus erythematosis | *Klebsiella pneumoniae* (B)[d]<br>*Staphylococcus aureus* (S) | 9 | Failure | Cure |
| 18 | 69 | Bronchogenic carcinoma | *H. influenzae* (T)<br>*Eikenella corrodens* (T)<br>α-Hemolytic streptococci (T)<br>*Neisseria* species (T)<br>*Fusobacterium nucleatum* (T)<br>*Bacteroides melaninogenicus* (T)<br>*V. parvula* (T) | 4 | N/A | Died |
| 19 | 51 | Alcoholism | *H. influenzae* (T)<br>*Streptococcus pneumoniae* (T) | 8 | Cure | Cure |
| 23 | 41 | Seizure disorder | α-Hemolytic streptococci (T)<br>*Peptostreptococcus micros* (T)<br>*V. parvula* (T) | 14 | N/A | Cure |
| 24 | 66 | COPD[e] | *S. pneumoniae* (S)<br>*Escherichia coli* (S) | 11 | Failure | Cure |
| 27 | 48 | Narcotic abuse; COPD | *S. pneumoniae* (T) (B)<br>*H. influenzae* (T)<br>*B. ruminicola* (T)<br>*B. melaninogenicus* (T)<br>*B. oralis* (T) | 5 | Cure | Cure |
| 28 | 55 | None | *S. pneumoniae* (T) (B)<br>α-Hemolytic streptococci (T)<br>*Corynebacterium* sp. (T)<br>α-Hemolytic streptococci (T)<br>*V. parvula* (T)<br>*Lactobacillus* sp. (T)<br>*B. melaninogenicus* (T)<br>*Eubacterium* sp. (T) | 11 | Cure | Cure |
| 31 | 57 | Bronchogenic carcinoma—radiation treatment | *S. pneumoniae* (T)<br>Group A streptococci (T)<br>*H. influenzae* (T)<br>α-Hemolytic streptococci (T)<br>*Bacillus* species (T) | 11 | Cure | Cure |
| 35 | 53 | Squamous cell carcinoma, mouth—resected | *S. pneumoniae* (T)<br>*N. meningitidis* (T)<br>*H. influenzae* (T) | 13 | N/A | Cure |

[a] S, Sputum.
[b] N/A, Not available.
[c] T, Transtracheal aspirate.
[d] B, Blood.
[e] COPD, Chronic obstructive pulmonary disease.

TABLE 3. *Soft tissue infections*

| Patient no. | Age | Underlying disease | Organisms isolated | Surgery | Days of therapy | Results | |
|---|---|---|---|---|---|---|---|
| | | | | | | Bacteriological | Clinical |
| 6 | 44 | Alcoholism | Group A streptococci | I&D[a] | 5 | Cure | Cure |
| 8 | 53 | DM[b] | *Staphylococcus aureus* (B)[c] | I&D | 31 | Cure | Cure |
| 9 | 59 | Laennec cirrhosis | *S. aureus*<br>Group A streptococci<br>*Peptostreptococcus micros* | I&D | 8 | N/A[d] | Cure |
| 10 | 42 | Narcotic abuse | *Clostridium perfringens* | Needle aspiration | 6 | N/A | Cure |
| 17 | 58 | DM | *S. aureus*<br>*Peptostreptococcus variabilis* | I&D and subsequent amputation | 16 | N/A | Cure |
| 22 | 25 | None | *S. aureus* | I&D | 6 | N/A | Cure |
| 25 | 27 | None | Group A streptococci | Needle aspiration | 10 | Fail | Cure |
| 26 | 58 | DM, peripheral vascular disease | *Pseudomonas aeruginosa*<br>*Serratia marcesens*<br>*Enterobacter cloacae*<br>*Escherichia coli*<br>Enterococci<br>*S. aureus* | I&D | 28 | Cure | Fail |
| 29 | 67 | Laennec cirrhosis | *S. aureus* | I&D | 19 | Cure | Died |
| 30 | 21 | Malabsorption, corticosteroids | *S. aureus* | None | 14 | N/A | Cure |
| 33 | 60 | Trauma | Group A streptococci<br>α-Hemolytic streptococci<br>*E. aerogenes* (B)<br>*S. aureus* | I&D | 14 | Cure | Cure |
| 37 | 32 | Narcotic abuse | *S. aureus*<br>α-Hemolytic streptococci<br>*Peptostreptococcus parvulus* | I&D | 10 | Cure | Cure |
| 38 | 54 | DM | *Propionibacterium acnes* | I&D | 20 | N/A | Cure |

[a] I&D, Incision and drainage.
[b] DM, Diabetes mellitus.
[c] B, Blood.
[d] N/A, Not available.

