MODERN MANAGEMENT OF DENTAL CARIES:

THE CUTTING EDGE IS NOT THE DENTAL BUR

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ABSTRACT

Treating the disease, not the symptoms, is the change in managing dental caries. As researchers supply the tools, dentists can apply more efficient and realistic methods for better patient care.

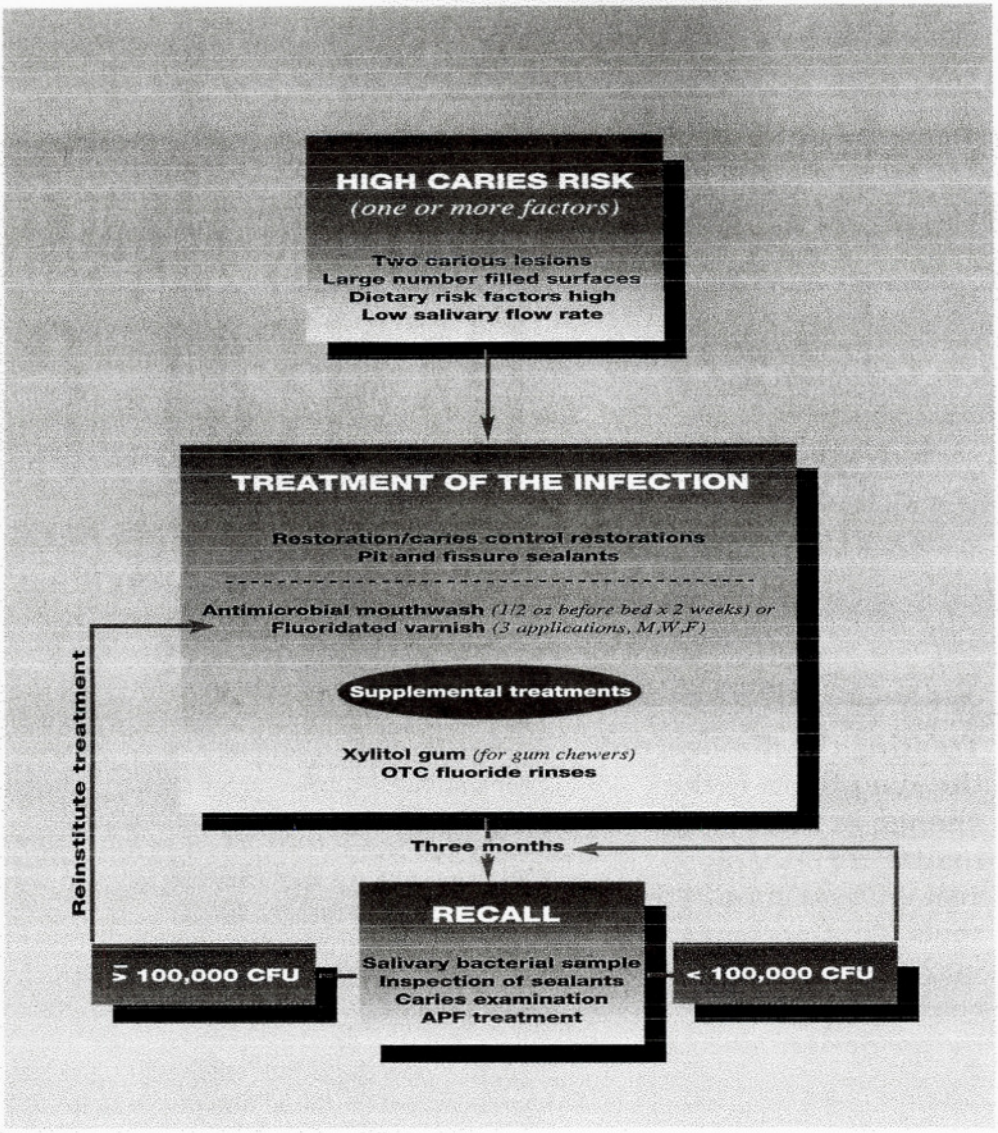
The practice of dentistry is constantly changing. New materials and techniques continuously replace older ones. Today we can place beautiful esthetic restorations, where just 30 years ago we were limited to less attractive restorations. Intraoral video cameras and computer-based voice-recognition promise to change the nature of the dental record. Digitized radiographs received on a computer-linked detector, with far greater control of the image than conventional radiographs, are nearing reality. These and many other changes are part of our ever-advancing profession.

