

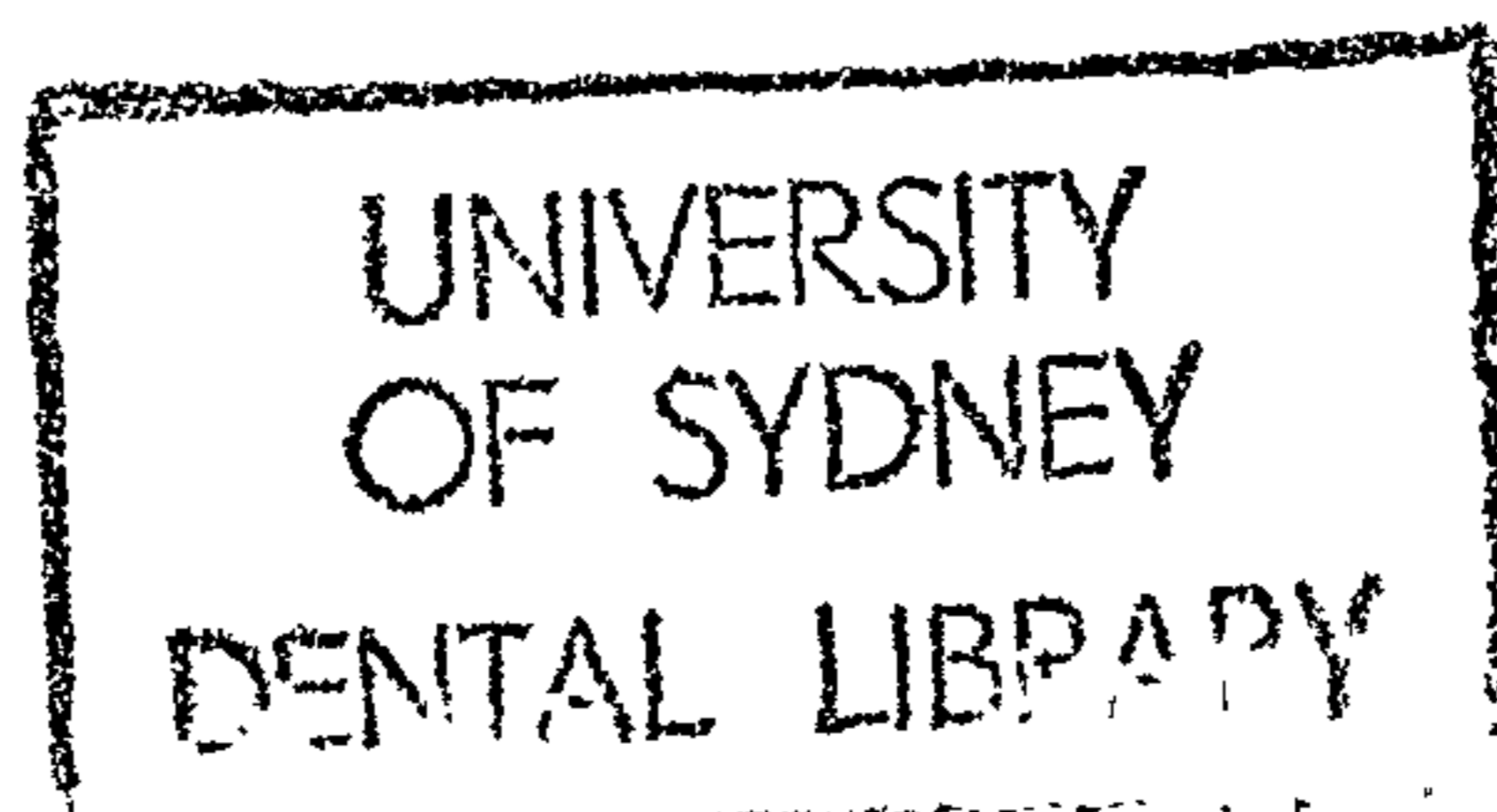
SOME ASPECTS OF ORAL BIOLOGY WITH
PARTICULAR REFERENCE TO THE PRESERVATION OF
THE DENTAL TISSUES

