Epidemiologic Studies in Dental Caries, III: The Interpretation of Clinical Data Relating to Caries Advance*†

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TWENTY-FIVE years ago we first observed that children with diabetes mellitus tended to have less advance of tooth decay than does the usual child.[1] Further observations indicated that such freedom from advancing decay was conditioned by the degree in which the child's food intake approached accepted nutritional standards, and in which his diabetic anomaly was controlled through the use of insulin.[2] Within two months after the routine of diet and insulin management had been established, open cavities would become stony hard at their base, and no new cavities would develop. Among diabetic children who failed to observe the prescribed regimen, caries tended to appear and to advance as rapidly or more rapidly than in the average child. The serial dental data for a group of diabetic girls are summarized in the first chart (Figure 1). Each horizontal bar represents the dental course of one child for successive years; the zones shaded black represent periods when no new caries could be detected. It will be noted that most of the periods of observation were characterized by non-advance of caries, and that the total increment of new caries was very low.[3]

To serve as an index of the commonness of such findings among non-diabetic children, we reviewed the dental records of 100 school children who had been cared for at the university dental infirmary for not less than three consecutive years. The graphic record of the 40 girls from this series is shown in the next chart (Figure 2). It will be noted that most subjects had some periods of non-advance of caries, but that caries progression was the rule and that for most of them the caries increment was notable from one year to the next.[4]

Influenced by the published work of Beust[5] and of Bodecker,[6] we had interpreted the hardening at the bases of the open cavities as evidence of deposition of sclerotic dentin. Occasional histologic studies of teeth which clinically had developed arrest of caries showed that the dentin lying between the enamel defect and the pulp had become dense and stain-resistant. Naturally shed, deciduous teeth of children who had been under diabetic management for long periods before exfoliation showed dentin more completely sclerosed than is customary in children's teeth: more nearly analogous to the sclerosed dentin of the permanent teeth of adults. We believed that caries had become arrested in the diabetic patient's teeth through the effect of homeostatic mechanisms working from within the tooth. This belief has led to the premise that the body

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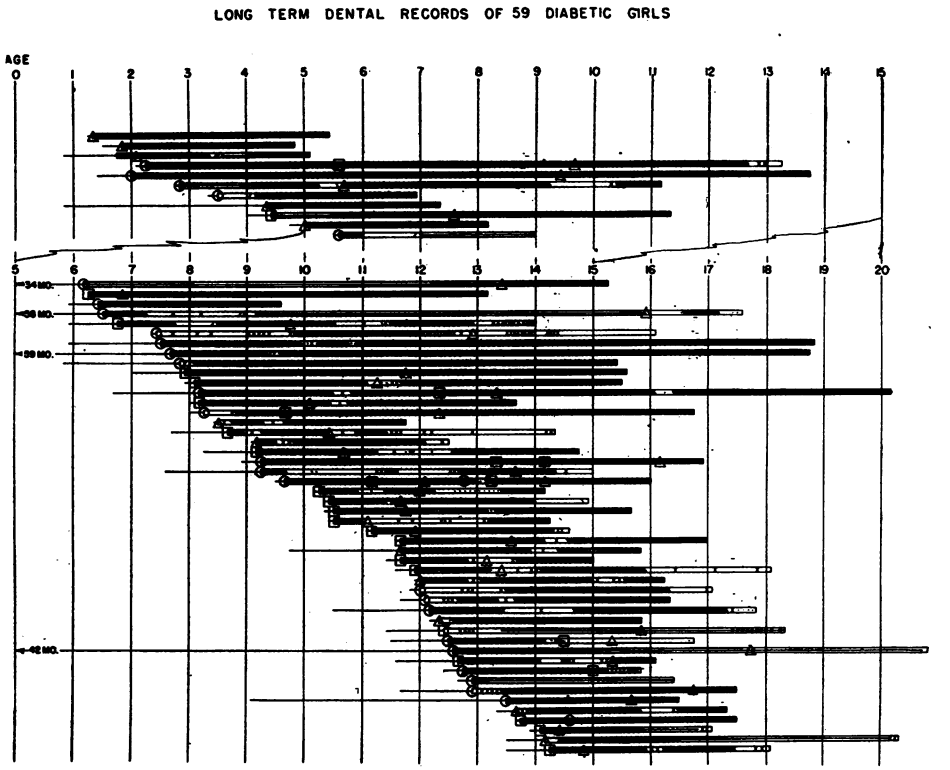

