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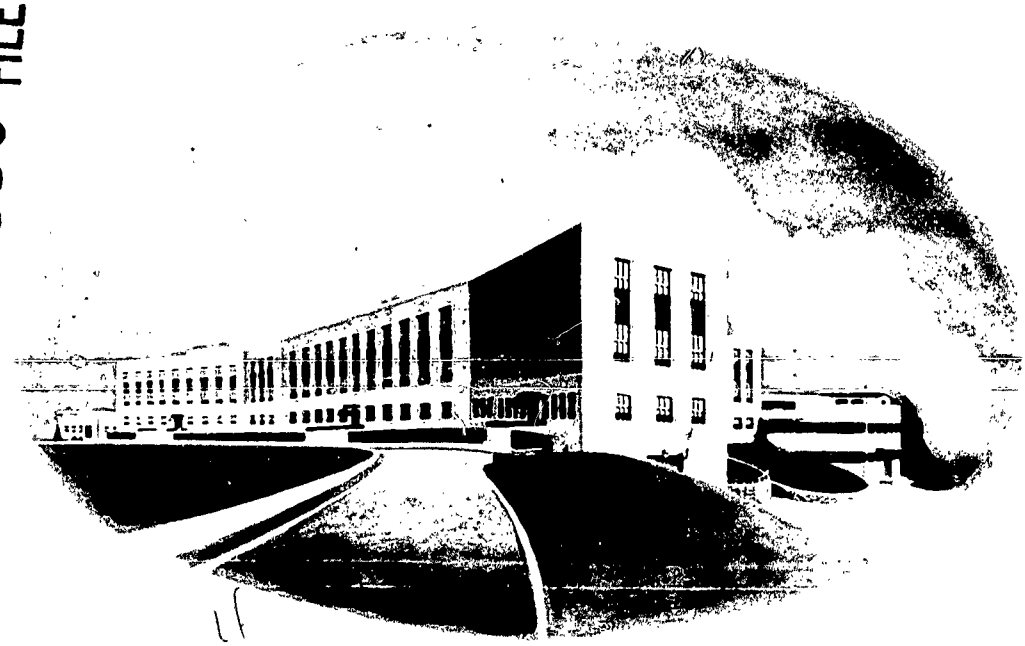

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THE TRAUMA OF COOLING IN OPERATIVE DENTISTRY

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By Carl A. Ostrom

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The Trauma of COOLING in Operative Dentistry

Histology shows that air spray is more traumatic to pulp tissue than air-water coolant. Now a new standardized experimental procedure adds evidence that air-cooling actually alters the nature of dentine in a cavity preparation

A recent detailed review by Stanley (*1*) has shown that each step in cavity preparation and restoration is accompanied by some degree of pulpal inflammatory change. Individual variations also influence response to modern high-speed cutting instruments. To minimize the variables and to obtain a sufficient number of experimental samples as nearly identical as possible, Maurice and Schour (*2*) developed a method using rat molars for the study of pulpal reactions to experimental injuries. Refinement of the method by Paynter and Wood (*3*) permitted more accurate control of cavity size.

Exploratory trials with the rat-molar method of replicate cavity preparation demonstrated pulpal inflammatory response in the form of displaced odontoblast nuclei and led to the identification of the coolant device as a factor to be controlled in further standardization of the method.

On the hypothesis that the normal permeability of dentine may be altered by operative techniques, the present studies compared the effects of cavity-cutting with air coolant and cavity-cutting with air-water coolant. The penetrability of dentinal tubules in freshly cut rat-molar cavities was tested with *Botulinus* toxin which, if it penetrated the dentine, would produce somatic muscle paresis. This toxin is known to paralyze the autonomic nervous system by action at the synapses of efferent parasympathetic nerves and somatic motor nerves, without itself causing morphologic changes (*4*).

Preliminary Studies

Subjects and Apparatus

Rats of either the NMRI-D or the Long-Evans strains, aged 70–85 days, yielded reproducible results with minimal difficulties at the gingival crest. The male Long-Evans rat at 80–85 days was used in these experiments because of its relative caries inactivity and its more hardy nature.

Cavity-cutting apparatus and technique were patterned after Paynter and Wood (*3*), with modifications. The ratchet on the fine-adjustment knob of the handpiece-

bearing microscope tube was made to click each time the handpiece was advanced 10 microns. Commercially available number 76 (0.332 mm.) high-speed wire-twist drills, fitted and soldered in brass shanks patterned after the straight handpiece bur, were used exclusively. A new drill was used for each cavity preparation. Irregularities made it necessary that each new drill, prior to use, be inspected at 15 magnifications, while stationary for uniformity of the cutting edge and while rotating slowly in the handpiece to insure that it ran true.