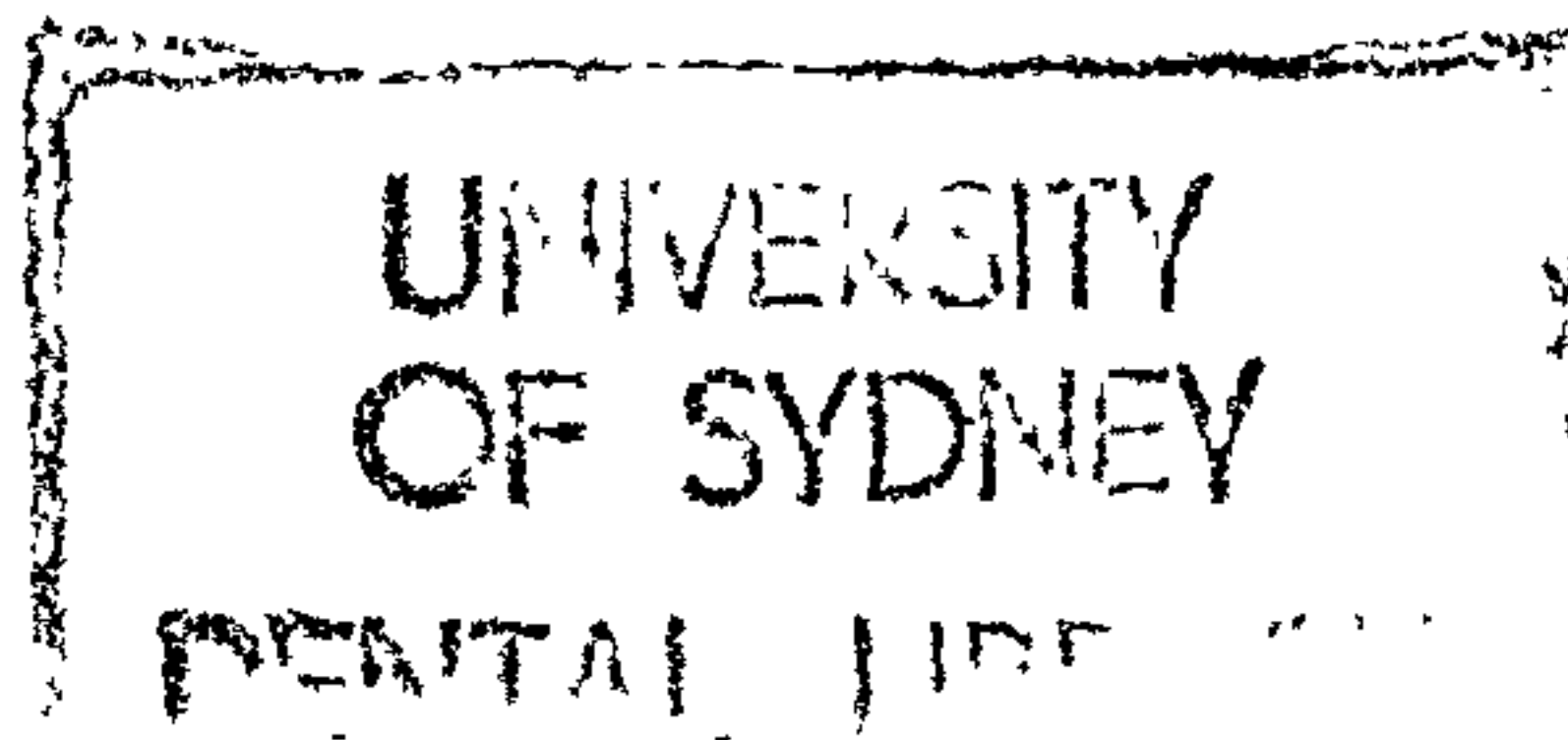
A collection of published works
submitted to the Faculty of Dentistry
of the University of Sydney for the
Degree of Doctor of Dental Science

ROBERT HARRIS, M.B.E., M.D.S.

Formerly, Director, Institute of Dental Research
Sydney

Sydney. May, 1976





List of Contents

Acknowledgements	i-ii
Index of publications	iii-x
Author's statement	xi-xii
Introduction and theme of published work	1-4
Comments on papers:	
Part 1. -- Dental caries	5-24
Part 2. -- Dental pulp	25-30
Part 3. -- Human periodontium	31-34
Part 4. -- Supplementary studies	35-37

Acknowledgements

I wish to record my gratitude to the late Dr. N. E. Goldsworthy and the late Dr. H. L. Sullivan, Director and Assistant Director of the Institute of Dental Research, Sydney, who invited me to participate in the Hopewood House, Bowral, N.S.W., studies. This opportunity provided access to clinical material for long term studies on dental caries. The observations and papers arising from these studies were of inestimable value in subsequent investigations on the efficacy of topical fluoride solutions and of a food additive -- calcium sucrose phosphate calcium orthophosphate complex -- in the reduction of dental caries in children.

My appreciation is gratefully extended to Professor K. W. Cleland, Department of Histology and Embryology, University of Sydney, who made it possible for me to work with Associate Professor C. J. Griffin in the Department and whose generous support enabled the studies on the ultrastructure

of the human dental pulp and periodontal tissues to be undertaken.

Sincere appreciation is also recorded for the cooperation and the effort made by Professor Griffin in our conjoint work.

A special note of appreciation is extended to Christa Lossin for her technical assistance in the preparation and photographing of material for histological examination and in assisting with the surgical procedures on the laboratory animals and their subsequent care and maintenance.

There are a number of other individuals -- members of the staffs of the Institute of Dental Research and of the CSR Research Laboratories, Roseville, who in many ways ensured that the various projects with which I was directly involved were brought to fruition. Special acknowledgement has been made in the various publications to individuals but particular mention should be made to the late Gladys Carey, M.Sc., to Judith Ritter and to Audrey Barnes for their labours in producing the typewritten drafts of the manuscripts of all the publications.

Index of publications

Part I

1. R. Harris: Developmental opaque areas and hypoplasia of teeth. Dent. Jnl. Aust., 24:4, 131-134 (Aug.) 1954.
2. H. R. Sullivan, and R. Harris: The biology of the children of Hopewood House, Bowral, N.S.W. II. Observations extending over five years, 1952-1956. 2. Observations on oral conditions. Aust. dent. Jnl., 3:5, 311-317 (Oct.) 1958.
3. R. Harris: The biology of the children of Hopewood House, Bowral, N.S.W. IV. Observations on dental caries experience extending over five years, 1957-61. Jour. dent. Res., 42:6, 1387-1399 (Nov.-Dec.) 1963.
4. R. Harris: The biology of the children of Hopewood House, Bowral. V. Observations on dental caries experience: proximal lesions. Aust. dent. Jnl., 8:6, 521-528 (Dec.) 1963.

5. R. Harris: The biology of the children of Hopewood House, Bowral. VI. The pattern of dental caries experience. *Aust. dent. Jnl.*, 12:3, 220-227 (June) 1967.
6. R. Harris, and H. R. Sullivan: The biology of the children of Hopewood House, Bowral, N.S.W. III. The eruption pattern of certain permanent teeth. *Aust. dent. Jnl.*, 5:2, 78-83 (Apr.) 1960.
7. Kalyani Amatayakul, M. R. Nibhasara Ladavalya, and R. Harris: A dental survey of a small group of Thai children. *Amer. Jnl. Clin. Nutrit.*, 8:2, 240-246 (Mch.-Apr.) 1960.
8. R. Harris, R. G. Schamschula, G. Gregory, Miriam Roots, and J. Beveridge: Observations on the cariostatic effect of calcium sucrose phosphate in a group of children aged 5-17 years. Preliminary report. *Aust. dent. Jnl.*, 12:2, 105-113 (Apr.) 1967.
9. R. Harris, R. G. Schamschula, J. Beveridge, and G. Gregory: The cariostatic effect of calcium sucrose phosphate in a group of children aged 5-17 years. *Aust. dent. Jnl.*, 13:1, 32-39 (Feb.) 1968.

