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The American Academy of Pedodontics/Vol. 5, No. 1

SCIENTIFIC articles

# Antibiotic therapy in pediatric dentistry I. Subacute bacterial endocarditis prophylaxis

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Although there are definite indications for the prescription of antibiotics in modern dentistry, a survey of the dental literature reveals wide variations in the prescribing practices of dentists. Even where specific protocols are promulgated by the American Heart Association (AHA) for prophylactic use of antibiotics in prevention of subacute bacterial endocarditis, actual compliance is lower than might be expected. This is the first part of a two-part paper reviewing the literature on the use of antibiotics for the dentist who treats children. In the second part, the use of antibiotics for the treatment of oral infection and in the management of children with systemic problems is discussed. This section will address the prophylactic use of antibiotics in the prevention of subacute bacterial endocarditis.

## Subacute Bacterial Endocarditis

Bacterial endocarditis is an infection of the endocardium, usually located on the valvular surfaces. Acute bacterial endocarditis runs a fulminant course, more often involves previously normal heart valves, and is usually caused by organisms of high pathogenicity.[1, 2] In subacute bacterial endocarditis (SBE) there is a more protracted course, the site of infection is usually in areas of existing cardiac defect, the etiologic agent is usually of low virulence, and there is a greater likelihood of recurrence.[2] For both types of the disease, debilitated patients are more at risk than healthy individuals.

Turbulence of blood flow due to abnormal heart valves causes interstitial changes in the endocardial surfaces. These areas provide an accepting surface for the formation of a platelet-fibrin thrombus in certain susceptible patients.[3] In the presence of a bacteremia the thrombi serve as a nidus for bacterial colonization. The bacteria soon are covered by more fibrin which protects the microorganisms from phagocytosis. The endocardial vegetation may seed bacteria and emboli into the bloodstream.

Prior to the availability of antibiotics, SBE was associated with mortality approaching 100%.[4] During the antibiotic era initial mortality has been reported to be about 25%.[4–6] Even when bacterial endocarditis is cured by early antibiotic intervention, congestive cardiac failure, cerebral infarct, and renal insufficiency may result.[5, 7]

Bacterial endocarditis occurs three times as often in males as in females.[2] Blumenthal has reported approximately the same rate of incidence in children and adolescents as adults (5 admissions for initial disease in 1,000 admissions) and Mostaghim and Millard cite admission records showing 10% of all admissions for initial SBE treatment involved children from birth to 10 years old, and 10% were 10–19 years old.[2, 3] In 82 cases of pediatric infective carditis, Blumenthal reported 50% of the patients had tetralogy of Fallot or ventricular septal defects.[3] Aortic stenosis, pulmonary stenosis, patent ductus arteriosus and aortic coarctation were underlying findings in 25% of these patients. Relatively few children with SBE have rheumatic heart disease.

A transient bacteremia is an important etiologic condition in the pathogenesis of SBE.[1] Although the advent of antibiotics has somewhat decreased its importance as an etiologic agent, *Streptococcus viridans* is still the most common causative organism.[1, 4, 6, 8] (It should be noted that the total incidence of SBE has not decreased since the 1930s, as other organisms have played an increasing role in the disease.) *S. viridans* has been isolated in 37–65% of SBE cases reported in the last two decades.[2, 4, 5, 7]

Investigators have attributed up to 50% of SBE cases to the infection of abnormal heart valves with bacteria in the bloodstream as a result of dental manipulation.[9, 10] There are several reasons for holding this concept.

1. *S. viridans*, the primary pathogen in SBE, is a normal component of the oral flora.
2. The only route for bacteria to reach the endocardium is via the bloodstream.
3. Frequently, bacteremia follows dental manipulation.
4. The onset of endocarditis often occurs shortly after dental manipulations.[3, 4, 6, 11]

Of course, dental treatment and/or dental infection are almost universal experiences, so the causal relationship is difficult to establish. However, as the chain of events outlined is widely accepted, it would be prudent for

dentists to do whatever is possible to reduce the incidence of this serious disease.