A more subtle but far more substantive change is also occurring. We are changing the way we approach the management of the two primary diseases of the mouth—caries and periodontal diseases. Dental researchers are providing the tools to treat these diseases as infections, rather than just treating the disease symptoms. This subtle change is moving dentistry from the traditional "surgical model" of care, into a more modern "medical model" of care. In this paper, we review the changes as they apply to dental caries.

DENTAL CARIES HISTORY: CONVENTIONAL MODEL

Dental caries has been part of the human condition since humans evolved.[1] Yet caries has been viewed as a bacterially mediated disease for only the past 103 years.[2] Dr. Willoughby Dayton Miller codified the chemoparasitic theory of tooth decay in 1890. Until his investigations, few researchers had examined the oral bacterial flora. Dr. Miller applied then state-of-the-art techniques to his research. But because of the limitations of 1890s technology, he was not able to isolate the specific pathogen(s) of caries within the bacterial plaque and was forced to treat the plaque mass as odontopathic. The derivatives of that forced assumption shaped our conventional treatment model. The treatment assumptions we have made under the premise that plaque is odontopathic are:

- No diagnosis is required, since everybody has plaque.
- The entire population must be treated, since everyone forms plaque.
- The goal of treatment is removal of plaque, and treatment must be continuous, since plaque forms continuously. (We implement this by encouraging our patients to brush and floss more often.)
- Patients are recalled for examination and for any new restorations required.
- Failure is the patient's fault, since a carious lesion is prima facie evidence that the patient did not keep the plaque off the tooth.[3]



Managing patients with caries.

The result of this treatment philosophy is our traditional or surgical model of care in dentistry. In this model, dentists are relegated to the role of artisans/technicians. We surgically excise diseased tooth structure and obturate the area with an inert filling material, but never fully address the cause of the disease. That is, the dentist-surgeon treats the clinical signs of the caries infection but, other than excising the carious lesion, doesn't attempt to eliminate the infection that caused the carious lesion. Unfortunately, excising the carious lesion doesn't remove all of the infecting organisms.

The effect of applying the same surgical model of care to the specific bacterial infection resulting in tuberculosis would be dramatic. The physician would diagnose the disease through a skin test, sputum sample and radiographs. After diagnosis, a surgeon would excise the diseased portion of the lung and fill that area with a restorative material. After recovery from surgery, the patient would be dismissed from care and recalled every six months until new lung lesions were diagnosed. When diagnosed, the surgeon would operate again. Of course this would be deemed medical malpractice, because the physician treated only the clinical signs of the disease, and never addressed the bacterial cause of the disease.

In this hypothetical case, the medical team used a strict surgical model of care in a disease in which a medical model of care would have been a more appropriate choice. A medical model of care would address both the lung lesion (the clinical manifestation of the infection) and the cause of the disease process (the tubercule bacillus).

The same medical paradigm

can be used to diagnose and treat dental caries. Dental research has identified the major causative organisms of dental caries in the human. These are the very organisms Dr. Miller sought more than a century ago but was unable to identify because of the limitations of the available technology.

SPECIFIC PLAQUE HYPOTHESIS

During the past 25 years, a philosophy of dental care delivery based on the ability to identify the causative organisms of dental caries has emerged. It was named the Specific Plaque Hypothesis by its principal author, Dr. Walter Loesche.[4] The hypothesis states that only a limited number of the organisms in dental plaque cause the disease process. Plaque per se is not odontopathic. Rather, a finite and identifiable number of organisms within the plaque of some patients are responsible for caries.

Caries is viewed as an infection rather than a lesion. The consequences of this paradigm shift give an entirely different, in fact dichotomous, set of assumptions from those derived from Miller's original model. These assumptions are:

- Diagnosis is essential because only those patients at risk for the clinical manifestations of this infection are treated.
- Treatment is directed at reduction or elimination of the odontopathogens.
- Treatment ceases at a therapeutic endpoint, and patients are recalled to diagnose any reinfection.
- Failure is the dentist's fault, since it is a failure to diagnose the infection.[3]

This is a medical, rather than a surgical model of dental care. The dental team is concerned with the timely restoration of diseased areas and the elimination of the infection that leads to the carious lesions. In this modern model of care, the dental team works together to control dental caries. Caries is diagnosed, the patient is treated for the infection and recalled regularly to ensure the infection has not been reacquired.