FIGURE 1—Individual records of progressive dental status of 59 girls with diabetes mellitus. Length of each bar indicates duration of intermittent dental observation; solid black areas represent periods of freedom from new dental lesions; unshaded areas contain symbols to indicate number of tooth surfaces with new lesions which appeared during the corresponding period. Symbols have meanings as follows: diagonal line, one dmf (deciduous) tooth surface; crossed diagonal lines, one DMF (permanent) surface; dot, one surface with a precarious lesion apparently limited to the enamel (preponderantly pits and fissures, developmental in origin, present when tooth first erupted). Intervals of prolonged non-observation are indicated by a horizontal line through the bar. The duration of diabetes mellitus prior to the initial dental examination is indicated by the length of the single horizontal line to the left of each bar graph. (DMF signifies decayed, missing or filled). (Taken from reference 3.)

possesses natural defense mechanisms which can prevent the development or the advance of tooth decay if proper metabolic conditions can be maintained.

This concept of natural defense mechanisms for the tooth has been difficult to reconcile with the classic expression of the chemico-parasitic theory of caries causation. Most individuals holding to the regional concept of caries causation have assumed that the erupted tooth is an inert structure, incapable of change of composition through interchange with the internal fluids of the body. Proponents of the chemico-parasitic theory explained our findings in diabetic children's teeth as dependent on the prohibition of sugar from diabetic diets. We had observed numerous instances where dental caries came to arrest among children who were not diabetic and who had continued to ingest sugar, but our data were not sufficient in numbers nor were the variable factors controlled sufficiently to give statistical significance to these findings.

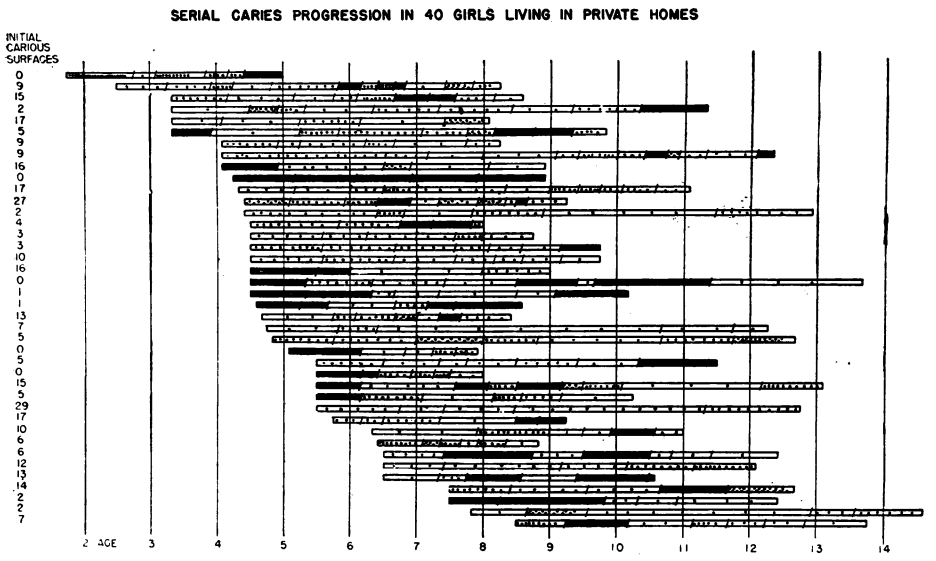

FIGURE 2—Diagrammatic representation of the serial dental status of 40 girls. Each horizontal bar portrays one subject's record. The position and length of the bar indicate age and the duration of observation; oblique lines indicate interim dental examinations; black areas indicate periods during which no newly carious areas were detected. The number of dots within unshaded segments of a bar indicate the number of newly carious surfaces detected at the close of the corresponding interval. The number at the left of each bar indicates the total number of DMF surfaces in the mixed dentition at the time of the first dental examination. (Taken from reference 4.)