## Congenital and Acquired Heart Diseases

Patients with heart valve abnormalities of a congenital or acquired nature are considered to be at risk for SBE. Specifically at risk are patients with congenital cardiac defects such as: aortic stenosis, pulmonary stenosis, ventricular septal defect, patent ductus arteriosus, coarctation of the aorta, and tetralogy of Fallot.[1, 3, 7, 8, 10, 12, 13] Patients with rheumatic heart disease have long been recognized to have a high susceptibility to SBE, as have patients with a previous history of bacterial endocarditis, even in the absence of clinically detectable heart disease.[14] Others who have had prosthetic heart valve replacements are most prone to SBE.[1, 11, 13, 15] Some authors believe that no antibiotic coverage is required six months after a surgical repair of patent ductus arteriosus or atrial septal defect has been performed.[13] Patients with internal cardiac pacemakers or ventriculoatrial shunts for hydrocephalus may also be at risk for SBE, as may those having arteriovenous fistulae or shunts for renal dialysis.[11, 14, 16] (Also of concern is the ease with which the shunt sites of these patients may become infected in the presence of any bacteremia.)

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Although SBE-susceptible patients with diagnosed cardiac defects or disease may give a history of heart "murmurs", not all "murmurs" necessarily indicate the need for antibiotic prophylaxis prior to dental treatment.[6] In cases of incomplete medical histories, it is best to consult the patient's physician regarding the need for SBE prophylaxis.

## Rheumatic Fever

Rheumatic fever, a systemic inflammatory disease occurring after infection with Group A beta-hemolytic streptococci, is the chief cause of heart disease in patients under 40 years of age.[1, 17] Penicillin often is prescribed for pediatric patients with pharyngeal and other Group A beta-hemolytic streptococcal infections to prevent the possibility of this serious sequela.[18] With this preventive antibiotic therapy the incidence of rheumatic fever is decreasing.[3] About 33% of rheumatic fever patients have residual heart damage after the initial episode of the disease, and approximately 66% of 10-year survivors have detectable valvular disease.[17] These patients are said to suffer from rheumatic heart disease. Common heart lesions of rheumatic heart disease are mitral stenosis and calcific aortic stenosis.[7]

Patients wtih rheumatic heart disease are known to be at risk for SBE. Some authors have advocated giving antibiotic prophylaxis against SBE to any patient with a history of rheumatic fever.[19] Others have pointed out that only patients with rheumatic heart disease are at increased risk when exposed to bacteremias.[15, 17] Some patients with a history of rheumatic fever are on long-term antibiotic prophylaxis against Group A beta-hemolytic streptococci to avoid rheumatic fever recurrence. This prophylaxis is not sufficient to protect the rheumatic heart disease patient from SBE.[20, 21] Appropriate prophylaxis against SBE for these patients will be discussed later in this paper.

## Bacteremia Following Dental Manipulation

Okell and Elliot first reported the existence of transient bacteremias after extraction of human teeth in 1935.[22] Since that time other investigators have reported incidence of bacteremia after dental extractions ranging from 39 to 100%.[23–28] Although Speck et al. and Faigel and Goskill reported no incidence of bacteremia after extraction of healthy teeth in children with no gingival or periodontal disease, these findings have been challenged because of possible technical problems in the blood sampling protocols used.[26, 29] Berry et al. and Elliot and Dunbar have reported a significant incidence of bacteremia in children after extraction of healthy and infected teeth.[25, 30] Robinson et al. found that the incidence of postextraction bacteremia was not related to the patients' age or sex, the pulpal status of the extracted teeth, or the gingival condition.[27]

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Some authors have related incidence of bacteremia to the type of anesthesia utilized during extractions, finding the most positive blood cultures with general anesthesia, an intermediate number with block anesthesia, and the lowest incidence with local infiltration.[24, 31, 32] Compounding variables in these studies are the possibility of increased trauma during procedures requiring general anesthesia, and the effect of the vasoconstrictor in local anesthetic solutions infiltrated into the area of extraction (port-of-entry of the organisms). A positive correlation between the amount of local trauma involved in an extraction procedure and the incidence of bacteremia has been shown.[33, 34] Patients having multiple extractions suffer more bacteremias than those having single extractions.[33, 34] It may be that the actual occurrence of bacteremia approaches 100%, and that results showing lower

rates of incidence are due to the small numbers of bacteria entering the blood stream and the relatively small blood samples drawn.