10. R. Harris, R. G. Schamschula, J. Beveridge, and G. Gregory: The cariostatic effect of calcium sucrose phosphate in a group of children aged 5-17 years. Part IV. Aust. dent. Jnl., 14:1, 42-49 (Feb.) 1969.
11. R. Harris, Miriam Roots, G. Gregory, and J. Beveridge: Calcium sucrose phosphate as a cariostatic agent in children aged 5-17 years. Part III. Dietary factors. Aust. dent. Jnl., 13:5, 345-352 (Oct.) 1968.
12. R. Harris: Observations on the effect of topical sodium fluoride on caries incidence in children. Aust. dent. Jnl., 4:4, 257-260 (Aug.) 1959.
13. R. Harris: Observations on the effect of eight per cent stannous fluoride on dental caries in children. Aust. dent. Jnl., 8:4, 335-340 (Aug.) 1963.

Part 2

14. R. Harris, and C. J. Griffin: Aldehyde fuchsin-Halmi reaction in fibres of the human dental pulp. Arch. oral Biol., 11: 649-658, 1966.
15. C. J. Griffin, and R. Harris: Ultrastructure of collagen fibrils and fibroblasts of the developing human dental pulp. Arch. oral Biol., 11: 659-666, 1966.
16. R. Harris, and C. J. Griffin: Histogenesis of fibroblasts in the human dental pulp. Arch. oral Biol., 12: 459-468, 1967.
17. R. Harris, and C. J. Griffin: Fine structure of nerve endings in the human dental pulp. Arch. oral Biol., 13: 773-778, 1968.
18. R. Harris, and C. J. Griffin: The fine structure of the mature odontoblasts and cell rich zone of the human dental pulp. Aust. dent. Jnl., 14:3, 168-177 (June) 1969.
19. R. Harris, and C. J. Griffin: The ultrastructure of small blood vessels of the normal human

dental pulp. Aust. dent. Jnl., 16:4, 220-226
(Aug.) 1971.

20. R. Harris, and A. W. Bull: The healing of the traumatized dental pulp following injury. Aust. dent. Jnl., 11:4, 236-247 (Aug.) 1966.
21. R. Harris: Further observations on pulp reactions to a tetracycline compound. Aust. dent. Jnl., 13:1, 82-86 (Feb.) 1968.
22. C. J. Griffin, and R. Harris: The ultrastructure of the blood vessels of the human dental pulp following injury. I. Aust. dent. Jnl., 17:4, 303-308 (Aug.) 1972.
23. R. Harris, and C. J. Griffin: The ultrastructure of the blood vessels of the human dental pulp following injury. II. Aust. dent. Jnl., 17:5, 355-362 (Oct.) 1972.
24. C. J. Griffin, and R. Harris: The ultrastructure of the blood vessels of the human dental pulp following injury. III. Aust. dent. Jnl., 17:6, 441-447 (Dec.) 1972.
25. R. Harris, and C. J. Griffin: The ultrastructure of the blood vessels of the human dental pulp following injury. IV. Aust. dent. Jnl., 18:2, 88-96 (Apr.) 1973.

Part 3

26. C. J. Griffin, and R. Harris: The fine structure of the developing human periodontium. Arch. oral Biol., 12:8, 971-982, 1967.
27. R. Harris, and C. J. Griffin: The protein polysaccharide complex of the developing human periodontium. Arch. oral Biol., 12: 1107-1114, 1967.
28. C. J. Griffin, and R. Harris: Unmyelinated nerve endings in the periodontal membrane of human teeth. Arch. oral Biol., 13: 1207-1212, 1962.
29. C. J. Griffin, and R. Harris: Innervation of human periodontium. I. Classification of periodontal receptors. Aust. dent. Jnl., 19:1, 51-56 (Feb.) 1974.
30. R. Harris, and C. J. Griffin: Innervation of the human periodontium. II. Fine structure of simple mechanoreceptors. Aust. dent. Jnl., 19:3, 174-181 (June) 1974.
31. C. J. Griffin, and R. Harris: Innervation of the human periodontium. III. Fine structure

- of compound receptors. Aust. dent. Jnl.,
19:4, 255-260 (Aug.) 1974.
32. R. Harris, and C. J. Griffin: Innervation of the
human periodontium. IV. Fine structure of
the complex mechanoreceptors and free nerve
endings. Aust. den. Jnl., 19:5, 326-331
(Oct.) 1974.
33. R. Harris: Innervation of the human periodontium.
In, The temporomandibular joint syndrome.
Edit. C. J. Griffin and R. Harris. Monographs
in Oral Science, No. 4. Basel, S. Karger,
1975 (pp. 27-44).
34. R. Harris: Some observations on periodontal
tissues in rats. Aust. dent. Jnl., 16:6,
382-388 (Dec.) 1971.
35. R. Harris and C. J. Griffin: Glomus tumour of
the periodontal tissues. Aust. dent. Jnl.,
10:1, 33-37 (Feb.) 1965.

Part 4

36. R. Harris: Implantation of chrome cobalt alloy tooth forms in the rabbits' mandible. Aust. dent. Jnl., 14:6, 396-406 (Dec.) 1969.
37. R. Harris, and Christa Lossin: The use of cobalt chromium and titanium endosseous dental implants. Aust. dent. Jnl., 16:2, 94-108 (Apr.) 1971.
38. R. Harris: The reaction of the oral tissues to implants. Aust. dent. Jnl., 18:3, 143-151 (June) 1973.

Note: The papers are not presented in strict chronological order of publication but rather according to subject matter. Wherever possible the material is in reprint form. However, papers No. 1, 7, and 33 have been reproduced by a photo-lithographic offset printing process.

Author's statement

The following statement is made in accordance with Clause 32 (1), (2), and (3) of the By-Laws of the University of Sydney governing the award of the Degree of Doctor of Dental Science.

