

Human Saliva: A Study of the Rate of Flow and
Viscosity and its Relationship to Dental Caries

A Thesis

by

Ralph E. McDonald, B.S., D.D.S.

November 22, 1950

Submitted to the Faculty of the Graduate School In Partial
Fulfillment of the Requirements In the Department of Dentistry
Indiana University

TABLE OF CONTENTS

INTRODUCTION	1
REVIEW OF THE LITERATURE.....	2
Salivary Secretion.....	2
Nervous Stimulation of Salivary Glands.....	3
Salivary Reflexes.....	4
Normal Salivary Secretion.....	5
Constituents of Saliva.....	5
Functions of Saliva.....	6
NORMAL SALIVARY FUNCTION.....	7
Rate of Flow.....	7
Relationship To Dental Caries.....	8
DYSFUNCTION OF SALIVARY GLANDS.....	9
Xerostomia.....	9
VISCOSITY OF SALIVA.....	10
MATERIALS AND METHODS.....	10
DMF Rate.....	11
Paraffin Stimulated Saliva.....	11
Determination of Viscosity.....	12
PRESENTATION OF DATA.....	13
DISCUSSION OF FINDINGS.....	14
SUMMARY.....	15
CONCLUSIONS.....	17
BIBLIOGRAPHY.....	18

Human saliva, a continuous secretion of the salivary glands, should be considered as the product of a natural body function, necessary for the health and well-being of the organism. As the knowledge of human physiology has increased additional importance has been given to the normal salivary secretion. Subsequently, as we advance with our understanding of the body's function new findings will no doubt serve to re-emphasize the importance of this fluid. A vast amount of literature can be found on the subject of saliva and there are excellent reviews^{1,2} which point out the relationship between normal and pathological conditions, such as dental caries. Since the teeth are continually bathed with the products of the salivary glands it has long been thought that the cause of dental caries, or the reason for the freedom from, could be found in the saliva. Therefore, much research has been directed in the direction of finding the relationship between dental caries and saliva.

Dental caries is a disease of the calcified tissues of the teeth. It is caused by acids resulting from the action of microorganisms on carbohydrates, is characterized by a decalcification of the inorganic portion, and is accompanied or followed by a disintegration of the organic substance of the tooth.³ This caries process is controlled to some extent by natural mouth protective mechanisms inherent within the saliva,⁴ many properties of which have been investigated to learn their possible role in the caries process. Considerable importance has been placed on salivary pH,^{5,6} the acid neutralizing power,^{7,8} and the calcium^{9,10} and phosphorus¹¹ content.

It has long been suggested that in addition to these properties the rate of flow and viscosity of saliva may influence the development of caries; however the findings of research workers have been contradictory and inconclusive.

12

The purpose of this investigation was to observe the stimulated flow of human saliva to determine whether there are significant individual differences in rate of flow and viscosity. These properties were then to be considered for their possible correlation with dental caries.

Review of Literature

Saliva is secreted by three paired masses of cells, the submaxillary, the sublingual, and the parotid glands. There are also small glands, each of which has its own duct, scattered over the buccal mucous membranes, which secrete mucoid fluid. The submaxillary gland contains alveoli, which secrete a mixed type of fluid, a thin, watery type and also a thick, viscid juice rich in mucin. The secretion enters the oral cavity through Wharton's duct, which opens upon the floor of the mouth to one side of the frenum of the tongue. The sublingual alveoli are predominantly of the mucous type, although a few serous alveoli are present. The secretion may be either thin and watery or viscid, the character depending upon the nature of the secretory stimulus. The ducts of the Rivinus, several fine tubes, open beside the frenum of the tongue permitting the saliva to flow into the mouth. This secretion is usually of the mucous type.

The parotid secretion, thin, and low in organic content, enters the mouth through the duct of Stensen, located upon the inner surface of the cheek opposite the second molar tooth.

13

14

Since there is no evidence of a specific hormonal mechanism no doubt a nervous mechanism alone brings about salivary secretion. The salivary

glands are under the control of the autonomic (involuntary) nervous system, receiving fibers from both its parasympathetic and sympathetic divisions. The parasympathetic fibers to the submaxillary and sublingual glands arise from a center - the superior salivary center - in the pons; they leave the brain in the facial nerve and are conveyed in the chorda tympani branch of the latter nerve to the cavity of the mouth. Here they join the lingual nerve which transmits them to the floor of the mouth where, separating again from the lingual fibers they make connections with ganglion cells situated in close relationship with the sublingual gland, or within the substance of the submaxillary gland. Postganglionic fibers - the axons of the ganglion cells - pass to the secreting cells.

The chorda tympani nerve also carries vasodilator fibers to the glands and taste fibers to the anterior two-thirds of the tongue. The submaxillary and sublingual glands also receive secretory and vasoconstrictor fibers from the sympathetic plexus investing the neighboring branches of the external carotid artery. The parotid gland derives secretory and vasodilator parasympathetic fibers through the tympanic branch of the glossopharyngeal nerve. These fibers arise from a group of cells in the upper part of the medulla oblongata called the inferior salivary center. Vasoconstrictor fibers to the parotid are derived from the sympathetic.

Stimulation of either the parasympathetic (chorda tympani) fibers or the sympathetic fibers to the submaxillary or sublingual gland causes a secretion of saliva. The secretion resulting from parasympathetic stimulation is, in most animals, profuse and watery in consistency; sympathetic stimulation on the contrary causes a scanty secretion of a thick mucinous juice. Apparently, then, the parasympathetic fibers innervate the serous

cells, and the sympathetic the mucous cells. Stimulation of the parasympathetic fibers to the parotid gland causes likewise a profuse watery secretion whereas no secretion follows stimulation of the sympathetic.