There was need for controlled observations to establish the spontaneous variability of caries progression that one might expect to encounter in the general child population.

Clinical studies of dental caries progression seldom have followed the serial course of tooth decay for the individual child for long periods of time, as we had done with the diabetic children and with those drawn from the dental clinic which I have cited. Most published estimates as to the rates of advance of caries have been based instead on cross-sectional observations for a given child population, with estimation of caries advance rates for the group as a whole through differences of values for respective age levels. Such studies serve a valuable purpose as they relate to the general population, but are of little assistance in indicating what one may expect to observe in the individual child, either as to the likelihood of variability from the estimated value or as to the regularity of progression of caries. For that reason they are of no particular value as bases for comparison with studies similar to the ones we have performed. Moreover, most reported studies have dealt with unregimented or poorly regimented populations, without attempt to appraise or to control the many agents which may affect the individual rates of caries advance.

We have recognized the need for a well controlled, long-term, individualized study of caries progression in children's teeth under conditions which would permit control of as many variables as possible. We had two objectives in mind for such a study: One was to learn more about the constancy of the long-term pattern of caries advance in the mouth of the individual child under standard conditions. The other was to compare

the rate and pattern of caries advance among children whose sugar intake was manipulated widely, with the corresponding pattern before and after the diet regimentation had been in effect, as well as with the rate and pattern observed concurrently in a group of control subjects.

When opportunity permitted, we undertook such a study. To meet the exacting requirements as fully as possible, it was necessary to use subjects already living under conditions of limited regimentation. The most appropriate group for our purpose was that to be found in one of our state schools for the mentally retarded. Out of a population of about 2,000 persons of all ages, we found 250 boys and girls between the ages of 13 and 20 years. This age group seemed best for study because it avoided the consideration of deciduous teeth. We would have preferred greater uniformity of age, but we needed more subjects than could be provided with a narrower age range. Not many more than 200 proved to have capacities for adequate coöperation in our study. The experimental subjects were chosen from the girls; the remaining girls and the boys served as separate control groups. This report will be limited primarily to consideration of the experimental and control groups of girl subjects (Table 1).

During midsummer of 1946 the initial dental examinations were made. Similar examinations have been made several times each year since then, and they still are in progress. As a corollary of the dental examinations, estimations of Lactobacillus frequency in saliva have been made recurrently for most of the subjects. During the first two years of the study we were able to establish rates for the spontaneous advance of caries for each subject while all phases of institutional life still remained unmodified.

During the second year, about 70 of the girls were chosen from the group as a whole to serve as a pool from which the experimental personnel would be drawn. Graduate dietitians appraised the diet practices at the institution and studied means for bringing the quality of food intake for the experimental group up to the National Research Council's recommendations. Two sets of menus were prepared, relatively constant as to content of protein, minerals, vitamins, and caloric equivalence, but so designed that one set avoided the use of any refined sugar or its products, while the other provided not less than three ounces of sucrose daily, usually as a constituent of foods, sometimes as candy which would be eaten at the table with the meal. Foods not available through the institution were purchased or else were furnished through the courtesy of various agencies.* Further details relating to the routine of management have been published.[7]

The diets devoid of refined sugar were

, TABLE 1

Initial Status of Girls Who Comprised the Experimental and Control Groups, and Number of Dental Examinations Each Year of Study