1. The submitted publications are the work of the listed author or authors. The information contained therein is a direct result of original research by the listed author or authors unless otherwise indicated by appropriate reference in the text.

2. Papers 2, 3, and 6 are under the joint authorship with Dr. Harold Sullivan. Paper 7 was initiated by, and the laboratory investigations directed by me. The clinical study could not have been undertaken without the aid of Dr. Ladavalya who acted as interpreter and Dr. Amatayakul performed the technical work.

Papers 8-11 derived from a conjoint medical and dental study. The dental programme was initiated, devised, and controlled by me; the clinical

observations were made by R. G. Schamschula; G. Gregory developed the statistical parameters in consultation, and Professor J. Beveridge was responsible for the brief note on the medical studies. The four papers were written by me.

Papers 14-19, 22-25, 26-32, 35 are joint authorship with Professor C. J. Griffin. The responsibility for writing is reflected in the alternation of the first named author in each case.

The material for these investigations unless otherwise indicated was obtained from patients referred to me at the Institute of Dental Research. The electron microscopy was carried out in the Department of Histology and Embryology of the University of Sydney. The clinical expertise of Professor A. W. Bull is recognized for the operative procedures performed where required in the studies on the human dental pulp.

Introduction

This collection of published papers describes research into three areas of importance in the preservation of the dental structures and a fourth area is an excursion into biological problems associated with attempts to alleviate the loss of teeth from certain areas of the jaws.

1. The first group of papers, No. 1-13, is devoted to aspects of the study in the progress and prevention and progress of dental caries and refers to the pattern of development of carious lesions in subjects living in a particular environment, the effect of dietary restrictions and of the use of a food additive, and the effect of topical fluoride therapy on the course of the disease. The subjects of the studies were children and adolescents and the section is introduced with a short paper on hypoplasia of enamel in subjects born and residing since birth in a non fluoridated metropolitan area.

The comments on the series of papers No. 1-13 contain a number of references to authors whose observations are considered to have some relevance. These references are placed at the end of this section.

2. The progress of the carious lesion, if unchecked or undiscovered, inevitably leads through its destructive process to an assault upon the integrity of the dental pulp. Various procedures are available as therapeutic aids for the treatment of an inflamed or traumatized dental pulp. No method is available to enable an absolute assessment of the effect of such procedures. The clinician can only be assured by the elimination of painful symptoms as reported by the patient. Subsequently radiographic or clinical evidence may appear indicating death of the pulp and periapical infection.

The second group of papers, No. 14-25, is devoted to studies on the ultrastructure, histochemistry, and development (including histogenesis of pulp fibroblasts) of the human dental pulp.

These studies were also concerned with the ultrastructure of blood vessels in the normal dental pulp and the effect of injury and certain therapeutic

procedures in these vascular elements. The reaction of the extracellular tissues was also reported. An important aspect of the ultrastructure was the disposition and morphology of free nerve endings in the 'plexus of Raschkow'.

3. Loss of teeth from dental caries leads to an imbalance in the occlusal patterns and masticatory function. A sequel to this can be extensive disturbance of the temporo-mandibular articulation and in some patients a syndrome of pain and disability develops.

During the collection of material for the studies on the developing human dental pulp the opportunity was taken to retrieve tissue from the developing periodontium and this was subjected to electron-microscopy. The data from this study are presented in the third group of papers, No. 26-33.

The knowledge obtained from papers No. 28-32 has been developed and extended in a chapter devoted to Innervation of the human periodontium, In, The Temporomandibular Joint Syndrome (Editors C. J. Griffin and R. Harris) No. 4 in Monographs in Oral Science, 1975 (Publishers: S. Karger).

A supplemental group includes a paper on the periodontal tissues in rat molars and a clinical paper on a glomus tumour in the periodontal tissues, No. 34, 35.

4. The final three papers are of supplemental importance to the general theme. They do, however, provide an example of biological problems related to the maintenance of the masticatory apparatus which is the prime objective of dental practice. They present information on the use of implants, materials, and techniques.

Part 1. Dental caries

Paper 1. Enamel defects

Controversy has persisted for many years on whether structural defects in the enamel have any effect on the resistance of the tooth to dental caries. Pickerill⁽¹⁾, Mellanby⁽²⁾, Malherbe and Ockerse⁽³⁾, King and Martin⁽⁴⁾, Carr⁽⁵⁾, and Boyde⁽⁶⁾ are representative of those writers who suggest that departures from optimum tooth structure tend to lower the resistance of teeth to dental caries. Davies⁽⁷⁾ has shown, in the deciduous dentition, that a high prevalence of caries in the incisors of children on the island of Pukapuka was associated with structural defects in the enamel. Jones, Larsen and Pritchard⁽⁸⁾ first described this condition, calling it odontoclasia and other writers have reported its presence from areas where the prevalence of caries otherwise is low⁽⁹⁾⁽¹⁰⁾⁽¹¹⁾⁽¹²⁾⁽¹³⁾.

The paper records a high percentage of patients aged 6-14 years in which alterations in the refractive index of enamel and pitted or ridged defects of the enamel surface were found in children born and residing in the Sydney metropolitan area during the period when the water supply had not been fluoridated. Unpublished data by Charlton⁽¹³⁾ and Woods* suggest that such defective areas are sites which are predisposed to the accumulation of bacterial colonies, the development of dental plaque and subsequently dental caries.

Papers 2-6. 'Hopewood House' studies

The purpose of this project was to observe over a long period the progress of dental caries in children who had entered a home in early life and lived on a particularly restricted diet. The diet was described⁽¹⁴⁾ as lactovegetarian and in addition to the total absence of meat and meat products, refined carbohydrates were excluded, and there was

*Woods, R. Studies in Australia. Personal communication.

a high proportion of uncooked fruits and vegetables. Furthermore, it is important to emphasize that the mealtime programme was strictly followed and in-between-meal snacks were not allowed.