Under natural conditions, the secretion of saliva is a reflex phenomenon brought about usually by the stimulation of the taste fibers in the mouth. But stimulation of the ordinary sensory nerves in the mucosa of the mouth, i.e., those fibers conveying sensations of touch, pain and temperature, evokes a flow of saliva. We are all familiar with the salivation which results from moving some tasteless object around in the mouth, or from the manipulations of the dentist. Indeed, secretion of saliva may follow stimulation of almost any afferent nerve in the body. In animals stimulation of the central cut end of the sciatic nerve excites salivary secretion and, in man, irritation of the nerves of the stomach or esophagus, in diseased states, is not uncommonly a cause of troublesome salivation.

Salivary reflexes are of two types. The one caused by the introduction of material into the mouth is called an unconditioned reflex. But it is well known that salivation very often results from the sight, smell or even the thought of food. We often hear a person say that his "mouth waters" when he sees or smells appetizing food. The response following such forms of stimulation is called a conditioned reflex. The unconditioned reflex is inborn; it can be elicited immediately after birth. The conditioned reflex, on the contrary, depends upon experience. In order for it to become established, an association, upon some previous occasion, must have been formed in the mind between sensations received through the nerves of taste and those gained through some other sense organ, eg., eye, nose, ear, etc.

In man the amount of saliva secreted in 24 hours amounts to 1000 cc. to 1500 cc. Ordinary mixed saliva contains about 99.5 per cent of water and 0.5 per cent total solids. It has a specific gravity between 1.002 and 1.012. Its main constituents are as follows:

1. Salts (approximately 0.2 per cent)

Sodium and potassium chloride

Sodium bicarbonate

Acid and alkaline sodium phosphates

Calcium carbonate and calcium phosphate

Potassium sulphocyanate

2. Gases

Carbon dioxide, oxygen and nitrogen

3. Organic Substances

Ptylin (salivary amylase) and maltase

Serum albumin and globulin

Urea, uric acid and creatine

Mucin, mainly in the submaxillary and sublingual secretions

Vitamin C

The bicarbonate and, to some extent, the phosphates act as "buffers". The chlorides are necessary for the activation of the amylase. The calcium salts which are soluble in acid but insoluble in alkaline media tend to be thrown out of solution when the pH rises. The carbonate and phosphate of calcium may be deposited in the form of concretions (salivary calculi) within the ducts or, in combination with organic material, may be laid down on the teeth as "tartar". The potassium sulphocyanate is an excretory product and is probably formed within the body from CN radicals derived from the metabolism of protein.

Saliva is normally given the role of providing the following functions in the mouth:

1. Digestive. The starch molecule is acted upon by ptylin in saliva and split into smaller molecules of the disaccharide maltose. The rapid passage of food through the mouth precludes the possibility that it is acted upon here to any important extent. Because of its impregnation a more significant degree of digestion may take place in the stomach.
2. Preparation of the food for swallowing. The saliva moistens the food, enabling it to be rolled into a plastic mass and gives it a lubricant coating.
3. Solvent action. Dissolving solid substances so that they may stimulate the taste buds.
4. Cleansing action. The constant flow of saliva exerting a necessary cleansing effect in the mouth and on the teeth. The oral structures are rinsed and kept comparatively free from food residues, shed epithelial and foreign particles. The saliva reduces the insoluble carbohydrates to a soluble form, the end result being a cleaner oral cavity.
5. Excretory. Drugs such as mercury, potassium iodide, and lead are excreted in part by saliva.

In chronic systemic disease urea or sugar is found in the salivary secretion. Several types of microorganisms and some intensely virulent virus are excreted in saliva. It has been shown¹⁷ that sulfonamides given systemically are excreted in the¹⁸

saliva. Likewise, when penicillin is taken systemically, the salivary excretion is of sufficient concentration to affect the oral flora.¹⁹

6. Antibacterial effects. Salivary enzymes aid in the solution of food debris on which bacteria might thrive and manifest a variety of antibacterial or other anti-infectious properties. There have been ascribed to mucus, lysozyme, and other bacteriostatic (inhibins), bacteriolytic and bactericidal substances.²⁰

"Salivary corpuscles," probably leukocytes which have migrated through the mucosa or come down the salivary ducts, are often seen. These cells may constitute one of the antibacterial factors of the mouth (e. g. phagocytic).

Normal Salivary Function

Attempts have been made to determine the normal stimulated salivary flow. Brown,³⁴ working with 88 medical students, observed the paraffin-stimulated salivary flow for two consecutive fifteen-minute periods. The average during the first period was 36 cc. and slightly less for the second. A variation of 23-138 cc. was noted for the entire course of the experiment. Trimble,³⁵ working with 107 dental students, observed that the paraffin-stimulated salivary flow during a fifteen-minute interval averaged 34 cc., the amounts ranging from 9-68 cc. Twenty-eight of the 57 students having less than average flow developed new smooth surface cavities during a twelve month observation period. In the group of 50 having greater secretion than normal only fifteen developed new cavities. In a continuation of the work Cushman,³⁶ working with 21 adolescent patients, observed that those who developed no new caries had an average of 22 cc. of saliva while

those who developed four or more new cavities secreted an average of 12 cc. of saliva. After studying a group of 198 patients, Becks³⁷ concluded that no statistically significant difference could be detected between the caries-free and caries active groups on the basis of rate of flow or calcium and phosphorus analysis. Another group of 661 individuals was observed to have a resting flow of 19 cc. per hour, the range being .5-111 cc.³⁸ Slower rates of flow in general were observed from five to nine years, while after ten years of age the averages by five-year age groups did not vary significantly. No sex differences were found between the average rates of flow of males and females. Karshan,³⁹ studying the calcium and phosphorus ratios in caries-free, caries-arrested, and caries-active individuals found that not only these values differed but there was a variation in the unstimulated rate of flow. He observed that the caries-active persons had a slightly greater flow than the caries-free persons, the difference being 2.7 cc. per hour.