When early trials indicated that air spray at room temperature, delivered to the cavity during its cutting, produced a moderate pulp reaction, the handpiece was fitted with room-temperature air-water spray, which usually produced no histologically evident pulp changes. In the present studies both cooling devices were used to compare their effects on pulp tissue.

Cavity Depth

To determine the depth of cavity which might be cut so that it entered the dentine but did not expose the pulp, cavities were cut in the maxillary first molars of thirty rats at various depths in random distribution (Table 1). Immediately after each cavity had been cut, it was loosely filled with a moist cotton pledget, which was left in place until the animal was terminated; each molar block was placed in formalin within 15 minutes after the cavity preparation.

As Table 1 shows, 400-micron cavities were too deep, while 250-micron cavities were too shallow. A depth of 350 microns was chosen for the replicate cavity preparations of this experiment.

Although observed pulp exposures were obvious, it seems likely that some dentinal cavities may also have been present without being apparent, since angulation of the paraffin block in which these small specimens were embedded proved extremely difficult.

Preparation of the Cavities

With the Nembutal-anesthetized rat fixed to the table and the subject molar under 15 magnifications and focused light, the drill was aimed at the gingival crest with about half the drill's diameter mesial to the groove on the mesiolingual slope of the maxillary first molar, taking care to approximate a visual right angle between the drill and tooth surface. After deflecting the gingiva, the drill was advanced to contact and then backed out of contact with the enamel surface. With the engine rotating at 1300 ± 40 r.p.m., the drill was advanced until the first enamel shavings were observed. At this point the air coolant or air-water coolant spray was turned on. Guided by an electrical interval timer and the ratchet, i.e., aural and digital senses, plus visual observation, the drill was ad-

TABLE 1

MINIMUM REMAINING DENTINE AT VARIOUS CAVITY DEPTHS

	400 μ	350 μ	300 μ	250 μ
Number of cavities cut	10	20	20	10
Relation of cavity to pulp:				
Pulp exposed	5	3	0	0
No cavity in dentine	0	2	4	4
Mean minimum remaining dentine, μ		104 (S.D. 23)	112 (S.D. 49)	

vanced 10 microns each 15 seconds until the planned cavity had been cut. Excess water and saliva were removed by aspiration.

After scheduled exposure to the test agent or control saline, each cavity was wiped with a cotton wick, and an amalgam restoration was placed—using minimal pressure to avoid the pulp response to pressure alone—as described by Swerdlow and Stanley (*5*). The first cavity was restored before the second was cut. The animal was then returned to his individual cage to recover from the anesthesia. Each cage contained lab chow pellets and distilled water to be taken ad libitum. Except where otherwise noted, all animals were observed at 2-hour intervals for 48 hours and then daily for 5 consecutive days. Upon completion of the observations, each animal scheduled for histologic examination was overdosed with ether, and each block of molars was excised and fixed in 10 per cent neutral formalin. Serial decalcified sections cut from paraffin blocks were stained with hematoxylin and eosin and then examined.

The Botulin Transmission Studies

The Test Agent

Exploratory intraperitoneal studies indicated that 1×10^{-3} ml. *Botulinus* toxin, type D, was fatal to all of two groups of ten animals; but 1×10^{-4} ml. caused paresis for as long as 24 hours and lethargy in the 72 hours afterward, but no deaths, in two groups of ten animals. Placement of 8×10^{-3} ml. toxin in the oropharnxy of two groups of ten Nembutal-anesthetized rats produced no symptoms in the following 96 hours. The latter volume completely filled a prepared molar cavity. Passive immunization from 0.1 ml. of the corresponding antitoxin intraperitoneally protected from 1×10^{-3} ml. I.P. and from the toxin placed in molar cavities in the manner to be described.

The first supply of *Botulinus* toxin was depleted in Experiments A and B, so that a second supply was obtained for Experiment C. A rough check showed this second supply's titer to be of the same general order of magnitude as the original, although 1×10^{-3} ml. I.P. was fatal to only seven of ten rats.

The Experimental Groups

A. *Air coolant; 0–60-minute exposure to botulin.*—In ten rats, 350-micron cavities were cut in each maxillary first molar, using room-temperature air as coolant. In five rats, 8×10^{-3} ml. botulin was placed in each cavity, while isotonic saline solution was placed in the two molar cavities of five control rats. After 30 minutes, the botulin or saline was absorbed from the first cavity with a cotton wick, an amalgam restoration was placed, and the second cavity was similarly prepared. After the second restoration, 8×10^{-3} ml. botulin was placed in the oropharynx of the five controls. Subsequently, five additional rats were treated with a 30-minute exposure to botulin in one of the cavities and 30 minutes of saline in the other.