The exercise in the control of the diet was such that the children, attending schools in the area, were segregated from other children during lunch-time interval.

The progress of carious lesions was extremely slow and few dental restorations were inserted. Two major features were noted in the early years of the study:-

- 1) Virtual absence of dental caries,
- 2) negative or low lactobacillus counts
in the saliva.

Even when one or two carious lesions were discovered the bacteriological investigations showed very low or negative counts of lactobacilli. On the contrary a few children were found to have high lactobacillus estimations but clinical and radiographic examination did not reveal any carious lesions.

The eruption pattern of certain teeth in these children did not differ from the pattern reported

from other general community studies and the pattern of development of carious lesions suggests little or no relation to the pattern of eruption.

The use of toothbrushes and dentifrices as a means of oral hygiene was not practised by other than one or two of the older girls.

At the end of fifteen years 52 of the original 82 subjects were examined and a change in the prevalence of carious lesions was noted. Nevertheless it was lower than that experienced by the general community. Concomitantly with the increase in numbers of carious lesions there was a fall in the number of children who were caries-free and a rise in the number of children with lactobacillus counts of 1000 or more colonies.

It is important to note that the lactobacillus counts were not a close series, being taken at six-monthly intervals; and it has been recognized that a single lactobacillus count cannot be taken as an indication of any association with carious activity. In these studies the caries experience refers to that of an unique population and any inferences drawn from the results as reported

should not be hastily applied to the general population. Nevertheless the conclusion reached in Paper 3 seems reasonable, viz. that as the children grew older the influence of the 'Home' regimen was vitiated; the concentration of lactobacilli increased and the increase in the incidence of dental caries appeared to be related to the change in dietary habits.

Papers 4, 5, 6.

These papers provide information on the pattern of caries experience and the eruption pattern of the teeth from which it appears that no correlation exists between the two. However the progress of the carious lesion as measured by its proximity to the pulp (determined from radiographic data) was slower than that found in subjects of the same ages in the general population.

It was mentioned above that the prevalence of carious lesions increased as changes occurred in the diet of the subjects as they grew older. Reasons for this are suggested in Paper 3. The experience undergone by one of the subjects

subsequent to the conclusion of the study and not reported in the literature is of importance. His dentition is illustrated in Paper 5 (Fig. 7, 8); two years after that paper was published he suffered an accident and was hospitalized for 10 months. During this time his diet had completely changed from the 'lactovegetarian' programme of his Hopewood House days and furthermore he was unable to carry out adequate oral hygiene. When his mouth was examined, after recovery from his injury and period of hospitalization, 13 carious lesions on the approximal surfaces of his posterior teeth were detected.

Paper 7. Dental caries in Thai school-children

This paper reports a study of a group of children, of similar age to the Hopewood House subjects, living in the province of Chiang Mai in Northern Thailand. It is significant to this series, despite limitations noted above, because of differences in social patterns and individual food items between children in rural and urban areas. The former had a lower dental caries prevalence than the latter and the combined figures for

DMF teeth (caries index) were somewhat less than for the Hopewood House subjects.

Papers 8-11. Cariostatic effect of a food additive

As a practical proposition in the control of dental caries, dietary restriction is difficult to implement probably because the relationship between diet and dental caries is not obvious to many people and the acceptance of such restrictive programmes requires great efforts of self-discipline, and motivation. Except for certain conditions such as haemophilia, diabetes mellitus, and hereditary intolerance to certain carbohydrates, motivation is almost absent. Consequently procedures have been sought which would enable some counteraction to the cariogenic properties of refined carbohydrate foods. An example has been the use of phosphate food additives.

Most studies in this area have shown them to be relatively unsuccessful. However following long and extensive investigations in the laboratory on the properties of certain sugar phosphates followed by in vivo studies in laboratory animals as to its efficacy and freedom from toxic effects a complex of calcium sucrose phosphates and calcium orthophosphate

was used in a three year clinical trial as a food additive in the diet of children.

Before a clinical trial can be justified it is essential that a number of conditions should be met.

These are:-

- 1) Experiments in vitro must demonstrate that the agent to be tested is efficacious in changing or eliminating an important stage of the disease.
- 2) It must be unequivocally demonstrated that the agent is devoid of any toxic properties.
- 3) Studies in laboratory animals must demonstrate that the in vitro expectations have been fulfilled.
- 4) The agent is stable, is not detrimental to and preferably assists the manufacturing processes, and does not change the colour or taste of the processed food.

Studies on the food additive demonstrated that all of the above conditions had been met and the clinical trial was planned and set up.

Since the trial was designed to test the efficacy of a food additive, it was essential that it consist of a medical and a dental component. Detailed medical examinations of all children involved in the trial were carried out before the beginning of the trial and at six-monthly intervals, until two years had elapsed, and then again at the end of the three year trial.

The following laboratory investigations were undertaken:-

- 1) examination of the urine for protein and glucose;
- 2) estimation of the haemoglobin value and haematocrit;
- 3) examination of blood smear; and
- 4) estimation of urinary concentration of calcium nitrogen and creatinine.

The medical observers reported no differences between the Control and Treatment Groups of children.

Criticism has been made that the results were not as great as expected to have clinical value. The results showed that there was a reduction in the increment of dental caries of one surface per year

and this can be compared with reports where similar reductions were found in similar age groups of children where other methods of controlling dental caries were used⁽¹⁵⁾⁽¹⁶⁾⁽¹⁷⁾. A recent study by Wilson⁽¹⁸⁾ where the calcium sucrose phosphate additive was used in chewing gum five times per day reported a reduction of 1.3 surfaces per year.