⁴⁰Burrell concluded from a study of 43 cases that higher rates of saliva flow and higher buffering capacities are associated with freedom from decay, but the ranges of values in each group overlapped to a great extent the ranges of values in other groups. Differences appeared only in the mean values and those differences were so small that they indicated only tendencies.

⁴¹Barany used as clinical material 155 men from the Swedish army. Their salivary flow was stimulated by chewing a piece of unvulcanized rubber for five minutes. He found there was a tendency to a higher ptylin content and a larger quantity of saliva in persons with little

caries; however the difference between persons with little and with much caries was 1.14 ± 0.55 cc.

Dysfunction

Congenital disfunction of the salivary glands is an extremely rare condition. When it occurs, changes in the oral cavity may be observed which markedly deviate from the usual oral condition. One is impressed by the septic oral condition associated with a decreased or deficient salivary flow, the usual picture being a rampant, highly destructive type of caries in a mouth which may appear dry and containing food accumulations.

Xerostomia (dry mouth) was first reported by Hutchinson in 1888.²¹ .
 The literature contains several reports of congenital disfunction of the salivary glands^{22,23,24,25,26,27} and also cases of acquired dry mouth²⁸ resulting from systemic and psychic disturbances. The oral picture in all of the cases reported followed a definite pattern -- the mucous membranes were unusually smooth though not completely dry, the tongue having a fissured appearance with deep central sulcus and lateral branching furrows. Prinz²⁹ has stated that if the flow of saliva is impaired or completely checked, all teeth will be destroyed by caries unless some other means for the removal of food debris is established, the rapidity of the destructive process being proportionally dependent upon the severity of the impairment. Experimental work in which the salivary glands of rats were extirpated seemed to confirm these conclusions;^{30,31,32} however in at least one experiment where the salivary glands of dogs were removed caries present did not progress nor did new carious lesions develop.³³

Viscosity

Few scientific observations have been made on the viscosity of human saliva. This physical property is governed not only by the particular set of glands stimulated but the type of nervous stimulation and the amount of mucin (glycoprotein) present. Miller⁴² believed that the importance of the viscosity of the saliva has been overrated. A number of cases were cited in which the saliva was so viscous it would clump on pouring and the patient was free from caries. He also recalled cases where the saliva was thin as water and yet the teeth were being rapidly destroyed by caries. Pickerill⁴³ made a similar observation -- a patient who had the least viscous saliva had an extremely septic mouth and gross caries. Zaus⁴⁴ concludes that while the degree of ropiness of the saliva is not a certain index to the degree of susceptibility it will almost uniformly be found that caries occurring in the presence of this condition will be more difficult to control than when found in similar degrees in a person with thin, watery saliva. Rae,⁴⁵ seeking to learn the relation between buffering capacity, viscosity, and Lactobacillus counts, concluded there seemed to be no significant variation in the viscosities of saliva. Centrifuged samples of saliva were used in this experiment and the viscosity was determined by means of an Ostwald viscosity pipette.

Materials and Method

Records have been taken of 132 children and young adults who were patients at the Indiana University School of Dentistry Clinics. A complete oral examination was made for each patient. The teeth were dried, and with the aid of good light, number 4 plane mirrors, and sharp exploring instruments, the number of carious tooth surfaces was determined. The

system used to record the number of involved tooth surfaces was patterned after that described by Klein.⁴⁶ The extent of caries in any single tooth was measured in terms of tooth surfaces involved. When such areas extended from one surface to others, the involved surfaces were counted separately as carious surfaces. Remaining roots and extracted teeth were considered equal to five carious surfaces. The oral examination for caries was supplemented by intra-oral periapical and/or bite wing radiographic survey. The patient's mouth hygiene and condition of oral structures were observed and if food debris, calculus, or stain was present on the teeth this finding was recorded.

A stimulated saliva sample was obtained from each patient. One gram pieces of paraffin were used throughout the study as the saliva activator since it has been shown⁴⁷ that the rate of saliva flow is increased considerably by paraffin and the flow more constant than when flavored gum or chicle is used as an activator. The patient was instructed to hold the paraffin in the mouth for half a minute before starting to chew; this softened and minimized the crumbling of the material. The paraffin was chewed for one minute and the initial saliva stimulated was swallowed along with any food debris removed by the excursion of the paraffin over the crowns of the teeth. The patient was then given a paper cup and instructed to "spit" all of the stimulated saliva into the container during the fifteen-minute period of continuous chewing.

Only one saliva sample was obtained from the majority of patients in the group. While it has been found⁴⁸ that the daily fluctuation of activated salivary flow is large when measured in terms of cc./hr., it was

⁴⁹ observed that less fluctuations occur with shorter collection periods. Patients who were readily available on five successive days were selected from the author's group for daily salivary collections. There was no significant variation in activated flow when the collection period was fifteen minutes. This has been shown ⁵⁰ to be true in experiments with dogs on a normal water intake; the salivary secretion remains nearly the same from day to day. Since previous study ⁵¹ did not disclose any seasonal influence on the rate of flow, the salivary samples were collected at convenient times during a ten-month period.