TABLE 4. *Urinary tract, peritonitis, and miscellaneous infections*

| Patient no. | Age | Location | Organism isolated | Surgery | Days of therapy | Results | |
|---|---|---|---|---|---|---|---|
| | | | | | | Bacteriological | Clinical |
| 1 | 59 | UTI[a] | *Escherichia coli* (B)[b] (U)[c] | No | 5 | Cure | Cure |
| 7 | 47 | UTI | *Serratia marcescens* | No | 8 | Cure | Cure |
| 15 | 60 | UTI | *E. coli* | No | 11 | Cure | Cure |
| 15 | 60 | Peritonitis | *E. coli*<br>*Bacteroides* species<br>α-Hemolytic streptococci | No | 11 | Cure | Cure |
| 14 | 58 | Peritonitis | *Staphylococcus aureus*<br>*Klebsiella pneumoniae*<br>*Streptococcus faecalis* | No | 16 | Cure | Cure |
| 4 | 53 | Infected aortofemoral graft | *S. aureus* | I&D | 28 | Cure | Cure |
| 21 | 51 | Osteomyelitis-epidural abscess | *S. aureus* (B) | I&D[d] | 5 | Cure | Fail |
| 34 | 45 | Necrotizing pharyngitis | *S. aureus*[e] | No | 5 | N/A[f] | Cure |
| 36 | 79 | Cholangitis | *E. coli* (C)<br>*Enterobacter aerogenes* | Biliary drainage | 8 | Cure | Fail |

[a] UTI, Urinary tract infection.
[b] B, Blood.
[c] U, Urine.
[d] I&D, Incision and drainage.
[e] Anaerobes clinically implicated as well.
[f] N/A, Not available.

**Bacteremia.** Bacteremia occurred in seven of the above patients (Tables 2–4), and follow-up blood cultures were negative after 14 days of incubation in all.

**Adverse effects.** Four patients showed deterioration of renal function while receiving cefoxitin. In patient 2, oliguria developed 6 h after therapy had begun. After fluid challenge, urine output resumed and remained adequate despite resumption of cefoxitin. The patient died on hospital day 3; postmortem examination of the kidneys showed minimal cortical scarring, minimal thickening of middle-sized arteries, and hyalinization of arterioles. In patient 11, with ascites and cirrhosis of the liver, renal failure, which was present before therapy, progressed and was a contributory cause of death; postmortem examination showed severe alcoholic liver disease and microscopically unremarkable kidneys. There was a very low urine sodium value and it was believed that the patient had hepatorenal syndrome. Two other patients who had no evidence of antecedent renal disease experienced transient elevations of blood urea nitrogen and serum creatinine. Patient 1, who received cefoxitin for a bacteremic urinary tract infection, also had cirrhosis of the liver with ascites. This patient's creatinine level rose to 3.7 μg/ml and returned to 2.1 μg/ml after discontinuation of the drug. Patient 14 had peritonitis that was also associated with ascites cirrhosis of the liver, and an elevated creatinine level (0.8 to 1.4 μg/ml), which returned to normal after therapy was stopped. No specific cause for the azotemia could be documented in either case.

Eosinophilia greater than 6% of the total leukocyte count was noted in six patients during or after the termination of therapy but, of these, only two, patients 8 and 24, had eosinophil counts in excess of 10%. Two patients had conversion of their direct Coombs tests from negative to trace positive during therapy. This was associated with an average decline in hemoglobin level of 1 g/100 ml. Another patient, who initially had a trace positive Coombs test, was negative on subsequent testing.

**Patient tolerance.** Burning on infusion of cefoxitin, noted by two patients, was not accompanied by phlebitis and did not require discontinuation of the drug. Phlebitis occurred in 12 of the 38 patients. Phlebitis, when it occurred, tended to appear within the first few days of therapy and was persistent, despite changes of infusion site. Degree of involvement was mild in six patients, moderate in four, severe enough to cause discontinuation of therapy in one, and would have resulted in discontinuation in a

second had the course not been completed. All responded within 3 days to local measures. Needle aspiration and culture of one severe case, patient 36, yielded no growth.

**Drug levels.** As shown in Fig. 1, mean serum levels in patients with normal renal function (serum creatinine, ≤1.2 μg/100 ml) after a 1-g infusion ranged from 32 μg/ml 30 min after the end of infusion to <3 μg/ml (lower limit of assay) at 6 h. After a 2-g infusion, levels ranged from a mean of 72 μg/ml at 30 min to 10.4 μg/ml at 4 h (Fig. 2).