Other criticism was that the use of a food additive would encourage the increased use of sugars and other refined carbohydrates. This is a speculation that should be examined. Records for sugar consumption in Australia for the decades between 1900 and 1970 show⁽¹⁹⁾ the following per capita consumption in pounds per annum:-

1900-1909	109
1910-1919	116
1920-1929	112
1930-1939	103
1950-1959	115
1960-1970	111

Figures for the decade 1940-1949 are now shown since data on sugar exported and used by Armed Services overseas were not available to allow for an accurate figure for home consumption to be given. On the basis

of that data the increase in sugar consumption expected by the criticism would seem to be unlikely. However, what may happen is that the pattern of eating will change. Evidence of this is the growth of 'snack bars' and take-away food stalls. It can therefore be argued with equal urgency that such changes in eating patterns may produce greater frequency in eating with emphasis on carbohydrate foods. If such a situation became universal the presence of the calcium sucrose phosphate complex would be increased by the frequency of eating and its effect enhanced. The study by Wilson⁽¹⁸⁾ would appear to support this hypothesis and the role of snack foods and confections with varying degrees of carbohydrate concentration has been emphasized by Bibby⁽²⁰⁾ and others⁽²¹⁾.

The trial developed out of an interesting set of circumstances. It demonstrated the effectiveness of industrial research combined with medical and dental investigations to study thoroughly the possibilities of the use of a food additive in the control of dental caries. It came into existence after the following studies had been made:-

- 1) Laboratory research on sugar phosphates for industrial purposes suggested the possibility

that they could be used as a vehicle for the transport of inorganic phosphates.

- 2) Studies⁽²²⁾⁽²³⁾⁽²⁴⁾ followed on the physical properties of the calcium sucrose phosphate complex which demonstrated its capacity to remineralize softened hydroxyapatite and dental enamel.
- 3) Studies with laboratory animals⁽²⁵⁾⁽²⁶⁾⁽²⁷⁾ suggested that organic phosphates played an important part in reducing the prevalence of dental caries; a further study⁽²⁸⁾ confirmed that the calcium sucrose phosphate complex followed the same pattern.
- 4) Extensive toxicological studies proved the substance harmless.
- 5) Manufacturers of processed foods found no difficulties with incorporating the complex with their products.

Papers 12-13. Topical fluorides

Two studies on the effect of fluoride solutions applied to the teeth of children aged 6-12 years were undertaken to compare the capacity of sodium fluoride

and stannous fluoride to reduce the incidence of dental caries. On the results obtained it would appear that sodium fluoride is more effective than stannous fluoride (33 per cent and 23 per cent reduction respectively). Since two different procedures were followed it is possible that the difference in efficacy of the two fluorides as reported was more apparent than real.

Summary

1. Evidence from various studies suggests that enamel structure may be a factor in the cause of dental caries and approximately 36 per cent of children living in a non-fluoridated area had some form of enamel defect.

2. The total elimination of refined carbohydrates from the diet produced a major retardation in the progress of dental caries and a diminution in concentration of lactobacillus acidophilus organisms in the saliva.

Subsequent relaxation in the strict diet as the subjects aged was associated with an increase in prevalence of carious lesions and an increase in lactobacilli in the saliva.

3. A similar pattern of lowered caries prevalence was observed in a group of Asiatic children.

4. The use of a calcium sucrose phosphate calcium orthophosphate complex as a food additive was shown to have some effect in reducing dental caries in children at the end of a three year clinical trial.

5. The topical application of sodium fluoride and stannous fluoride reduced dental caries by 33-23 per cent.

References

1. Pickerill, N. P.--Prevention of dental caries and oral sepsis. London, Bailliere, Tindall and Co., 1912.
2. Mellanby, M.--Diet and teeth. An experimental study III. Spec. Rep. Ser., Med. Res. Council (London) No. 191, 1934.
3. Malherbe, M., Ockerse, T.--Dental caries in high and low incidence in South Africa. S. Afric. J. Med. Sc., 9: 75, 1944.
4. King, J. D., and Martin, W. J.--Dental disease in parts of Trinidad and British Guiana. Brit. dent. Jnl., 90:2, 35-50 (Jan. 16) 1951.
5. Carr, L. M.--A correlation between Mellanby hypoplasia and dental caries. Dent. J. Aust., 25:7-8, 158-160 (July) 1953.
6. Boyde, A.--Scanning electron microscopy of enamel surfaces. Brit. Med. Bull., 31:2, 120-124 (May) 1975.

7. Davies, G. N.--A comparative epidemiological study of the diet and dental caries in three isolated communities. Proc. Conference on diet and oral health. Birmingham, Alabama, 1956.
8. Jones, Martha, Larsen, N. P., and Pritchard, G. P.--Dental disease in Hawaii. Dent. Cosmos, 72: 439-450, 1930.
9. Kirkpatrick, R. H.--Dental caries and odontoclasia in New Guinea. Dent. J. Aust., 7:11, 707-714 (Nov.) 1935.
10. Sinclair, Barbara, Cameron, D. A., and Goldsworthy, N. E.--Some observations on dental conditions in Papua-New Guinea, 1947, with special reference to dental caries VII. Dent. J. Aust., 22:3, 120-127 (March) 1950.
11. Davies, G. N.--A report on the dental status of native Fijians. New Zeal. Med. Council, 1949.
12. Williams, J. F.--Report on dental caries in Nive Island, Wellington. N.Z. Department of Health, 1953.

13. Charlton, G.--Studies in Papua New Guinea and Australia. Personal communication.
14. Sullivan, H. R., and Goldsworthy, N. E.--The biology of the children of Hopewood House, Bowral N.S.W. II. Observations extending over five years (1952-1956). Review and correlation of data presented in papers 1-6. Aust. dent. Jnl., 3:6, 396 (Dec.) 1958.
15. Barnard, P. D., Gillings, B. R., and Gillings, K. J.--Assessment of a preventive dentistry programme in private practice. New Zeal. Dent. Jnl., 70:1, 15-24 (Jan.) 1974.
16. Roder, D. M.--The association between dental caries and the availability of sweets in South Australian school canteens. Aust. dent. Jnl., 18:3, 174-182 (June) 1973.
17. Fanning, Elizabeth A.--Some aspects of a clinical study on dental caries in children. Arch. oral Biol., 13: 583-508, 1968.
18. Wilson, C. J.--Anti-caries effect of calcium sucrose phosphate in chewing gum. I.A.D.R., North American Division Abstracts 835, 1973.