At the end of the experiment the saliva was immediately transferred to a 100 cc. graduate cylinder and the number of cc. of saliva determined. Five cc. of the freshly secreted saliva were then transferred to an Ostwald pipette (Fig. 1), a modification of Poiseuille's apparatus, generally employed for the determination of viscosity. ⁵² It consists of a fine capillary tube d - b (about 10 cm. long and 0.4 mm. bore) through which a definite volume of liquid, namely, that contained between the two marks, c and d, was allowed to flow under the force of its own weight. The apparatus illustrated in Fig. 1 was used and kept in a constant temperature bath (Fig. 2) during the determination. The test was first made with distilled water. By means of a pipette, 5 ml. of water was introduced into bulb e. By blowing into a piece of rubber tubing attached at f, the liquid was forced above mark c. The liquid column was then allowed to flow back and the time for the meniscus to pass from c to d was recorded with a stop watch. This was repeated until constant values were obtained. The water was then removed from the pipette and replaced with 5 ml. of saliva, (Fig. 3). Temperature equilibrium was attained, the procedure repeated, and

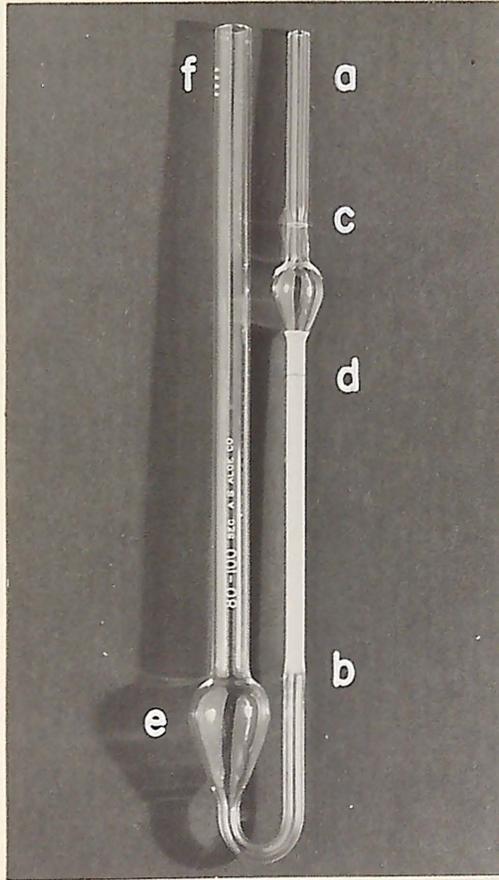


Fig. 1. Ostwald pipette for the determination of viscosity.

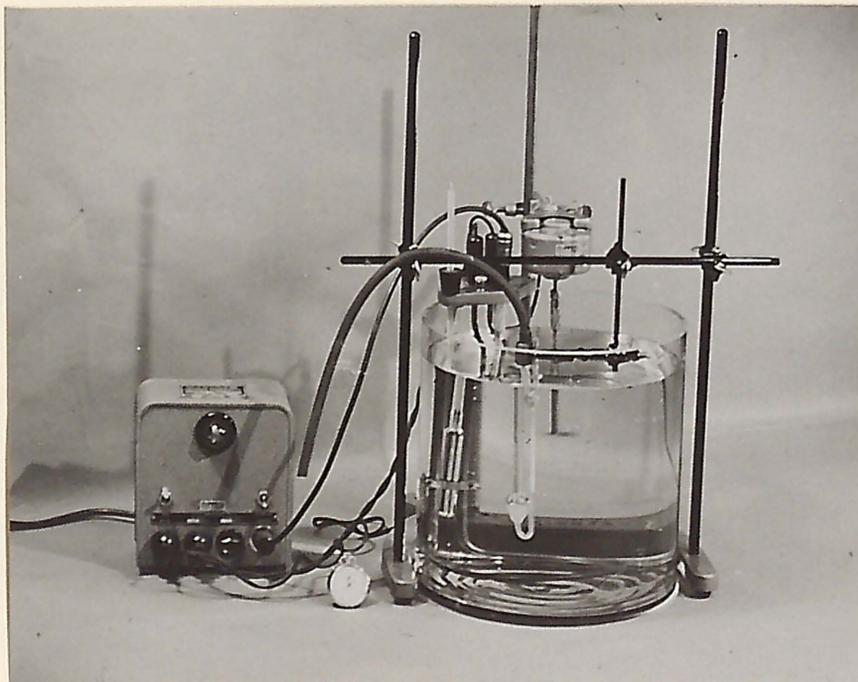


Fig. 2. Constant temperature bath used during the viscosity determination.

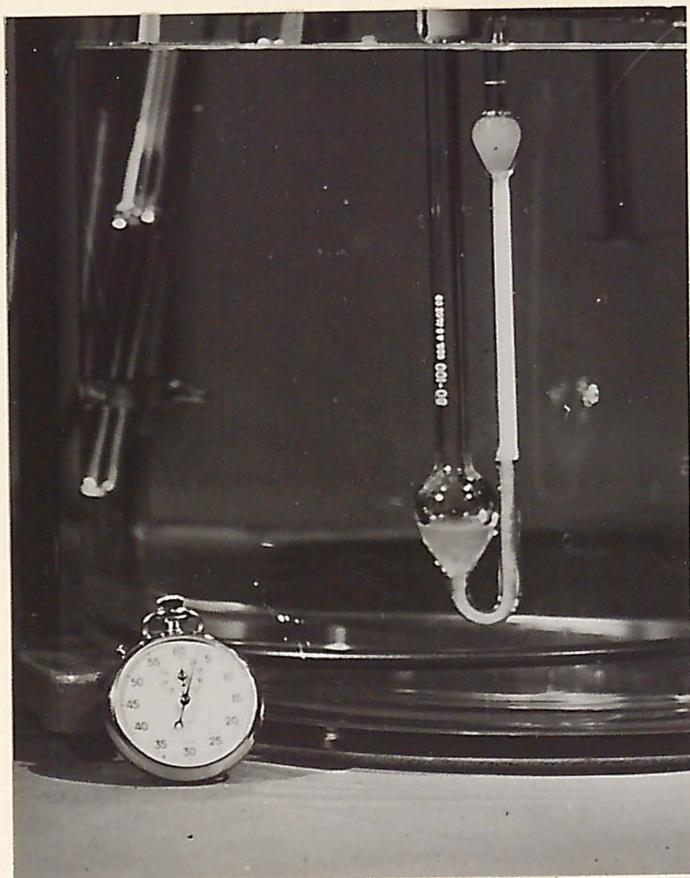


Fig. 3. A pipette containing 5 ml. of saliva immersed in the constant temperature bath during the viscosity determination.

the relative viscosity calculated as follows:

$$N = \frac{\text{time - saliva}}{\text{time - water}}$$

If sixty seconds were required for water and 120 seconds for saliva then the relative viscosity of saliva was 2.0.