B. *Air-water coolant; 0–10-minute exposure to botulin.*—In eighteen rats, replicate cavities were cut, using air-water spray as coolant. Six were treated with botulin in both molars; six had botulin in one molar and saline in the other; and six had saline in both. All exposures in this group were 5 minutes, whether to test or control material.

C. *Randomized group.*—Replicate cavities were cut in two groups of ten rats. Air coolant was used with one group; air-water coolant with the other. In five animals of each group, both cavities were exposed to botulin for 5 minutes each. In the other five animals of each group, both cavities were filled with botulin, each for 30 minutes.

Evidence of the Penetration of Botulin

Table 2 summarizes the conditions of the three experiments and the incidence

TABLE 2

BOTULIN TOXICOSIS THROUGH EXPOSURE OF CUT CAVITIES IN RELATION TO COOLING DEVICE USED

No. of Rats	Exposure to Botulin (min.)	Conditions	Evidence of Toxicity
	A. Air Coolant: 0–60-Minute Exposure to Botulin		
5.....	60	Botulin in two molars	Paresis in all
5.....	0	Saline in two molars	None
5.....	30	Botulin in one; saline in other molar	Paresis in all
	B. Air-Water Coolant: 0–10-Minute Exposure to Botulin		
6.....	10	Botulin in two molars	Paresis in 4
6.....	5	Botulin in one; saline in other molar	Paresis in 1
6.....	0	Saline in two molars	None
	C. Randomized Group: Botulin in Two Molars of Each Animal		
5.....	10	Air coolant	Unco-ordinated gait—1
5.....	60	Air coolant	None
5.....	10	Air-water coolant	Unco-ordinated gait—1 Paresis in 2
5.....	60	Air-water coolant	Unco-ordinated gait—1 Paresis in 4

of toxicity, which constitutes evidence of normal dentinal permeability. In Group A, the five rats with botulin in two cavities and the five with botulin in only one all showed paresis during the first 72 hours after treatment, and all those observed appeared normal thereafter. The five control rats showed no recognizable toxic reactions.

In Group B, less evidence of toxicity appeared with shorter exposures to botulin; however, four of the six rats with two botulin-exposed cavities and one rat with one test cavity showed paresis at some time during the 14–48 hours after exposure. After 48 hours, no paresis was observed, and, after a week, all remaining rats appeared normal.

In Group C, in which long versus short exposure to botulin and air versus air-water coolant were presented in the four possible combinations to five animals each, air coolant clearly emerges as the most traumatic to dentinal permeability. Only one rat of the ten prepared with air-cooled cavity-cutting showed signs of botulin toxicosis during a 48-hour period of observation. Eight of the ten prepared with air-water coolant gave evidence that the toxin had passed through the cut dentinal tubules.

Histologic Findings

A. *Air coolant.*—One molar of experimental Group A showed a pulp exposure

which had occurred during cavity preparation; except for a moderate accumulation of acute inflammatory cells plus intracellular eosinophilia subjacent to the exposure, this pulp was unremarkable. The other nineteen molars from rats who had had either botulin or saline in both cavities showed similar normal-appearing pulps except for the pulpal ends of the cut dentinal tubules. In that area, in a single section, as many as ten displaced odontoblast nuclei might be seen in the dentinal tubules and up to twenty-five in the predentine tubules. The remainder of the odontoblast layer was irregular and displaced toward the predentine. The normally cell-free zone was occupied by cellular and fibrous elements of the pulp invaded by relatively low numbers of erythrocytes and leucocytes. Intravascular hyperemia, if it existed, was not recognized.

Similar pulpal responses were seen in three rats of this group who had had botulin in one cavity and saline in the other and who were terminated for histologic study at 24 hours. This rapid response of the pulp to cavity preparation employing air cooling was also seen in the animals so prepared in the cavity-depth study. These findings are consistent with those of Brännström (*6*) and Langeland (*7*).

The two remaining rats of Group A were terminated after 6 weeks. The pulp tissue appeared entirely normal, with no odontoblast nuclei or other material within the tubules, but with a slight widening of the predentine layer related to the pulpal ends of the cut tubules. The disappearance of intratubular odontoblast nuclei after 6 weeks' healing from mild trauma coincides with the observations of James and Schour (*8*), who observed that after 2 weeks the displaced nuclei had degenerated to granular debris.

The twenty specimens of Group A showed a mean minimum remaining dentine of 128 microns (S.D. 37).

B. *Air-water coolant.*—Histologically, all the pulps of Group B were essentially normal, and it was not possible to distinguish between the botulin-treated and the control teeth. Of the fourteen teeth from animals terminated at 24 and 48 hours, eleven showed no recognizable response. The other three showed a low degree of extravascular hyperemia in the normally cell-free zone, an occasional displaced odontoblast nucleus, and some edematous areas subjacent to the cut tubules. The twenty-two cavity preparations which were allowed to heal for a week showed no intratubular material, but they did show a hyperactive predentine zone and disarrangement of the odontoblast layer. Related to the findings of Langeland (*7*) and Zander (*9*), these might be considered minimal pulp responses.