	Experimental Group	*Control Group*
Number of subjects	64	52
Age when first examined (months): 1st quartile	153	148
Median	178	190
3rd quartile	193	208
Initial number of DMF tooth surfaces: Mean	7.6	8.3
S.D.	4.3	6.1
Filled tooth surfaces: % of total number of carious surfaces	22%	19%
Incidence of poor dental hygiene	25%	40%
Number of dental examinations made during each of the four years of the study: 1946–1947	129	197
1947–1948	173	176
1948–1949	254	131
1949–1950	118	60

* We are indebted to the Pineapple Research Institution of Hawaii, to the Iowa Dairy Industry Commission, and to the Vitex Corporation for the provision of pineapple juice, dried skim milk, and Vitamin D concentrate, respectively.

TABLE 2

A—The Pattern of Caries Increment among Both Male and Female Subjects during the First Two Years of the Study, When None of the Subjects Were under Diet Regimentation

	Mean	Quartile Distribution			S.D.	S.E. Mean
		1st	Median	3rd		
Number of DMF surfaces when first examined	8.4	4	7	11		
New DMF surfaces noted during 2-year period	5.13	3	5	7	3.74	.275
Duration of freedom from newly carious surfaces (months): longest consecutive period	9.7	6	7	15		
Aggregate duration	11.3	6	11	17		

B—Contrast of the Duration of Freedom from New Caries among the One-fourth of the Whole Group Who Had the Lowest Net Rates of Caries Advance during the Two-year Period with the One-fourth Whose Net Rates Were the Highest

	Consecutive Duration of Freedom from Newly Carious Tooth Surfaces (months)				
	Mean	1st Quartile	Median	3rd Quartile	Maximum Value
Range of values for net caries progression rates:					
lowest one-fourth	13.4	6	12	18	24
highest one-fourth	6.8	5	6	7	18

inaugurated in May of 1948 and remained in effect until December. Because of the holiday season, diet regimentation was relaxed during December. On January 1, 1949, the high sugar regimen went into effect and was maintained for the succeeding six months. Sixty-four of the girls from the experimental group served as subjects for diet regimentation for the 12 month period. During that time the remainder of the girls and all the boys continued to follow the usual institutional diet regimen, with no special emphasis on the amount of sugar consumed. Since July of 1949, all subjects have been merged again into the usual pattern of institutional life and routine.

RESULTS

This report is based on the data collected during the first four years of study of this institutionalized population. It deals with the variability of rates of caries progression under spontaneous conditions of life and of oral environment, and with the observed effect of widely different levels of intake of refined sugar on rates of caries progression. It does not take into account any possible role which native sugars from vegetables or fruits might have on the teeth. Five observations from our study will be considered, as follows:

1. Almost without exception, new cavities tended to appear in an intermittent manner in the mouth of the individual child, even though the conditions of life and of diet remained similar. Long periods of freedom from new cavities were the rule rather than the exception. Basing our observations on the dental course of 212 boys and girls throughout the two first years of the study, when no regimentation was in force, 90 per cent of the subjects had at least 3 consecutive months of freedom from new cavities; 40 per cent had at least 10 consecutive months of such freedom. Even among the 25 per cent of the subjects whose scores for new caries lesions were highest, 11 months or longer of freedom from new lesions was not infrequent (Table 2). From this observation, as well as from earlier studies, we believe that the clinical progression of tooth decay usually is an inconstant process and that the isolated observation of non-progression of caries cannot be accepted as evidence of increased caries resistance.[8]
2. During each of the four years of observation, there was little constancy from one subject to another as to the number of new carious lesions. The scatter of values was notable both among the experimental and the control members. The coefficient of variability seldom was less than 60 per cent for any set of values. The average value, when con-

TABLE 3

Average Number of Newly Affected Tooth Surfaces During Each of Four Successive Years of Observation of the Girls from the Experimental and the Control Groups