Presentation of Data

The observation group consisted of 130 subjects between the ages of 7 and 48 years. The 9, 10, 11, and 12 year old groups, however, contained the greatest number, 13, 17, and 19 respectively. Since this seemed to be the most homogeneous group their records were evaluated for possible correlation between rate of salivary flow, viscosity, and dental caries. A detailed distribution of the cases selected for evaluation can be seen in Table 1. The amount of saliva secreted during the 15-minute period ranged from 3 to 50 cc. with a mean of 14.1 cc. Considerable difference was also noted in the viscosity, the range being from 1.06 to 2.16 with a mean of 1.31.

The results of the clinical examination of the 68 children in the 9 - 12 year group revealed that a small number were free of dental caries, some presented gross destruction of the teeth, but the majority of cases demonstrated the usual oral picture seen in the mouth's of an average group of children.

A boy, age 9, with xerostomia resulting from the congenital absence of salivary glands was examined. There was no evidence of an ectodermal dysplasia. A complete pediatric examination failed to show any other deficiency or abnormality. His teeth were being destroyed by the most



Fig. 4. Gross decay of the permanent teeth of a boy, age 9, with congenital absence of salivary glands.

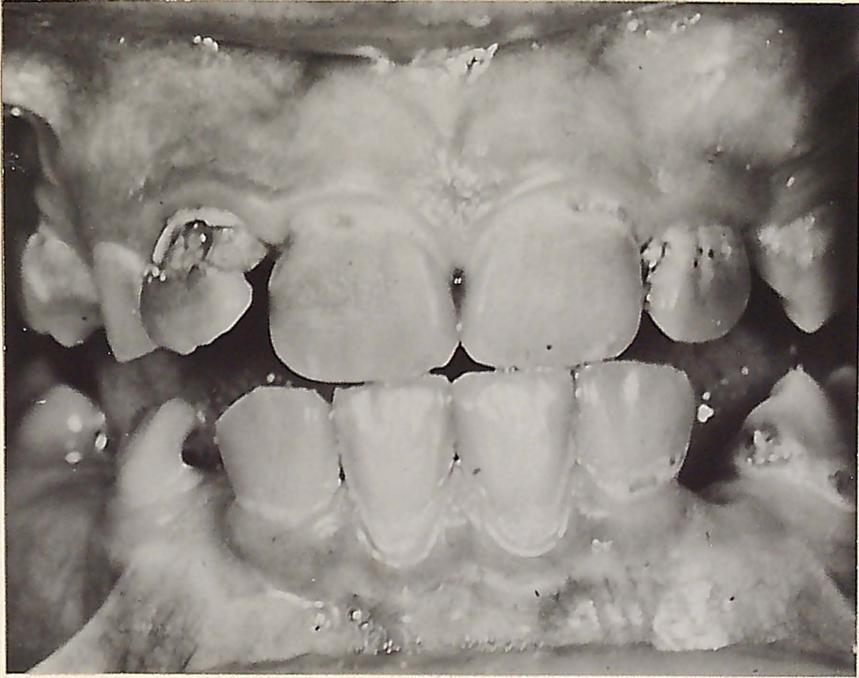


Fig. 5. Oral condition of a boy, age 11, with a deficient salivary flow and (highly viscous saliva?).



Fig. 6. Intra-oral radiographs of the above case.

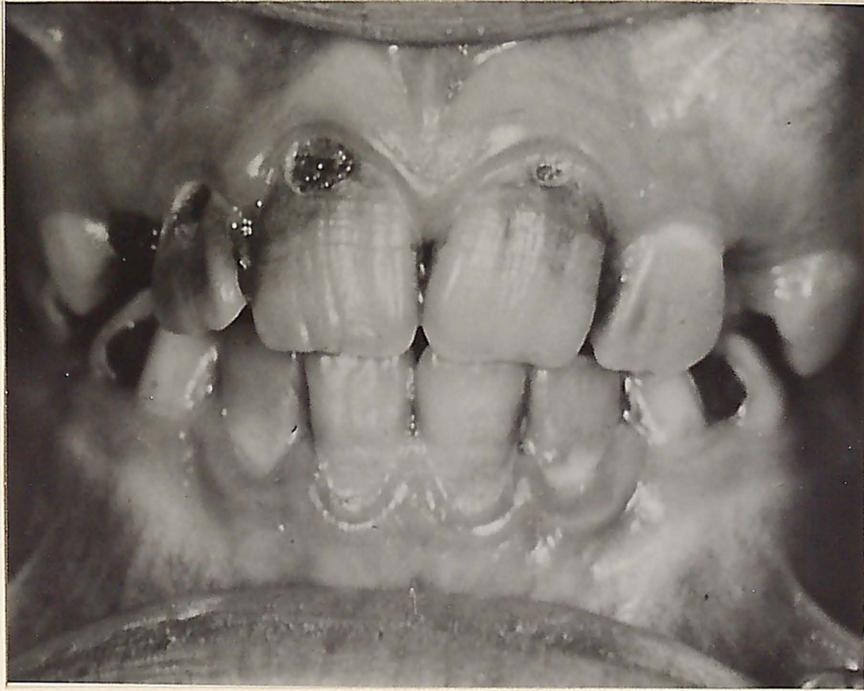


Fig. 7. Boy, age 11, with a salivary flow of 7 and viscosity of 1.34.