The early literature regarding bacteremias of dental origin focused on dental extractions. However, bacteremias also have been found after brushing teeth, using an oral irrigation device, chewing wax, and luxating or rocking teeth in their sockets.[27, 35-38] More recent research indicates that the degree of gingival trauma is probably the determining factor.[8, 9, 25, 34, 36, 39] DeLeo et al. found 28% of 7- to 12-year-olds had positive blood cultures 5 minutes after oral prophylaxis.[37] Bacteremial incidence has been reported following peridontal treatments in adults with varying degrees of peridontal disease.[27, 34]

Baumgartner et al. have demonstrated an incidence of bacteremia of 3.3% in cases of aseptically performed nonsurgical endodontic therapy.[28] Farrington sampled the venous blood of 25 patients after they had received formocresol pulpotomies. Only one positive culture was reported, probably due to the fact that in the pulpotomy procedure the *periapical* capillary blood is not directly exposed to oral flora.[40]

The predominant microorganisms of the oral cavity are *S. viridans*, a group of alpha-hemolytic (greening) streptococci. These oganisms have been isolated from sputum, root canals, periodontal pockets, calculus, carious lesions, periapical tissues, and the apices of extracted teeth.[31, 41-43] These same bacteria are the organisms most commonly reported in the bacteremias following dental manipulations.[25, 32, 37, 44] It should be noted, however, that many earlier studies did not utilize sampling and culture techniques that are now used to ensure proper analysis for anaerobic organisms.

*. . . any procedure interrupting the integrity of the gingival or pulpal tissue is capable of promoting bacteremia.*

In the pathogenesis of SBE the "danger period" extends from the beginning of the dental procedure until such time as there is no longer a significant number of bacteria in the blood or on the heart valves. Most bacteremias after dental procedures are short (5–30 minutes) in duration.[3, 9, 22, 34] The number of organisms in the blood depends on the facility for invasion of the bloodstream during and immediately after the procedure, bacterial multiplication in the circulatory system, and the activity of the body's clearing mechanism.

Antibiotics are given not to prevent the development of a bacteremia, but in the belief that they will reduce the bacteremia and will help to destroy organisms lodging on the endocardium.[3, 5] Cooley and Haberman found 39.21% of control patients had postextraction bacteremia.[24] In patients given antibiotics prior to extractions the incidence of positive blood cultures was 14.28%. Elliot and Dunbar found 36.3% of children receiving three doses of oral penicillin before extractions had postextraction bacteremias.[25] Of patients receiving benzyl penicillin 30–60 minutes prior to the treatment, only 7.8% had bacteremias.

In reviewing the evidence, it is apparent that any procedure interrupting the integrity of the gingival or pulpal tissue is capable of promoting bacteremia. Any professional dental care which is likely to cause oral bleeding must be suspected of putting the SBE-susceptible patient at risk for the disease. Although there are no reported cases of SBE related to use of gingivally impinging rubber dam clamps, the seating of orthodontic bands, or crown and bridge procedures, logic points to the conclusion that these procedures may, indeed, cause the bacteremia necessary for SBE to occur. As Beeley stated, "Periodontal tissues, whether healthy, or diseased are the most important source of infection in bacterial endocarditis due to *S. viridans*."[45]

## Antibiotic Prophylaxis Against SBE

SBE is such a destructive disease that every attempt should be made to prevent its occurrence. It is well known that patients with specific congenital and acquired heart diseases are most likely to contract this disease. It also is known that the most common causative organisms of SBE is allowed entrance to the systemic circulation during many dental procedures. From this understanding, the concept of antibiotic prophylaxis against SBE for patients at risk undergoing dental procedures has developed.