19. Smythe, B. M.--Records compiled by the C.S.R.
Research Laboratories, Roseville, N.S.W.
20. Bibby, B. G.--The cariogenicity of snack foods
and confections. *J.A.D.A.*, 90:1, 121-131
(Jan.) 1975.
21. Edgar, W. M., Bibby, B. G., Mundorff, S., and
Rowley, J.--Acid production in plaques
after eating snacks: modifying food
factors. *J.A.D.A.*, 90:2, 418-425
(Feb.) 1975.
22. Napper, D. H., and Smythe, B. M.--The dissolution
kinetics of hydroxyapatite in the presence
of kink poisons. *Jnl. dent. Res.*, 45:6(2),
1775-1783 (Nov.-Dec.) 1966.
23. Brady, B. H. G., Napper, D. H., and Smythe, B. M.
--Effect of additives on the rate of
dissolution of hydroxyapatite in unstirred
acid buffers. *Jnl. dent. Res.*, 42:4,
603-612 (July-Aug.) 1968.
24. Brady, B. H. G., Napper, D. H., and Smythe, B. M.
--Dissolution of kinetics of hydroxyapatite.
Nature, 212:5057, 77-78 (Oct.) 1966.

25. Fedorov, Y. A.--Investigations of carious changes in teeth and indices of phosphorus and calcium metabolism in rats. *Doklady Biol. Science Section*, 150: 767-769, 1963 (Trans.).
26. McClure, F. J.--Cariostatic effect of phosphates. *Science*, 1337-8, 1964.
27. Bowen, W. H.--The monitoring of acid production in dental plaque in monkeys. *Brit. dent. Jnl.*, 126:11, 506-508 (June 3) 1969.
28. Lilienthal, B., Bush, E., Buckmaster, M., Gregory, G., Gagolski, J., Smythe, B. M., Curtin, J. H., and Napper, D. H.--The cariostatic effect of carbohydrate in the diet. *Aust. dent. Jnl.*, 11:6, 388-395 (Dec.) 1966.

Part 2. Dental pulp

Paper 14. Aldehyde fuchsin Halmi reaction

Developing human dental pulps were shown to react with aldehyde fuchsin Halmi stain after the sections had been oxidized with peracetic acid and the reaction was more intense in the developing than in the mature pulp. This reaction could not be demonstrated if digestion with hyaluronidase and, to a lesser extent, with *S* glucuronidase followed oxidation. If pulp tissue was removed from the tooth before fixation the staining of the ground substance was more intense which suggests that some of the acid mucopolysaccharides were lost during decalcification. The effect of enzymic digestion suggests that the material lost is a component of newly elaborated ground substance and may be associated with collagen synthesis.

Papers 15, 16. Ultrastructure of collagen and
fibroblasts and histogenesis of
fibroblasts

Fibroblasts were identified by the presence of numerous profiles of the rough-surface endoplasmic reticulum and dilated cisternae. An amorphous irregularly beaded electron dense substance, sometimes filamentous was demonstrated in cisternal vesicles. Extracellularly collagen fibrils of differing electron density associated with amorphous material, and straight, coiled and branched filaments were observed. It was suggested that the amorphous material could correspond to the aldehyde fuchsin Halmi positive material identified in Paper 14. Differences were noted in the morphology of fibroblasts of very young pulps as compared with those where the stage of root formation had been reached. In the former grossly dilated perinuclear cisternae containing small vesicles and beaded filaments, an extensive Golgi complex with many small vesicles and small mitochondria were seen. In the latter numerous secretory vesicles, some aggregated at the

plasma membrane were present. In the more mature pulp the extracellular substance had more collagen fibrils formed into bundles and associated with coiled irregularly beaded filaments. Some cisternal vesicles were seen external to but in close association with the plasma membrane of the cells.

Paper 17. Fine structure of nerve endings

This paper was concerned with the fine structure of nerve endings in the sub-odontoblastic zone (plexus of Raschkow). Endings resembling 'boutons terminaux' were described and identified by the presence of electron-dense constricted axons ending in bulbs containing synaptic-like vesicles, small mitochondria, neuro-tubules and neuro-filaments. They appear to arise from nerve terminals with varicosities. Other endings were seen associated with Schwann cells.

Papers 18, 19. Mature odontoblasts, cell rich zone and small blood vessels

A feature of the odontoblasts was the presence of numerous mitochondria, lipid droplets and numerous

highly electron-dense membrane-bounded structures possibly lysosomes. Between the odontoblast processes von Korff's fibres and unmyelinated nerve fibres were seen. In the sub-odontoblastic zone fibroblasts and undifferentiated mesenchymal cells and capillaries with a lumen formed from one or two endothelial cells were observed. The ultrastructure of capillaries, small arteries, terminal arterioles and the arterio-venous anastomosis are described and provide a basis for comparison with changes in those structures following injury to the dental pulp and following therapy.

Papers 20, 21. Healing and reaction of the pulp to injury

Light microscopy of the reactions in the dental pulp to trauma and treatment with particular reference to the use of calcium hydroxide and a glucocorticosteroid compound revealed deficiency in calcific repair and the presence of atubular dentine.

A constant feature reported by the patient was the absence of pain when the glucocorticosteroid was applied to the pulp. The transit of the glucocorticosteroid through the dentine and pulp was rapid.

Papers 22-25. The ultrastructure of the blood vessels
following injury

Four papers demonstrate changes in the traumatized human dental pulp at intervals of 2, 4, 14-21 days. Comparison between the inflammatory reaction to a non-therapeutic dressing (gutta percha) and a glucocorticosteroid compound shows an early suppression of the reaction occurred with the latter. As the interval between time of injury and recovery of the tissue extended, the tissues immediately beneath the zone of injury at 21 days showed evidence of sterile necrosis, with complete disintegration of the cells in some areas, and denaturation of collagen fibrils. A reactive zone was established around the necrotic tissue with macrophages. Changes in the microfibrillar material in the extracellular tissues with denaturation of the collagen suggests the former plays an important role in the maintenance of collagen. Some changes were noted in the blood vessels mainly in the arterioles and the capillaries in the apical zone. These were either partially occluded or patent and contained electron-dense granules.