The thirty-six specimens of Group B showed a mean minimum remaining dentine of 143 microns (S.D. 57).

Interpretations

The fact that the specimens in Table 1 showed more uniform thickness of remaining dentine than did the specimens in the botulin Experiments A and B—as revealed in the lower standard deviation—may be related to the fact that the former cavities were all cut by a single operator, whereas the latter were randomly cut by three. Dentine thickness in a given specimen might be influenced by two independent factors: the operator's positioning of the drill and the angulation of the specimen block in relation to the microtome blade. The variation in cavity depths suggests that even this carefully controlled cavity-cutting technique is subject to human judgment and dexterity.

The high frequency of botulin transmission through dentine cut under air-water coolant compared to cases cut under air coolant is a striking feature of Experiment C. The apparent discrepancy of Experiment A, in which all ten air-coolant animals showed botulin toxicity, may be due to a difference in titer between the two supplies of botulin.

Experiment C suggests that the greater

FIG. 1.—Pulp response 24 hours after cavity preparation using air-water coolant followed by a 5-minute exposure to botulin and an amalgam restoration. There is insignificant displacement of odontoblast nuclei (*a*) but moderate hyperemia (*b*) and extravascular edema (*c*) in the subodontoblastic area subjacent to the cut dentinal tubules (H&E, ×462).

FIG. 2.—Seven days after cavity preparation using air-water coolant plus a 5-minute exposure to botulin and amalgam restoration. The odontoblastic layer is more disrupted than earlier (*a*). In relation to the cut dentinal tubules, at lowest depth of cut cavity, a hyperactive zone of predentine appears (*b*) (H&E, ×192).

trauma of air-cooled cutting caused reduced permeability of the cut dentinal tubules. It is not known whether this was the result of alteration of tubular proteins due to heat, whether it was the result of increased pressure and stasis associated with pulp inflammation and edema localized at the ends of the cut tubules, or whether it resulted from partial occlusion of tubules by displaced odontoblasts. The fact that botulin of another source did penetrate tubules cut under air coolant in Experiment A indicates that, in any case, the altered permeability is only partial.

These observations on the influence of the coolant used in operative dentistry support those of Stanley, who concludes in his recent review (*1*) that adequate sealing of cut tubules to offset the irritating properties of dental materials was more important after modern high-speed cutting techniques using an adequate air-water spray than it was after the conventional slower cutting and air spray.

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NOTE.—The opinions or assertions contained herein are the personal ones of the writer and are not to be construed as reflecting the views of the Navy Department or the naval service at large.

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FOR THE BUSY READER

Summaries and Biographies

The Trauma of Cooling in Operative Dentistry · *Ostrom*

Large numbers of virtually identical cavity preparations, made available by a new experimental procedure, permitted comparison of the traumatic effects of cooling devices commonly used in modern practice. Air-water coolant usually produced little or no pulp reaction, while cavity-cutting associated with air spray resulted in immediate intratubular displacement of odontoblast nuclei. This study also introduced a test agent, *Botulinus* toxin, into cavities cut using one or the other cooling device. The more effective passage of the toxin through tubules cut under air-water spray showed normal permeability of the dentine, while air-cooling in some way altered the dentine and inhibited penetration of the test agent.

EDITOR'S NOTE.—High-speed cutting devices have brought the attention of many dentists to the reaction of pulp tissue to high-speed cutting of enamel and dentine. This study is well done and treats the effect of air-water coolant on odontoblast displacement.

Dr. Ostrom's second study on "Pulp Damage by Induced Inflammation" will appear in a later issue of *Dental Progress*.—G. W. T.

Carl A. Ostrom holds the rank of captain, Dental Corps, United States Navy, and is head of the Dental Division, Naval Medical Research Institute. He earned B.S.D. and D.D.S. degrees from Northwestern University Dental School in 1938. Private practice in Chicago combined with an instructorship at Northwestern occupied his time until 1941, when he joined the navy. After various clinical tours, he attended a navy-sponsored course at Northwestern and earned an M.S.D. degree in 1948. Ostrom's next assignment was to the joint laboratories of the Naval Biological Laboratory, Naval Medical Research Unit No. 1 and the University of California, Berkeley. As a result of his work in oral aspects of respiratory infection, he was awarded the Legion of Merit. At the Naval Medical Research Institute, Bethesda, Maryland, he is continuing efforts in the field of preventive dentistry. He is author or co-author of some twenty-four scientific contributions.

REPRINTS.—Carl A. Ostrom, Captain, Dental Corps, USN, Naval Medical Research Institute, Bethesda 14, Maryland.