Attempts at sterilizing the oral cavity with topical agents and with systemic antibiotics have not been successful.[5, 7, 9, 15] Realizing that some microorganisms will, therefore, invade the bloodstream, the goal of SBE prophylaxis must be twofold:

1. to destroy the organisms in the bloodstream and prevent them from multiplying
2. to destroy any organisms which have settled on the endocardial surface before they are protected from phagocytosis by a fibrin layer.[7]

Applying the principles of antibiotic therapy, the prophylaxis should be directed at the expected microorganism: in this case, alpha-hemolytic streptococci. The antimicrobial should have as narrow and specific a spectrum of sensitivity as possible.[46] The drug should be administered before the bacterial invasion occurs to provide adequate blood levels at the time of tissue insult, and should be given in relatively low doses for a short time, usually 24–48 hours.[14, 46] A short course helps to prevent the development of resistant strains, simplifying treatment if infection develops.[11]

Antibiotic prophylaxis against SBE must begin prior to the dental procedure, so that at the time of manipulation and bacteremia an adequate blood level of the antimicrobial agent is present. Although in the earlier literature there were many recommendations for prophylaxis to begin several days prior to the procedure, this is not in keeping with the present goals of SBE prophylaxis.[3, 14, 15] The earlier recommendations were based on an attempt to sterilize the oral tissues, and this has proven unsuccessful.[7] Rather than providing a beneficial effect, the use of antibiotics preoperatively may be detrimental. Penicillin and other drugs which act against *S. viridans* enhance the development of antibiotic-resistant strains of the microorganisms.[25, 47–49] Within a few hours of the onset of penicillin therapy, it has been documented that the *S. viridans* count of saliva may drop from $10^7$/ml to $10^3$/ml or less.[49] However, within 24–72 hours, penicillin-resistant strains begin to multiply, and they can reach a count as high as $10^6$/ml. Two or three days after the antibiotic is discontinued, the antibiotic-sensitive streptococci begin to return. In one survey, the flora was back to its normal constituency after four weeks, but other authors reported the presence of some penicillin-resistant colonies 42 days after penicillin administration ceased.[48, 49] Stirland and Schotts point out that patients treated in hospital wards acquired more resistant bacteria at a faster rate than patients treated at home.[49]

Penicillin is the antibiotic of choice for prophylaxis against *S. viridans* bacterial endocarditis. In patients allergic to penicillin, erythromycin is advocated. The American Heart Association (AHA) released new recommendations for SBE prophylaxis regimens in 1977.[14] The recommended regimens appear in Table 1. It should

**Table 1.** Prophylaxis for Dental Procedures and Surgical Procedures of the Upper Respiratory Tract

| REGIMEN A OR B | REGIMEN B |
|---|---|
| 1. all dental procedures that are likely to result in gingival bleeding[d] | prosthetic heart valves[a] |
| 2. most congenital heart disease[b] | |
| 3. rheumatic or other acquired valvular heart disease | |
| 4. idiopathic hypertrophic subaortic stenosis | |
| 5. mitral valve prolapse syndrome with mitral insufficiency[c] | |

[a] This does not include shedding of deciduous teeth or simple adjustment of orthodontic appliances.

[b] This includes: ventricular septal defect, tetralogy of Fallot, aortic stenosis, pulmonic stenosis, complex cyanotic heart disease, patent *ductus arteriosus, or systemic-to-pulmonary artery shunts.*

[c] Although cases of infective endocarditis in patients with mitral valve prolapse syndrome have been documented, the incidence appears to be relatively low and the necessity for prophylaxis in all of these patients has not yet been established.

[d] Some patients with a prosthetic heart valve in whom a high level of oral health is being maintained may be offered oral antibiotic prophylaxis for routine dental procedures except the following: parenteral antibiotics are recommended for patients with prosthetic valves who require extensive dental procedures, especially extractions; or oral or gingival surgical procedures.

REGIMEN A—PENICILLIN

**1. Parenteral-oral combined:**

Adults: aqueous crystalline penicillin G (1,000,000 units intramuscularly) *mixed with* procaine penicillin G (600,000 units intramuscularly). Give 30 minutes to one hour prior to procedure and then give penicillin V (formerly called phenoxymethly penicillin) 500 mg orally every six hours for eight doses.+

Children:* aqueous crystalline penicillin G (30,000 units/kg intramuscularly) *mixed with* procaine penicillin G (600,000 units intramuscularly). Timing of doses for children is the same as for adults. For children less than 60 lbs the dose of penicillin V is 250 mg every six hours for eight doses.+

**2. Oral:‡**

Adults: penicillin V (2.0 gm orally 30 minutes to one hour prior to the procedure and then 500 mg orally every six hours for eight doses.)+

Children:* penicillin V (2.0 gm orally 30 minutes to one hour prior to procedure and then 500 mg orally every six hours for eight doses. For children less than 60 lbs use 1.0 gm orally 30 minutes to one hour prior to the procedure and then 250 mg orally every 6 hours for eight doses.)+