Ascitic fluid levels were measured in three patients, 11, 14, and 15, of whom two had abnormal renal function. In all three, ascitic fluid levels of cefoxitin were slightly in excess of simultaneous serum levels 2 h after a 1-g infusion.

Cerebrospinal fluid levels were measured in two patients; one had no meningeal inflammation, the other had mild pleocytosis. At 75 and 180 min after intravenous infusion, respectively, no drug was detected.

**Susceptibility results.** Susceptibility of gram-negative aerobes was one or more twofold dilutions lower with cefoxitin than with cephalothin; cefoxitin was essentially inactive against *Pseudomonas aeruginosa* and *Enterobacter* species. Gram-negative anaerobic organisms were also more susceptible to cefoxitin than to cephalothin, but gram-positive anaerobic and aerobic organisms were less so (Table 5).

## DISCUSSION

Cefoxitin is characterized by a methoxyl group at the 7-position of the cephalosporanic acid nucleus (16). This confers a high degree of resistance to destruction by cephalosporinases (12), notably those produced by indole-positive *Proteus*, certain *S. marcescens* and *B. fragilis* (4, 17), and many cephalosporin-resistant strains of *Escherichia coli*, *Klebsiella*, indole-negative *Proteus*, *Providencia*, *Salmonella*, and *Shigella* (7, 8, 11, 14, 16, 17). It is also active against gram-positive and gram-negative organisms susceptible to currently available cephalosporins (17). Cefoxitin is es-

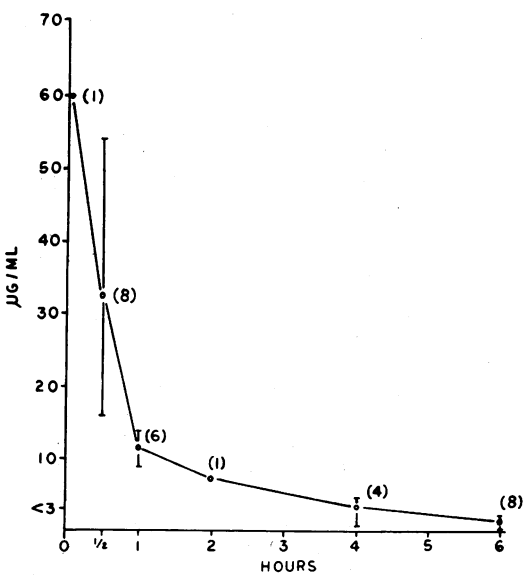


FIG. 1. *Cefoxitin serum levels after a 1-g intravenous dose. Symbols: O, Mean levels; barred lines, range of results; time zero, end of infusion; numbers in parentheses, number of specimens assayed.*

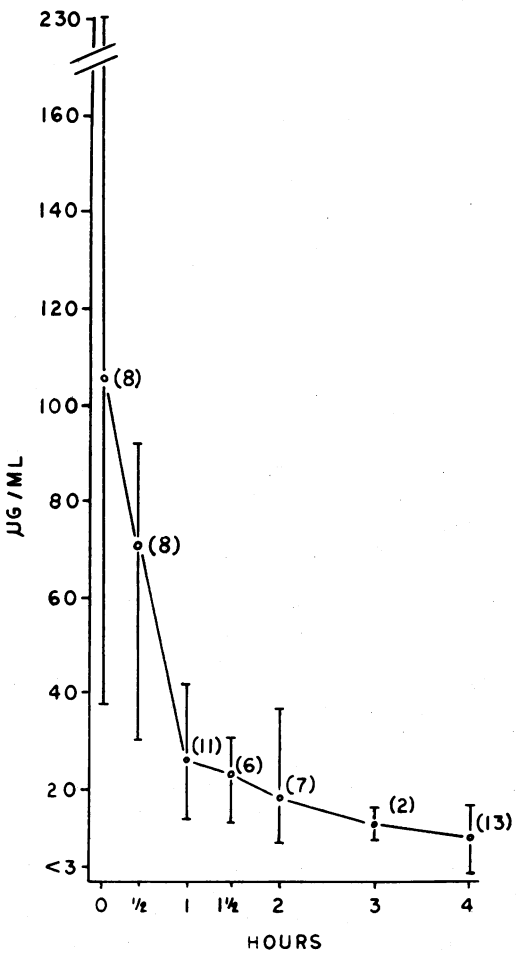


FIG. 2. *Cefoxitin serum levels after a 2-g intravenous dose. Symbols: O, Mean levels; barred lines, range of results; time zero, end of infusion; numbers in parentheses, number of specimens assayed.*