Since the medical model assumes that there are a finite number of caries-causing organisms, knowledge of the odontopathogens is essential. We need to know which of the 300-plus oral organisms lead to the clinical signs of dental caries.

Studies reveal that two of the primary organisms involved in human caries are *mutans streptococci* and *Lactobacilli* species.[4-10] Clarke first identified *Streptococcus mutans* in caries in 1924.[11] Recently, *S. mutans* has been recognized as more than one bacterial species. This group of bacteria has been functionally labeled *mutans streptococci (ms)*.

Some 36 years after Clarke's findings, Fitzgerald and Keyes found that these organisms were capable of causing caries in hamsters.[12] At about the same time, the U.S. Navy examined its recruit population for levels of *ms* and the presence or absence of caries.[13] They found that, generally, those recruits with low or nonexistent *ms* levels had no caries. Those with high *ms* titers in their saliva had high caries rates.

Several researchers showed that *ms* were not found in predentulous newborns.[7,14,15] Yet by age 5, more than half of the children were infected with *ms*.[7,16] Berkowitz and Jordan used serotyping to show that children who became infected had the same organism as their mothers.[7,16,17] Van Houte and Green found that a critical level of *ms* was necessary to establish an intraoral infection.[18] Kohler and Bratthall then showed that it was possible to pass sufficient *ms* via a spoon to transmit the infection from parent to child. They also found that caries-free children generally had <10,000 colony-forming units of *ms*/milliliter of saliva.[19]

Caufield and his associates showed that *ms* appear to be transmitted to the child at specific times.[16] Seventy-five percent of the *ms*-infected children acquired the infection between 19 and 28 months of age (median age, 24 months). Eighty-three percent were infected by age 4 years. This study also showed that only those infants with the *ms* infection had carious lesions. The non-infected group did not develop carious lesions.

These studies show that in humans, dental caries is the clinical symptom of a bacterial infection, mediated primarily by *ms*, which are transmitted by salivary exchange within the family unit.

Lactobacilli have also been associated with dental caries.[20] These organisms are sometimes found in elevated proportions in patients with caries. Basing treatment on the identification of this organism in the saliva has not generally been successful as *Lactobacilli* appear to be a secondary caries organism.

Lactobacilli do not have the tenacious attachment mechanisms of *ms* to effectively initiate the carious process. However, once the carious process is established, the environment becomes acidic and conducive to growth for *Lactobacilli.* Tests for *Lactobacilli* may show that there is caries activity present and that dietary controls are not in place, but *Lactobacilli* generally do not predict the carious process.[3]

In a medical model of care, initial diagnosis should be performed by standard clinical examination. Patients with active caries have a caries-causing infection. It makes little sense to test them bacteriologically while carious lesions are unrestored. Tests at this time often provide mixed results, since *Lactobacilli* or other acidic organisms may actually overgrow *ms* in carious lesions. In addition, initial treatments are not predicated on the level of infection. However, as we see later, the magnitude of a recurrent infection dictates treatment at recall appointments. Patients without caries and with no recent history of caries are generally not candidates for this caries control program.

TREATMENT

The treatment plan is designed first to remove the nidi of infection from the oral ecosystem and then attack the now vulnerable *ms* infection (Figure).

Sound medical and dental histories should be obtained at the initial examination. These should include a dietary screening to rule out simple dietary patterns that are affecting caries. Medications or other causes of limited salivary flow should be identified. Each of these has an important part in the caries process.

Caries-active or caries-susceptible patients should be treated for their infection. A rationale for treatment in a medical model of care would encompass those elements required to treat a bacterial infection. A review of medical protocols shows most bacterial infections are treated on a short-term basis, intensively, and to a therapeutic endpoint.[3] An example is a bacterial pneumonia where the infection is treated with large doses of an appropriate antibiotic (intensively) to disrupt the pathogen and allow the body's defenses to take over in a short time. When the causative organism has been defeated, the treatment is discontinued (the therapeutic endpoint).