*For Patients Allergic to Penicillin:*

Use either Vancomycin (see Regimen B), *Or* use:

Adults: erythromycin (1.0 gm orally 1½–2 hours prior to the procedure and then 500 mg orally every 6 hours for 8 doses.)+

Children:* erythromycin, (20 mg/kg orally 1½–2 hours prior to the procedure and then 10 mg/kg every six hours for eight doses.)+

REGIMEN B—PENICILLIN PLUS STREPTOMYCIN

Adults: aqueous crystalline penicillin G (1,000,000 units intramuscularly) *mixed with* procaine penicillin G (600,000 units intramuscularly), *plus* streptomycin (1 gm intramuscularly). Give 30 minutes to one hour prior to the procedure; then penicillin V 500 mg orally every six hours for eight doses.+

Children:* aqueous crystalline penicillin G (30,000 units/kg intramuscularly) *mixed with* procaine penicillin G (600,000 units intramuscularly), *plus* streptomycin (20 mg/kg intramuscularly) timing of doses for children is the same as for adults. For children less than 60 lbs the recommended oral dose of penicillin V is 250 mg every 6 hours for eight doses.+

*For Patients Allergic to Penicillin:*

Adults: vancomycin (1 gm intravenously over 30 minutes to one hour). Start initial vancomycin infusion one half to one hour prior to procedure; then erythromycin 500 mg orally every six hours for eight doses.

Children:* vancomycin (20 mg/kg intravenously over 30 minutes to one hour).** Timing of doses for children is the same as for adults. Erythromycin dose is 10 mg/kg every six hours for eight doses.

FOOTNOTES TO REGIMENS: +In unusual circumstances or in the case of delayed healing, it may be prudent to provide additional doses of antibiotics even though available data suggest that bacteremia rarely persists longer than 15 minutes after the procedure. The physician or dentist also may choose to use the parenteral route of administration for all of the doses in selected situations.

be noted that parenteral administration of antibiotics is preferable, especially for high-risk patients, because it allows more predictable blood levels.

Patients already on antibiotic therapy at the time of dental treatment pose a difficult problem. Certainly patients with a history of rheumatic fever on low-dose penicillin prophylaxis against Group A streptococcal infections are not adequately protected against SBE.[14, 34, 50] Several strategies are available for these patients. If the patient's physician is willing, the penicillin regimen may be dropped for a period of 2–3 weeks to allow normalization of oral flora.[51] Then the dental treatment can be accomplished under the regular AHA Regimen A, and after 48 hours, the patient can return to his low-dose routine. Alternatively, the penicillin dose can be increased and/or a second antibiotic added.[5, 7, 14, 15, 50] Merely increasing the penicillin dosage is not adequate because penicillin-resistant strains are almost certainly present. Moreover, if SBE did occur under these conditions it would be much harder to treat subsequent to dental treatment.[49] Cephalosporins, vancomycin, streptomycin, lincomycin, and erythromycin have been suggested for supplementation of the penicillin in these cases.[5, 7, 14, 15, 50] The AHA suggests the use of oral erythromycin as in Regimen A, or one of the Regimen B choices.[14] Stimmel et al. reported that 95.5% of the penicillin-resistant *S. viridans* cultured from saliva of children receiving long-term penicillin (because of congenital or acquired heart disease) were sensitive to erythromycin.[50]

*Even when all precautions are taken, SBE may still develop . . .*

Patients with multiple dental needs may be handled in two ways. If possible, a longer than average appointment can be made, completing all necessary treatment in one visit. If multiple visits are necessary, they must be spaced to allow for a return of the normal flora (which has been depressed by the antibiotic prophylaxis), and the disappearance of antibiotic-resistant strains. Without a period of 10–14 days between dental procedures, the patient may be at risk for a treatment-resistant SBE caused by a strain of bacteria resistant to the antibiotics used for prophylaxis.[5] Kennedy has suggested intervals of 4–6 weeks between appointments when antibiotic coverage is required, in order to prevent sensitization of the child to the antibiotic being given.[52] But, as Tizard has stated, sensitivity to penicillin is unusual in children.[53]

In the presence of infection, antibiotic coverage of patients susceptible to SBE must be continued after the eight doses. Acute infections may cause a bacteremia or septicemia every bit as dangerous to the patient. Therefore, antibiotic therapy appropriate to the infection should be maintained until after all signs of infection have subsided.