TABLE 5. *Susceptibilities of isolates to cefoxitin and cephalothin*

| Organism | No. of isolates | Minimal inhibitory concentration (μg/ml) | | | |
|---|---|---|---|---|---|
| | | Cefoxitin | | Cephalothin | |
| | | Median | Range | Median | Range |
| *Staphylococcus aureus* | 13 | 2 | (≤1 to 4) | ≤1 | (≤1 to 1) |
| *Streptococcus pneumoniae* | 5 | ≤1 | (≤1 to 2) | ≤1 (All) | |
| Group A | 4 | 2 | (≤1 to 8) | ≤1 (All) | |
| Group D | 5 | >128 (All) | | 64 | (32 to 128) |
| *Escherichia coli* | 4 | 4 | (≤1 to 16) | 32 | (≤1 to 64) |
| *Klebsiella pneumoniae* | 3 | 32 | (16 to 32) | 64 | (32 to 128) |
| *Enterobacter* species | 4 | >128 | | >128 | |
| *Serratia marcescens* | 2 | | (64, 128) | | (128 and >128) |
| *Pseudomonas aeruginosa* | 1 | >128 | | >128 | |
| *Haemophilus influenzae* | 4 | 32 | (8 to 32) | 16 | (8 to 16) |
| *Bacteroides fragilis* | 2 | | (8, 32) | | (64, 64) |
| *Bacteroides* species (non-*fragilis*) | 7 | 1 | (0.25 to 16) | 8 | (≤0.062 to 256) |
| *Peptostreptococcus micros* | 2 | | (0.5, 2) | | (0.125, 0.25) |
| *Peptostreptococcus parvulus* | 1 | 2 | | 0.5 | |
| *Peptococcus variabilis* | 1 | 0.25 | | 0.5 | |
| *Propionibacterium acnes* | 1 | 0.25 | | 0.125 | |
| *Clostridium perfringens* | 1 | 2 | | 2 | |
| *Fusobacterium nucleatum* | 1 | 1 | | ≤0.062 | |
| *Lactobacillus* species | 1 | 1 | | 0.5 | |
| *Veillonella alcalescens* | 1 | 4 | | 4 | |
| *Veillonella parvula* | 4 | 0.5 | (0.5 to 2) | 0.25 | (0.25 to 2) |

sentially inactive against *P. aeruginosa* and enterococci (11, 16).

Susceptibility testing of clinical isolates from our patients supports the finding that cefoxitin has enhanced activity against cephalothin-susceptible gram-negative organisms and good activity against gram-negative anaerobic organisms (4, 8, 11, 13, 14); this occurs, however, at the expense of some decrease in activity against gram-positive organisms (16, 17). It is of concern that serum levels might not always reach the reported minimal inhibitory concentrations for *B. fragilis*. Certain isolates of *B. fragilis* have been inhibited only by 64 μg of cefoxitin per ml (13, 14).

Except for cephaloridine, the cephalosporin compounds rival the penicillins in their freedom from undesirable side reactions and organ toxicities (10, 18). In pharmacological testing, cefoxitin appeared free of nephrotoxic properties in the animal species studied (monkeys, rats, mice, and rabbits) and in healthy human volunteers (2, 7). In the present study, unexplained transient deterioration in renal function of two patients, 1 and 14, was noted. Since, in each case, other factors (hepatic failure, bacteremia) may have contributed to the renal dysfunction, the possible toxicity of cefoxitin must await further study.

In pharmacological trials, the half-life of intravenous cefoxitin was significantly longer than that of cephalothin (2, 7). It appears that levels of cefoxitin in serum in our patients were somewhat prolonged as compared with those reported for cephalothin. Patients receiving the 2-g doses did have appreciable serum levels at 4 h. Hepatic function did not affect serum levels in the absence of associated renal dysfunction.

Cefoxitin caused phlebitis in approximately one-third of our patients; the incidence was unrelated to the dosage regimen. Phlebitis caused by cephalosporins has been reported with approximately this incidence (1, 3). Transient eosinophilia and Coombs positive reactions have also been described with cephalosporins (10). We observed no adverse clinical reactions associated with these findings.

A broad experience with cefoxitin therapy in a variety of infections commonly encountered in clinical practice was obtained in this study. Many of the patients were seriously ill, and seven were bacteremic. Cefoxitin was effective in eradicating infection in the majority of patients and appeared relatively nontoxic. Although dosages and frequency of administration were increased because of our findings of low serum levels after a 1-g dose, no patient had a clinical failure or developed resistant

organisms, even on the lower dosage. Our overall clinical results are comparable to those reported with cephalothin and cefazolin (9).

Further study is required to evaluate the efficacy of cefoxitin in infections. It may prove of specific advantage in the management of intra-abdominal sepsis, where broad-spectrum activity against gram-negative facultative and obligate anaerobes is required.

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