The treatment regimen for *ms* infections uses the same model. In the case of caries, we should not use antibiotics that might be needed to treat a life-threatening infection in the future. However, we can use antimicrobials and sound bacteriologic principles to control the *ms* infection.

RESTORATION

The first step. In treating the infection, restore the existing carious lesions with frank cavitation. Failure to remove these nidi of infection could lead to an actual superinfection with *ms*.[3] The rationale for this argument is that treatment with antimicrobials before restoration would disrupt the normal surface flora. The bacteria in protected areas, such as carious lesions or infected pits and fissures, would face no competition for the disinfected tooth surface. The organisms within these protected environments would be free to grow out of the cavity and populate the tooth.

The type of restoration placed is important. When a patient has a large number of carious lesions, place caries-control temporary restorations until *ms* have been eliminated.[9] This could prevent seeding of the margins of new restorations with *ms* and may reduce the potential for recurrent caries. When only a small number of lesions exist, definitive restorations may be performed. Materials selection becomes increasingly important, since some restoratives have good antibacterial properties, while others have little or no effect on the bacterial flora.[21]

Minimal carious lesions such as white-spot lesions and radiographically incipient lesions or other lesions where the outer surface does not display cavitation should not be surgically repaired. It is possible, with the proper therapy, to remineralize these lesions.

SEALANTS

The second step. Simultaneously with the restorative process in step one, apply a fluoride-releasing pit and fissure sealant like Fluoroshield (Caulk). In the permanent dentition, these should be applied to the molars and premolars. The intent of sealant application is to sequester this ecosystem from the remainder of the mouth. This prevents seeding the mouth from already infected pits and fissures, and prevents reinfection of these pits and fissures if *ms* are

reintroduced after being suppressed or eradicated. In the primary and mixed dentition, the primary molars should be sealed. Sealants are extremely effective in the long-term reduction of pit and fissure caries.[22]

Sealants may be effectively applied to questionable and early carious pit and fissure sites.[23-26] Odontopathic organisms trapped below a sealant will decrease in number and remain quiescent while they're under the sealant.[27] Fortunately, dentinal fluids don't supply enough nutrients for the carious organisms within the dentin.

Fractured, non-carious margins of amalgam restorations should also be sealed with pit and fissure sealants. Ditched amalgam margins are the most common problem of amalgam restorations.[28] It is estimated that between 9 and 39 million amalgams are replaced annually in the United States because of faulty margins.[29] Yet ditched amalgam margins alone do not predict restoration failure nor present or future caries.[30-32] The sealant is most effective when the enamel is etched according to the usual sealant procedures and the amalgam surface is microabraded with an intraoral abrader using 50 microns aluminum oxide (Danville Engineering).

Microabrasion of the amalgam surface decreases the surface tension between the sealant and the amalgam and allows better wetting of the surface.[33] Applying pit and fissure sealants completes the initial treatment phase. The restorations and sealants have removed *ms* favored and protected ecologic niches.[34] Now we can deal with the surfaces where the organism is more vulnerable.

The third step. Make the initial attack on the now vulnerable *ms* infection. Apply antimicrobials intensively, on a short-term basis, and to a therapeutic endpoint.

The primary treatment consists of the short-term use of the antimicrobial, chlorhexidine, which is highly effective against *ms* infections.[35,36] To maintain an intensive, short-term treatment to a therapeutic endpoint, one 16-ounce bottle of CHX rinse should be prescribed for the patient. The patient uses a half-ounce, 30-second rinse just before bed. Salivary flow diminishes to nearly zero overnight, and the concentration of the drug in the mouth remains high until morning. This increases the amount of time the drug remains in contact with *ms* and prolongs its effectiveness.