Considering the volume of literature concerning methods to protect patients against a disease as deadly as SBE, it is difficult to understand any clinician's failure to provide antibiotic coverage for patients at risk. Unfortunately, it appears that the majority of dentists may not be complying with the AHA recommendations. In 1968 Tarsitano and O'Hara wrote that "most dentists are familiar with recommended drugs and methods of prevention that are advocated by the AHA."[17]

However, just one year earlier Elliot had reported that a survey of practitioners revealed patients with congenital and acquired heart disease were receiving dental treatment of all types without antibiotic coverage.[47] McGowan and Tuohy questioned a number of patients they considered at risk for SBE about their dental experiences since they had been informed of their cardiac disease.[19] Only 36% recalled having been questioned by their dentists about the presence of heart disease, and only 19% remembered being told by their physicians that they would need special medication for dental procedures. These patients had undergone 424 dental procedures, including scaling, endodontics, and extractions since their cardiac diagnosis, and only on 23% of these occasions were the patients covered with antibiotics. These statistics would seem to underscore our emphasis on the absolute necessity for taking an accurate medical history for each dental patient. Moreover, Brooks reported in 1980 that although most dentists in her study questioned patients about a history of rheumatic fever and heart murmur, and although most of these dentists reported they believed that the 1977 AHA guidelines for prophylaxis were realistic, only 14.5% of them followed the guidelines.[54]

*. . . the most common causative organism of SBE is allowed entrance to the systemic circulation during many dental procedures.*

The clinician must not only provide the necessary treatment, but also must teach the patient why these steps are necessary. Bear suggests that the patient be asked to report to his physician regarding any fever which may develop within one month after the dental procedure.[55] Even when all precautions are taken, SBE may still develop, so it is best to be suspicious.[14]

Unfortunately, there is no hard evidence supporting the SBE prophylaxis recommendations, and there have been no human studies comparing the efficacy of different methods of preventing the disease.[11, 56, 57] There is

evidence showing a reduction of bacteremia with preoperative antibiotic treatment.[24, 25] There have been cases of SBE which were attributed to dental manipulation while the patient was receiving antibiotic coverage; however, most of these were cases in which the patient was on long-term penicillin or erythromycin therapy preceding the extractions.[4, 21]

Although statistical proof of the validity of SBE prevention is not available, and the suggested antibiotic regimens are arbitrary, it is doubtful that evidence comparing incidence of SBE in those patients with SBE prophylaxis and those without ever will be presented, as most authors agree that the risks of not offering prophylaxis far outweigh the risks of the antibiotic therapy.[3, 14, 56, 58] In the absence of evidence that SBE prophylaxis is not effective, every attempt should be made to protect patients at risk with appropriate antibiotics.

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# Quotable Quotes

Travel expands the mind and loosens the bowels. The naive voyager is initiated to many unexpected pleasures and experiences, but a not infrequent accompaniment to such adventures is a bout of debilitating diarrhea. Annually, more than 250 million people travel from one country to another, and tourism has become economically important, especially in developing countries. United States citizens traveling to Mexico number three million per year alone. With an attack rate of 25-50%, diarrheal illness may affect more than a million of these visitors yearly; 30% will be ill enough to be confined to bed, and another 40% will have to alter their scheduled activities . . .

Some intrepid travelers take a "grin-and-bear-it" attitude, accepting the risk of diarrhea and allowing it to take its natural course (an average of 93 hours), which at the pace of modern travel means missing several museums, monuments, and exotic nightspots. . . Alternatives (measures) are highly reasonable. For mild diarrhea, the traveler can use bismuth subsalicylate, and I would add as an option the opiate drugs. For more severe illness, the trimethoprim/sulfamethoxazole regimen of one pill twice a day seems appropriate. Current knowledge suggests that if you can't completely stop the runs, you can at least cut your losses.

From: Gorback, Sherwood L.
Traveler's Diarrhea (Editorial).
N Engl J Med 307: 881-83, 30
September 1982.