Year of Study	Nature of Diet Control		Number of New DMF Tooth Surfaces Per Annum					
			Experimental Group			Control Group		
	Experimental Group	Control Group	Mean	S.D.	S.E. Mean	Mean	S.D.	S.E. Mean
A—Beginning with Midsummer, 1946:								
First	Unmodified	Unmodified	2.82	2.46	0.43	3.0	1.8	0.39
Second	Unmodified	Unmodified	3.02	2.30	0.346	3.12	2.1	0.446
Third	No refined sugar for 6 months; then 3 oz. sucrose daily for the next 6 months	Unmodified	4.9	1.03	0.134	5.62	4.90	0.83
Fourth	Unmodified	Unmodified	3.56	2.54	0.352	2.39	2.44	0.508
B—Beginning with Midwinter, 1946:								
First	Unmodified	Unmodified	2.6	2.32	0.34	3.5	1.6	0.34
Second	First 6 months unmodified; then no refined sugar for the next 6 months	Unmodified	5.2	4.1	0.55	5.6	3.2	0.70
Third	3 ounces sucrose daily for 6 months; unmodified diet for the next 6 months	Unmodified	3.2	2.26	0.31	4.0	4.35	1.06

TABLE 4

Analysis of Variability of Rates of Caries Progression for Experimental and for Control Groups for the Same Year *

A—For the Four Successive Years Beginning With Midsummer of 1946:

	Mean Value, New DMF TS	Difference of Means	S.E., Diff. of Means	Critical Ratio
First year				
Experimental group	2.82			
Control group	3.0	— 0.18	0.58	0.31
Second year				
Experimental group	3.02			
Control group	3.12	— 0.10	0.565	0.178
Third year				
Experimental group	4.9			
Control group	5.62	— 0.72	0.93	0.775
Fourth year				
Experimental group	3.56			
Control group	2.39	+ 1.17	0.62	1.88
B—For the Three Successive Years Beginning With Midwinter, 1946:				
First year				
Experimental group	2.6			
Control group	3.5	— 0 9	0.48	1.87
Second year				
Experimental group	5.2			
Control group	5.6	— 0.4	0.89	0.45
Third year				
Experimental group	3.2			
Control group	4.0	— 0 8	1.43	0.56

* Based on data from Table 3.

TABLE 5

Analysis of Variability of Rates of Caries Progression for the Same Group of Subjects During Successive Years *

A—For the Four Successive Years Beginning With Midsummer, 1946:

	Contrasted Years:				
	1:2	*1:3*	*1:4*	*2:3*	*2:4*
Experimental group:					
Difference of means	0.20	2.08	0.74	1.88	0.54
Standard error, difference of means	0.55	0.605	0.555	0.548	0.494
Critical ratio	0.364	3.44	1.33	3.43	1.09
Control group:					
Difference of means	0.12	2.62	0.61	2.50	0.73
Standard error, difference of means	0.91	1.1	0.95	1.17	0.98
Critical ratio	0.12	2.38	0.64	2.14	0.745

B—For the Three Successive Years Beginning With Midwinter, 1946:

	Contrasted Years		
	1:2	*1:3*	*2:3*
Experimental group:			
Difference of means	2.6	0.6	2.0
Standard error, difference of means	0.65	1.04	0.633
Critical ratio	4.0	0.58	3.15
Control group:			
Difference of means	2.1	0.5	1.6
Standard error, difference of means	0.79	1.236	1.27
Critical ratio	2.66	0.49	1.26

* Based on data from Table 3.

sidered together with the standard deviation, served to indicate the course of the entire group but was of little value for predicting the course of any individual subject (Table 3).