The effectiveness of CHX lies in its chemical charge. Chlorhexidine is a biguanide and strongly cationic. Since almost all oral surfaces are negatively charged, the positive charge of this cationic drug causes it to adhere to almost everything, giving the drug substantivity.[37,38] (Substantivity is the ability to keep an agent in contact with an organism long enough to kill or disable the organism.) The drug maintains bactericidal activity for the duration of sleep. A 14-day regimen will suppress the *ms* infection below the lower sensitivity limit of most caries tests (D.P. Cote and M.H. Anderson, 1990, Graduate Research Project, Naval Dental School, Bethesda, Md., unpublished data). If this is the only treatment, *ms* suppression will last between 12 and 26 weeks.[39]

These CHX and *ms* data are derived primarily from studies in Europe where CHX is currently approved for wider use than in the United States. In the United States, CHX is approved for the treatment of gingivitis. For the past 25 years in Europe, CHX has established an excellent record of efficacy and safety in treating caries infections. The teratogenic and carcinogenic safety has been adequately demonstrated in animals and human populations.[40]

Because CHX is poorly absorbed from the gastrointestinal tract, the lethal dose for 50 percent of a human population (LD50) for this drug is estimated to be 2,000 mg/kg body weight.[41] That means that a 50-kg (110 lb) child must drink 83 liter-bottles of a 0.12 percent solution in a short time to risk a 50/50 chance of dying. At 11 percent ethyl alcohol, or 22 proof, this is highly unlikely. However, patients who are sensitive to alcohol-containing products should be offered an alternative.

Recently CHX has been used to treat mothers highly infected with *ms*. Treatment was sequenced to reduce or eliminate *ms* at the time the first teeth were erupting in these mothers' children.[42] The treated group's children had significantly fewer cavities at age 3 than the untreated group.

Other therapies also exist. In Europe and Canada, fluoridated varnishes (Durafluor/Duraphat or Fluor Protector) are available and effective in preventing carious lesions on smooth surfaces.[34,43] Where available,

these varnishes may be used when you believe that compliance may not be as high as you would like or for the convenience of the patient. Fluoride is a highly effective bactericidal agent. It also provides an environment conducive to remineralization.

The suggested application sequence follows the work of Petersson in which the varnish is applied to clean, isolated and dried quadrants three times weekly (for example, Monday, Wednesday, Friday), once a year.[44] This meets the mandate of short-term, intensive treatment. Sampling at the recall appointment after treatment generally confirms that a therapeutic endpoint (elimination of *ms* infection) is reached after this intense regimen.[45]

DIETARY AND SALIVARY FLOW EXAMINATION

Although it is often difficult to change the long-term dietary habits of patients because of their social or cultural backgrounds, single-event problems like large amounts of sucrose-sweetened coffee, are relatively easy patterns to alter. Dietary advice should be offered to those with the need.

Xerostomia is a serious problem in specific populations. Saliva is one of the major buffering systems in the mouth. It can significantly decrease the acid challenge of cariogenic bacteria by quickly buffering the bacterial acids to a pH that will not demineralize the tooth. In addition, saliva is the primary remineralization fluid for the teeth. Decreased flow prevents remineralizing the areas that have been demineralized by acid attack. This lack of remineralization results in a more rapid progression of caries. Salivary substitutes and other strategies should be used to help counter these problems.

ADJUNCTIVE THERAPIES

One treatment consists of recommending xylitol gum (Xylifresh, Henry Schein) for patients who chew gum. This gum not only demonstrates non-cariogenic properties, but actually appears to be anti-cariogenic.[46] Xylitol is a five-carbon sugar alcohol that is not a fermentable substrate for *ms*. (In humans it is a normal sugar found in the pentose shunt in the Krebs cycle.) It has the same agreeable taste as sucrose and appeals to children. Xylitol's anti-cariogenic properties have been adequately demonstrated in the Turku sugar studies.[47] These longitudinal trials from Finland show not only decay reductions, but also actual reversal of minimal lesions.[46]

An initial negative effect on *ms* populations has been demonstrated in vivo.[48] The actual cause of the reduction in carious lesions is speculative. The essence of all the arguments is that *ms* lose a competitive advantage in the oral ecosystem when exposed to adequate quantities of xylitol. The bacteria are affected even with concurrent sucrose intake.[46]

Chewing gum is a highly suitable delivery vehicle. The protocol is to chew two pieces, three times per day, for five minutes per chewing experience. Less exposure significantly reduces the gum's efficacy.[49] Encourage gum-chewing patients to chew this gum in lieu of regularly sweetened gums or so-called sugar-free gums containing mannitol and sorbitol. *Mutans streptococci* ferment these sugars to acid, albeit at a reduced rate.