Fig. 8. Intra-oral radiographs showing many carious lesions.



Fig. 9. Oral and radiographic picture of a boy, age 9, with a salivary flow of 15 cc. with a relative viscosity of 1.25. The permanent teeth were free of dental caries.



rampant type of decay as shown in Fig. 4. Other children with a sparse salivary flow presented similar pictures. In Figs. 5 and 6 can be seen the condition of teeth of a boy with a salivary flow of 6 cc. in 15 minutes and with a viscosity of 1.50. A similar case can be seen in Figs. 7 and 8 where the salivary flow was 7 cc. and the viscosity 1.34.

Discussion of Findings

In contrast to the rampant decay found in those cases of deficient salivary flow, in general those children with a greater than average salivary flow experienced a relative freedom from disease of the hard tissues of the teeth (Figs. 9). The same was true of the viscosity of the saliva. A heavy viscous saliva was almost always associated with greater than average dental decay. To rule out the possibility of this being a chance observation the coefficient of correlation was determined to measure the relationship between the D M F rate and salivary flow. The coefficient of correlation was found to be -0.35999 , which means there was an inverse relationship between the two factors. In order to test the significance of this number the standard deviation was determined and found to be 0.12309 . Dividing the coefficient of correlation by the standard error the quotient was 2.925 . This was found to be statistically significant.

The coefficient of correlation between the viscosity of the saliva and the D M F rate was likewise determined. A direct correlation of 0.20056 was found, indicating some degree of relationship but it did not seem to be statistically significant for the data at hand.

The coefficient of correlation between D M F rate and saliva viscosity was calculated for the group of 132 patients of all ages. The direct rela-

TABLE I

DISTRIBUTION OF CASES SELECTED FOR SALIVARY ANALYSIS EVALUATION

<u>AGE</u>	<u>SEX</u>	<u>DMF*</u>	<u>RATE OF FLOW</u>	<u>VISCOSITY</u>
9	F	10	3	1.92
9	F	3	10	1.53
9	F	2	10	1.33
9	M	8	17	1.26
9	F	4	12	1.29
9	F	12	10	1.47
9	M	4	14	1.29
9	F	3	10	1.22
9	M	4	10	1.52
9	M	3	14	1.30
9	M	7	20	1.22
9	M	33	3	1.17
9	M	0	12	1.25
10	M	7	13	1.39
10	F	4	27	1.16
10	F	3	12	1.16
10	M	0	15	1.28
10	M	18	4	1.37
10	F	14	12	1.73
10	F	6	11	1.35
10	M	9	16	1.21
10	M	22	19	1.14
10	M	22	17	1.28
10	F	25	10	1.28
10	F	16	6	1.34

<u>AGE</u>	<u>SEX</u>	<u>DMF*</u>	<u>RATE OF FLOW</u>	<u>VISCOSITY</u>
10	F	37	6	1.22
10	F	7	12	1.22
10	M	7	15	1.20
10	F	26	24	1.22
10	F	3	23	1.28
11	F	5	22	1.26
11	M	2	25	1.32
11	F	2	14	1.32
11	M	4	8	1.16
11	M	41	6	1.50
11	F	5	18	1.38
11	F	18	6	1.37
11	F	4	21	1.35
11	M	2	20	1.15
11	F	48	6	1.56
11	F	7	13	1.49
11	M	7	16	1.29
11	F	5	10	1.22
11	F	3	50	1.10
11	M	2	30	1.37
11	F	9	23	1.06
11	F	23	14	1.23
11	M	13	13	1.35
11	F	0	18	1.20
12	M	17	13	1.15
12	F	6	13	1.28

<u>AGE</u>	<u>SEX</u>	<u>DMF*</u>	<u>RATE OF FLOW</u>	<u>VISCOSITY</u>
12	F	11	13	1.31
12	F	13	8	1.29
12	F	14	16	1.40
12	F	31	20	1.28
12	M	18	15	1.35
12	F	9	11	1.22
12	F	33	5	2.16
12	M	10	10	1.27
12	M	36	8	1.25
12	M	30	7	1.34
12	M	42	10	1.30
12	M	19	21	1.26
12	M	14	7	1.19
12	F	15	6	1.24
12	M	20	22	1.28
12	F	9	20	1.13
12	F	2	16	1.36

* Decayed, missing and filled permanent tooth surfaces.

tionship or coefficient of correlation was found to be 0.46243. In order to test the significance of the coefficient of correlation values, .46243, the standard error was determined. It was found that the coefficient of correlation was over 5 times the standard error indicating the value was not due to chance. If the coefficient of correlation is over 3 times the standard error the correlation can be considered significant.

Summary

This study was undertaken to determine whether a relationship existed between the amount of tooth decay and stimulated salivary flow and also between the viscosity of the saliva and dental decay. Paraffin-stimulated saliva samples were obtained from 132 patients between the ages of 7 and 48 years and recorded in cc. per 15 minutes. The viscosity was measured from whole, fresh saliva by means of an Ostwald pipette suspended in a constant temperature bath. It was found that since there is no compensating factor for age in the D M F rate and since the D M F rate naturally increases with age, particular age groups had to be selected for statistical analysis. Since the age groups 9 through 12 contained 68 children, the largest for any homogeneous grouping, their records were carefully reviewed. There was found to be a statistically significant inverse correlation between the D M F rate and the salivary flow. While no statistically significant correlation existed between the viscosity and D M F rate for the 9-12 group there was found to be some degree of relationship. Analyzing the entire observation group of 132 subjects however there was found to be a definite direct statistically significant degree of correlation.