3. Using the average number of newly carious surfaces within each group for each year as a criterion, there was great variability from one year to another, both for the experimental and for the control groups. For a given year, the type and degree of oscillation of the average value tended to be parallel for the experimental and the control groups. Even among the subjects who were under no manner of imposed regimentation, and who seemed to be living under comparable conditions throughout the four years of the study, the differences of the means from one year to another presented significant differences (Table 4).
4. The differences between the experimental subjects and the control group during the periods of contrasted diet tended to be smaller than those between either of the groups' values for successive years of the study. There was no significant difference as to the average rate of caries advance between the experimental and the control group, either for the year of sugar manipulation or for the next year. Moreover, when the 12 month period of sugar manipulation was redivided so that the 6 months of sugar abstention were included with the 6 months of unmodified diet which preceded them, and similarly the 6 months of high sugar ingestion were included with the next 6 months of unmodified diet, still there was no significant difference between the mean values. The pattern of frequency distribution of individual values within the two groups also was similar (Table 5).
5. We have not been able to demonstrate any significant relationship between the number of Lactobacilli in the saliva of our subjects and the respective rate of caries advance during the year which preceded, coincided with, or followed the collection of the saliva samples. No significant difference was evident when the average values for new caries among those with zero counts were compared with those with counts exceeding 30,000 Lactobacilli per ml. of saliva.[9]

DISCUSSION

Many fundamental questions relating to dental caries as a human disease remain unanswered. Answers often have been taken for granted, even among students of dental caries, and the errors of interpretation which have resulted

have deprived various critical studies on caries progression of the meaning which they might otherwise have afforded. Further carefully controlled studies will be needed to establish answers to these basic questions, and it is important that such studies be so designed that as many variables as possible be avoided, or their potential effect on the outcome properly weighted. If not, the summated effect of the uncontrolled variables may well outweigh the differences ascribed to the agent being investigated.

One important question calling for answer is the length of time which must elapse before an agent being investigated can lead to clinically recognizable caries. Another equally important question deals with the course of the carious lesion once initiated: Is its progress inexorable? Is it steady? Under what circumstances can it be brought to arrest, and how can the observer recognize such arrest?

These recent studies of ours illustrate the variability of caries progression from individual to individual and the variability in the mouth of a given subject from month to month under supposedly standard conditions. Many studies previously reported have ignored the likelihood of latency of response of teeth to change; have assumed constancy of caries progression; have failed to establish patterns of caries advance for members of their study groups prior to the regimentation of their experimental subjects; and have depended on previously determined rates of caries advance as control values for the current studies. Our observations indicate that such techniques can lead to false interpretations of the efficacy of any regimen being studied.

Consideration also should be given to the initial state of the teeth before experimental conditions have been imposed. If one is measuring caries advance in terms of the rate of appearance of newly carious areas, then opportunities for advance are limited by the number of teeth or tooth surfaces which initially are free from caries attack at the outset of the study. Likewise one may expect a difference in results on the basis of the number of susceptible tooth surfaces which have been filled prior to the start of the study.

Even more important as potential sources of experimental error are the nature and amount of reparative dental services which the patient receives while the study is in progress. The placement of fillings is itself a technique employed to lessen caries progression. Consequently we should not compare caries progression rates among children whose patterns of initial or concurrent dental repair differed significantly. The authors have been impressed by the general lack of consideration of these important factors by experimenters in their choice of samples for the study of caries advance. Our own subjects had received only minimal amounts of dental repair prior to the beginning of our study, and during its course only emergency reparative services were given. Cavities initially present remained untreated and unfilled throughout the course of the study unless they caused the child manifest discomfort.

Even criteria for the diagnosis of dental caries are inconstant. There may be widespread disagreement among examining dentists as to whether or not a specific dental lesion is carious. Serial examinations of the same tooth, even though made by the same examiner at different times, may result in different interpretations from one examination to another as to whether a break in the continuity of the enamel represents the early stage of a cavity or whether it is a developmental defect. When a series of examiners is used and each employs his own criterion of judgment and technique of examination, the results for the total caries score for a given mouth may

differ by as much as 100 per cent.[10] In serial examinations, cavities once described may disappear from the record, due to a change in interpretation.