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An additional adjunct is frequent administration of ADA-approved over-the-counter fluoride rinses and the use of a fluoride dentifrice. Fluoride rinses may begin at the end of the 14-day chlorhexidine regimen. Fluoride has three basic mechanisms of action in caries:

- It is a powerful bactericidal agent for *ms* and other acid-producing organisms.
- It facilitates remineralization by prejudicing the remineralization/demineralization rate equation toward remineralization.
- It forms acid-resistant carbonate apatite crystals during the remineralization process.[50]

Instruct patients to use the OTC rinse, in addition to their fluoridated toothpaste, at least twice per day, at times separate from their brushing. The presence of the fluoride ion, with xylitol-stimulated saliva providing a supersaturated solution of

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calcium and phosphate, aids in remineralizing early carious lesions.[49]

RECALL

This completes the primary and supplemental treatments for controlling the infection. Patients who continue to chew the xylitol gum generally have a low caries recurrence.

Similarly, those who choose to continue to rinse twice daily with the OTC fluoride rinse will continue to suppress *ms*, while remineralizing the previous demineralized areas.[50] The first recall should occur three months after the completion of CHX or fluoride varnish antimicrobial treatment. Recall consists of:

- an *ms* level determination;
- a clinical examination;
- examination and repair of any defective pit and fissure sealants.

We need to know when an *ms* infection has recurred and the magnitude of infection, since there appear to be critical levels of reinfection that lead to carious lesions. An infection of 3,000 CFU/mL of *ms* in saliva appears necessary to colonize susceptible pits and fissures.[9] A level of 43,000 CFU/mL can establish a smooth surface infection.[18] Although these numbers are not absolutes, they provide guidance for therapeutic treatment of an *ms* infection. We recognize that 10,000 CFU/mL is highly significant in the patient with only three remaining teeth. It is not generally significant for the patient with sealants in place and a full complement of 28 teeth.

In-office tests can diagnose the presence and magnitude of an *ms* infection (Strip Mutans, Vivadent/Ivoclar NA). These tests are primarily selective media culture tests in that they limit growth in or on a nutrient media to the target organism. These simple tests can accurately diagnose levels of *ms* in saliva.[51] These tests have rather low sensitivity for caries (true-positive), but relatively high specificity (true-negative).[52-54] This means that these tests are poor at predicting who will get carious lesions based on specific salivary levels of *ms*. But the tests have high specificity. They are good at predicting low caries levels when a patient has low *ms* counts. To use these tests to the best advantage, the specific sequential strategies outlined here should be used and microbial testing reserved for the recall appointments of caries patients.

Establishing a consistent recall program is important. If the pit and fissure sealants fail, they usually fail relatively soon after placement.

Notice that a pit and fissure sealant application has eliminated consideration of the 3,000 CFU/mL *ms* diagnosis point from a treatment scheme, because the pits and fissures are not available for reinfection.

Treatment is directed toward only those patients who have demonstrated a lack of resistance to this organism (previous history of caries) and who have >100,000 CFU/mL *ms*. This figure is selected as the retreatment value, since it is a convenient testing point and precludes overtreating patients.

Establishing a consistent recall program is important. If the pit and fissure sealants fail, they usually fail relatively soon after placement.[55, 56] After the first three-month appointment, recall should be scheduled at three-month intervals. These recalls use the same protocol as the first three-month recall. If the infection recurs, the clinician once again gains control with the CHX regimen or the fluoridated varnish.

These recall programs should ideally be managed by the dental office staff. They can easily learn the sampling techniques required for the bacteriologic testing, and they can perform any treatments allowed by their training and by local licensing laws (for example, pit and fissure sealant placement). Additionally, assistants should schedule the recall of patients, make appointments and keep the dentist informed of the patient's bacteriologic status.

CONCLUSION

This paper has presented a cognitive or medical treatment model for dental caries based on the current state of our knowledge. It deviates from the traditional surgical model in that treatment is directed toward elimination or reduction of the caries causing bacteria. The bacterial and clinical diagnostics and treatments will change over time. As better diagnostic and treatment

modalities emerge, new schema will evolve. But the underlying model will remain the same. Treatment directed at the causative organisms is the key to our truly becoming the healers of the mouth. ■

Information about the products mentioned in this article may be available from the authors. Neither the authors nor the American Dental Association has any commercial interest in the products mentioned.

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