The results of this study give additional information to the complicated obscure picture of the dental caries process. It has long been

realized that certain individual local factors exist which influence the incidence of dental caries. In the past it has been difficult to explain the nature of many of these factors which seem to influence the caries process. Children within the same family consuming relatively the same diet and exercising similar oral hygiene habits frequently display markedly dissimilar dental pictures. Some of the children in the family may be almost entirely free of dental decay while others have decay of the most rampant type. It has been shown in this study that individual differences in rate of flow and salivary viscosity occur within the same age group and that correlations can be made between the decay rate and these two factors.

It became evident early in the study that casual observation was not sufficient in order to reach any conclusion relative to the salivary flow. What often appeared to be a copious flow, when actually measured during a fifteen minute stimulation, was sometimes found to be rather scant. In these cases the initial flow was great immediately following the placement of an instrument or paraffin pellet in the mouth, but then in a short time the rate of flow was found to drop to a very low level. Therefore no conclusion should be reached regarding salivary flow unless it is actually measured for a definite period time. The same held true, to a lesser degree, for the viscosity.

The results obtained warrant a continuation of this study to increase the size of the sample to the point where any degree of chance could be ruled out as influencing the data. Further work is indicated to determine methods or means whereby the salivary flow and viscosity may be altered to the individual's benefit. It is possible that this may be accomplished

through dietary regulation or the administration of drugs which influence the nervous mechanism.

Conclusions

1. There was found to be a considerable individual variation in the rate of flow and viscosity of human saliva.
2. In general those children with a greater than average salivary flow experienced a relative freedom from disease of the hard tissues of the teeth.
3. In cases where there was a deficient salivary flow the dental caries experience was great.
4. A heavy viscous saliva was almost without exception associated with greater than average dental caries activities while saliva of normal viscosity was more often observed in patients with relative freedom from dental caries.

10. Swartz, R. L., and Sponck, J. C. Studies concerning the oral cavity and saliva. *Journal of Dental Research* 31:47-49, December 1952.
11. Wainwright, W. W., and Baker, Gordon. Human Saliva. Is There a Seasonal Influence on the Rate of Flow and Viscosity and Phosphorus Content of Salivary Glands? *Journal of Dental Research* 35:170-171, Aug. 1956.
12. The Michigan Workshop on the Prevention of Dental Caries. *Journal of American Dental Association* 56:4, Oct. 1957.
13. Best, C. E., and Taylor, H. B. *The Physiological Basis of Medical Practice*, 10th ed., Baltimore, Williams and Wilkins, 1945. 1189 p. (2110-422).
14. 1944.
15. Best, C. E., and Taylor, H. B. *The Living Body*. New York, 1930. 230 p. (2110-405).
16. Best, C. E., and Taylor, H. B. 1931, 1932.

Bibliography

1. Bibby, B.G. What About Saliva? Oral Surgery, Oral Medicine, and Oral Pathology 2:72-81, Jan. 1949.
2. Dewar, M.R. Some Observations on The pH of Saliva. Dental Journal of Australia 21:113-122, Mar. 1949.
3. The Michigan Workshop on the Evaluation of Dental Caries Control Technics. Journal of American Dental Association 36: 4, Jan. 1948.
4. Fosdick, L.S. Theoretical Considerations of Certain Phases of the Caries Problem. Northwestern University Dental School Research Bulletin 49: 4-14, Nov. 1949.
5. Anderson, D.D.J. The Relationships Between Hydrogen-Iron Concentration and the Rate of Flow of Saliva. Journal of Dental Research 28:583-588, Dec. 1949.
6. Dewar, M.R. op. cit., p. 113-122.
7. Hanke, M.T. The Buffer Value of the Saliva and Its Relation to Dental Caries. Dental Digest 43:235-238, 252-256, May 1937.
8. Dreizen, Samuel., et al. The Buffer Capacity of Saliva as a Measure of Dental Caries Activity. Journal of Dental Research 25:213-222, Aug. 1946.
9. Brawley, R.E. Studies Concerning Oral Cavity and Saliva. Journal of Dental Research 17:293, Aug. 1938.
10. Brawley, R.E., and Sedwick, J.S. Studies Concerning the Oral Cavity and Saliva. Journal of Dental Research 17:477-491, December 1938.
11. Wainwright, W.W., and Becks, Herman. Human Saliva. Is There a Seasonal Influence on the Rate of Flow and Calcium and Phosphorus Content of Resting Saliva? Journal of Dental Research 25:285-291, Aug. 1946.
12. The Michigan Workshop on the Evaluation of Dental Caries Control Technics. Journal of American Dental Association 36:8, Jan. 1948.
13. Best, C. H., and Taylor, N. B. The Physiological Basis of Medical Practice. 4th ed., Baltimore, Williams and Wilkins, 1945. 1169 p. (P416-422).
14. Ibid.
15. Best, C. H., and Taylor, N. B. The Living Body. New York, Holt, 1938. 538 p. (p.223-225).
16. Best, C. H., and Taylor, N. B. op. cit., p420.