Such statistical dilemmas have led us to adopt the serial consideration of each tooth surface as a measure of caries advance, rather than to place dependence on the serial total values for affected tooth surfaces for a given mouth. We take into consideration also the extent of the described dental lesion. Most examiners make no distinction in their reports between an incipient cavity and one which has become unmistakable. As a consequence, the total caries score may vary widely through errors of judgment possibly greater in degree than the difference which the examiner ascribes to some agent he has been testing for its power to affect caries progression.

Thus there are many potential sources of error in clinical studies relating to rates of caries advance. The summation of such errors can lead to manifest mistakes of interpretation. It becomes necessary to choose techniques of sampling and of control, of examination and of record, which are as stringent as the objective of the study requires if data of value are to be obtained. Consideration of affected tooth surfaces or of individual caries lesions is more reliable as an estimate of caries progression than is the limitation of attention to the number of affected teeth.

Our own data permit us to distinguish caries progression on the basis of the size, depth, location, and consistency of each carious area listed in our records. The serial study of specific teeth will eliminate many errors which otherwise might arise from the faulty interpretation of individual caries lesions. When it is possible to express results clearly and individually for respective teeth, this seems a better statistical approach than to lose identity of teeth or even of subjects through the merging of data in terms of group averages.

SUMMARY AND CONCLUSIONS

Time does not permit any further exploration of sources of potential errors which are inherent in dental data. We have tried to illustrate the errors which may be concealed in dental statistics and to lead the reader to question the results of reported dental studies until the validity of the experimental conditions, as well as of the conclusions, has been established. In view of the fact that spontaneous variability from one child to another is great, and that such variability seldom is given consideration in clinical studies of caries progression, there is reason to question those reports which have attributed beneficial or harmful effects on the teeth to the use of any specific agency.

Within specific limitations of investigation, we have been unable to demonstrate differences in the caries advance rates among adolescent children when the ingestion of refined sugar was arbitrarily prohibited or was given daily in large amounts. However we wish to draw attention to these limits of our observations, and request that no inferences be drawn which lie beyond the conditions of our study. These conditions are as follows:

a. The study dealt with altered intake of refined sugar and its products only; during both periods of diet control our subjects received liberal amounts of the sugars which are native constituents of vegetables and fruits.
b. During the period of high sugar ingestion, the sugar was given only with meals, and usually as a constituent of prepared foods.
c. The girls receiving the diets of altered sugar content were under a regimen which was designed to provide a complete diet, both quantitatively and qualitatively. The caloric allowance was constant during the periods of contrasted sucrose intake.
d. Lactobacillus counts proved to offer no reliable index of the likelihood or the rates of caries progression among our subjects, either among those under diet regimentation or among the members of the control groups.

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White House Conference Continues

A committee to carry out the objectives of the Mid-Century White House Conference on Children and Youth has been formed. Called the National Mid-Century Committee for Children and Youth, its chairman is Leonard W. Mayo, director of the Society for the Aid of Crippled Children, who was also chairman of the executive committee of the recent White House Conference.

The committee will base its program on the purpose of the White House Conference: ". . . to consider how we can develop in children the mental, emotional, and spiritual qualities essential to individual happiness and to responsible citizenship." It believes a top priority to be the conservation and strengthening of family life amid the stresses of a defense economy.

The National Committee will offer field service to state committees and national organizations with a continuing program, to achieve Conference objectives. It will maintain an information service, and will shortly consider what its research program should be in relation to the many gaps in knowledge about how children develop healthy personalities. Beginning in September, it will publish a bulletin regularly.

Elma Phillipson, who was the White House Conference consultant on national organizations, is executive secretary of the new committee, Lois Gratz is its staff field consultant, and Benjamin Goldenberg the information consultant. Offices are at 4th and Independence Ave., S.W., Washington 25, D. C.