17. Best, C. H., and Taylor, N. B. op. cit., p. 421.
18. Kocour, E. J., and Zaus, A. E. Salivary Excretion of Sulfanilimide and the Accompany Effect on Mouth Flora. Journal of Dental Research 19:319, June 1940.
19. Appleton, J. L. T. Bacterial Infection in Dental Practice. 4th. ed., Philadelphia, Lea and Febriger, 1950. 628 p. (p. 627).
20. Appleton, J. L. T. op. cit., p. 178-179.
21. Hutchinson, Jonathan. A Case of Dry Mouth. Transaction of the Clinical Society of London 21:180, 1888.
22. Sharp, H. S. A Case of Absence of Salivary Secretion. Journal of Laryngology and Otology 52:177-178, Mar. 1937.
23. Ramsey, W. R. A Case of Hereditary Congenital Absence of the Salivary Glands. American Journal of Diseases of Children 28:440, Oct. 1924.
24. Losch, P. K., and Weisberger, David. High Caries Susceptibility in Diminished Salivation. American Journal of Orthodontics and Oral Surgery (Oral Surgery Section), 26:1102-1104, Nov. 1940.
25. Faber, Mogens. A Case of Congenital Xerostomia. Acta Paediatrica 30:148-151, 1942-43.
26. Gurley, W. B. Unilateral Dental Caries: Report of a Case. Journal of American Dental Association 26:163-164, Jan. 1939.
27. Fasoli, G. A Case of Congenital Absence of Salivary and Lacrimal Secretion. Dental Cosmos 56:999, Aug. 1914.
28. Chamberlin, W. B., Xerostomia. Journal of the American Medical Association 95:470-472, Aug. 1930.
29. Prinz, Herman. Dental Materia Medica and Therapeutics. 6th. ed., St. Louis, Mosby, 1927. 632 p. (p. 290-294).
30. Shaw, J. H., and Weisberger, David. Carious Lesions in Cotton Rat Molars. II. Effect of Removal of Principal Salivary Glands. Proceedings of the Society for Experimental Biology and Medicine, 70:103, Jan. 1949.
31. Cheyne, V. D. Inhibition of Experimental Dental Caries by Fluorine in the Absence of Saliva. Proceedings of the Society for Experimental Biology and Medicine 43:58-61, Jan. 1940.
32. Weisberger, David, Truman, Nelson, and Boyle, P. E. The Development of Caries in the Teeth of Albino Rats Following Extirpation of the Salivary Glands. American Journal of Orthodontics and Oral Surgery 26:88, Jan. 1940.

33. Montgomery, Mary F. Effect on the Teeth of Total Absence of Salivary Secretion. Journal of the American Dental Association, 17:1338-1339, July 1930.
34. Brown, J. B., and Klotz, N. J. Attempts to Correlate Composition of Mixed Human Saliva with Rate of Secretion. Journal of Dental Research 14:435-438, Dec. 1934.
35. Trimble, H. C., Etherington, J. W., and Losch, P. K. Rate of Secretion of Saliva and Incidence of Dental Caries. Journal of Dental Research 17:299, Aug. 1938.
36. Cushman, F. H., Etherington, J. W., and Thompson, G. E. Quantative Relationship Between Saliva and Caries in an Adolescent Group. Journal of Dental Research 19:298, June 1940.
37. Becks, Herman, Wainwright, W. W., and Young, D. H. Does Salivary Calcium and Phosphorus Differ Significantly in Caries-Free and Caries-Active Individuals? Journal of Dental Research 20:171-188, June 1941.
38. Becks, Herman, and Wainwright, W.W. Rate of Flow of Resting Saliva of Healthy Individuals. Journal of Dental Research 22:391-396, Oct. 1943.
39. Karshan, Maxwell. Do Calcium and Phosphorus in Saliva Differ Significantly in Caries-Free and Active-Caries Groups? Journal of Dental Research 21:83-86, Feb. 1942.
40. Burrill, D. Y., and Fosdick, L. S. The Buffering Capacity and Rate of Flow of Saliva in Relation to Dental Caries. Northwestern University Dental School Research Bulletin 44:6-8, Mar. 1944.
41. Barany, Franz. Resistance to Caries in Relation to Certain Properties of Saliva. Acta Medica Scandinavia 127:370-375, Dec. 1947.
42. Miller, W. D. New Theories Concerning Decay of Teeth. Dental Cosmos 47:1293-1301, Nov. 1905.
43. Pickerill, H. P. The Prevention of Dental Caries and Oral Sepsis. 3rd. ed., New York, Holber Inc., 1924 340 p. (p.199).
44. Zaus, E. A., and Hanson, H. L. The Saliva. p.170-178. (In Black, G.V., ed. Operative Dentistry. Vol. 1, 7th ed. Chicago, Medico-Dental, 1936 422 p.).
45. Rae, J. J., and Clegg, C. T. The Relation between Buffering Capacity, Viscosity and Lactobacillus Count of Saliva. Journal of Dental Research 28:589-593, Dec. 1949.
46. Klein, Henry, Palmer, C. E., and Knutson, J. W. Dental Status and Dental Needs of Elementary School Children. Public Health Reports 53:751-765, May 1938.

47. Becks, Herman, and Wainwright, W. W. The Effect of Flavored Activators on Salivary Flow and Calcium and Phosphorus Composition. Journal of Dental Research 21:87-97, Feb. 1942.
48. Wainwright, W. W. Human Saliva: A Study of Rate of Flow of Activated Saliva. Journal of Dental Research 18:441-446, Oct. 1939.
49. Becks, Herman, and Wainwright, W. W. Human Saliva: The Effect of Activation of Salivary Flow. Journal of Dental Research 18:447-455, Oct. 1939.
50. Gregerseu, M. I. A Method for Uniform Stimulation of the Salivary Glands in the Unanesthetized Dog by Exposure to a Warm Environment With Some Observations on the Quantative Changes in Salivary Flow During Dehydration. American Journal of Physiology 96:107-116, April 1931.
51. Wainwright, W. W., and Becks, Herman. op. cit., p. 285-291.
52. Findlay, A. Practical Physical Chemistry. 5th. ed., London, Green., 1933. 307 p. (p.73-76).