Summary

The developing fibroblasts appear to be involved in the production of a microfibrillar reticulum and amorphous material associated with the synthesis of collagen fibrils. The amorphous material appears to be β glucuronidase and hyaluronidase labile after oxidation with peracetic acid. Exposure of the pulp without some form of medication results in severe inflammatory changes. Calcium hydroxide or a glucocorticosteroid compound stimulates some calcific reaction but the repair process is not complete. Exposed pulps treated with corticosteroid plus tetracycline show a zone of sterile necrosis immediately adjacent to the exposure. This necrotic area at 21 days was surrounded by macrophages. Blood vessels show changes in their walls and the arterioles were occluded in some instances at four days.

Part 3. Human periodontium

Papers 26, 27. Fine structure and protein
polysaccharide complex

These two papers are complimentary to Papers 15, 16 which were devoted to a similar examination of tissues in the human dental pulp. The main theme in these papers is fibrillogenesis. It is apparent that the synthesizing fibroblasts have similar characteristics in both tissues. Collagen fibrils of somewhat smaller diameter were seen and a microfibrillar reticulum was readily demonstrated lying in close association with them. The most important difference was the demonstration of the oxytalan fibrils which were associated with coiled, beaded microfibrils. Peracetic acid unmasked and ~~and~~ glucuronidase digestion' disrupted the microfibrils and the oxytalan fibrils and also the amorphous component associated with those structures.

Papers 28-33. Unmyelinated nerve endings and
innervation

The first of this series of papers describes the unmyelinated nerve endings, then follow four classifying and describing periodontal receptors. These are simple mechanoreceptors, compound, and complex mechanoreceptors. A brief description of the neural complex is given. The final paper describes in greater detail some of these elements and neural clusters, capsular cells, end rings and free nerve endings. It concludes with a discussion of functional considerations.

Paper 34. Periodontal reactions to trauma
and calcium deficiency

This paper is a light microscopy study of the rat periodontium following repeated local trauma, restrictions in calcium content and additions of sucrose to the diet. Bone resorption was observed at the region of trauma and some fragments of cementum were found in the periodontal tissue. Calcium deficiency was

associated with increased bone resorption. The addition of sucrose to finely ground diet did not appear to influence calculus formation.

Paper 35. Glomus tumour

The fortuitous finding of a small encapsulated soft mass of tissue attached to the root of an upper premolar led to an examination of the dental roentgenograms which revealed a slightly radiolucent zone in the alveolar septum. The soft tissue was found to have the histological features of a glomus tumour. From the history recorded it was found that the patient had complained of severe pain which had been diagnosed as arising from an 'antral abscess'.

Summary

The structure of the human periodontium has been examined. Electron micrographs show it has components with similar characteristics to those found in the human dental pulp. The major differences being the amount of collagen fibrils, the presence of oxytalan fibrils, and neural elements similar to mechanoreceptors and end rings of other neural elements. Observations on reactions of the periodontium to external influences and dietary changes show that both may have some part in the cause of periodontal lesions. The presence of a glomus tumour in periodontal tissues indicates the necessity of the clinician being aware of this entity in diagnosis.

Part 4. Supplementary studies

Papers 36, 37. Endosseous implants

These two papers cover investigations into the use of cobalt chromium and titanium endosseous implants in the alveolar bone of the jaws of rabbits and dogs and a small number in patients.

The results of the studies on the reactions to titanium in the jaws of dogs were favourable and prompted the trial of similar implants in patients. Four of these are reported in Paper 37 and at the time of writing the present comment have been functioning with prosthetic restorations for more than six years. The patient was highly motivated and cooperated excellently in maintaining a high standard of oral hygiene.

The histological studies on the tissues from the dog jaws show the limitations of the procedure, viz. the absence of an attachment between metal and the underlying bone and fibrous tissue. There was some inflammatory reaction in the gingival tissue associated with the protruding neck of the implant.

Paper 38. Wholly implanted materials

This paper reports the reactions of the hard and soft tissues to a number of materials implanted into the edentulous alveolar ridges of dogs' jaws.

The materials were:-

- i) Silicone rubber;
- ii) polytetrafluoroethylene (Fluon) block
- iii) polytetrafluoroethylene felt;
- iv) bovine cartilage.

i), ii), iii) are non absorbable.

The silicone rubber was exfoliated. The solid block of Fluon was accepted by the tissues

but required a special technique for its use and the Fluon felt created a giant cell reaction.

The bovine cartilage was lyophilized and then irradiated. Implanted within the bone it was tolerated by the tissues and functioned as a matrix being gradually replaced by bone.

---0---

Developmental Opaque Areas and Hypoplasia of Teeth

Robert Harris, M.D.S.*

INTRODUCTION.

During the course of examinations of numbers of children in the United Dental Hospital, Sydney, the opportunity was taken to observe the prevalence of opaque areas and minor hypoplastic defects in permanent teeth.

The children in this survey were all born in the County of Cumberland and all teeth recorded in the main survey are exclusive of any from the mouths of children afflicted with gross enamel dystrophies.

Since two surveys^{1, 2} made within this State indicate that the community drinking water supplied to these children is without fluorides, it can be concluded that the opacities and hypoplastic defects cannot be attributed to fluoride as a possible source.

Black³ defines mottled enamel as an endemic deformity distinguished especially by the absence of the cementing substance between the enamel rods in the outer fourth more or less of the enamel and presenting great variety of colour. In certain regions of comparatively few square miles, many thousands of persons have this deformity. The essential features in this definition, when considered from a clinical view, are (1) the condition is endemic, (2) there is a variety of colour and (3) many individuals are affected.

* Head, Department of Preventive Dentistry, United Dental Hospital of Sydney, and Part-Time Lecturer, Faculty of Dentistry, University of Sydney.

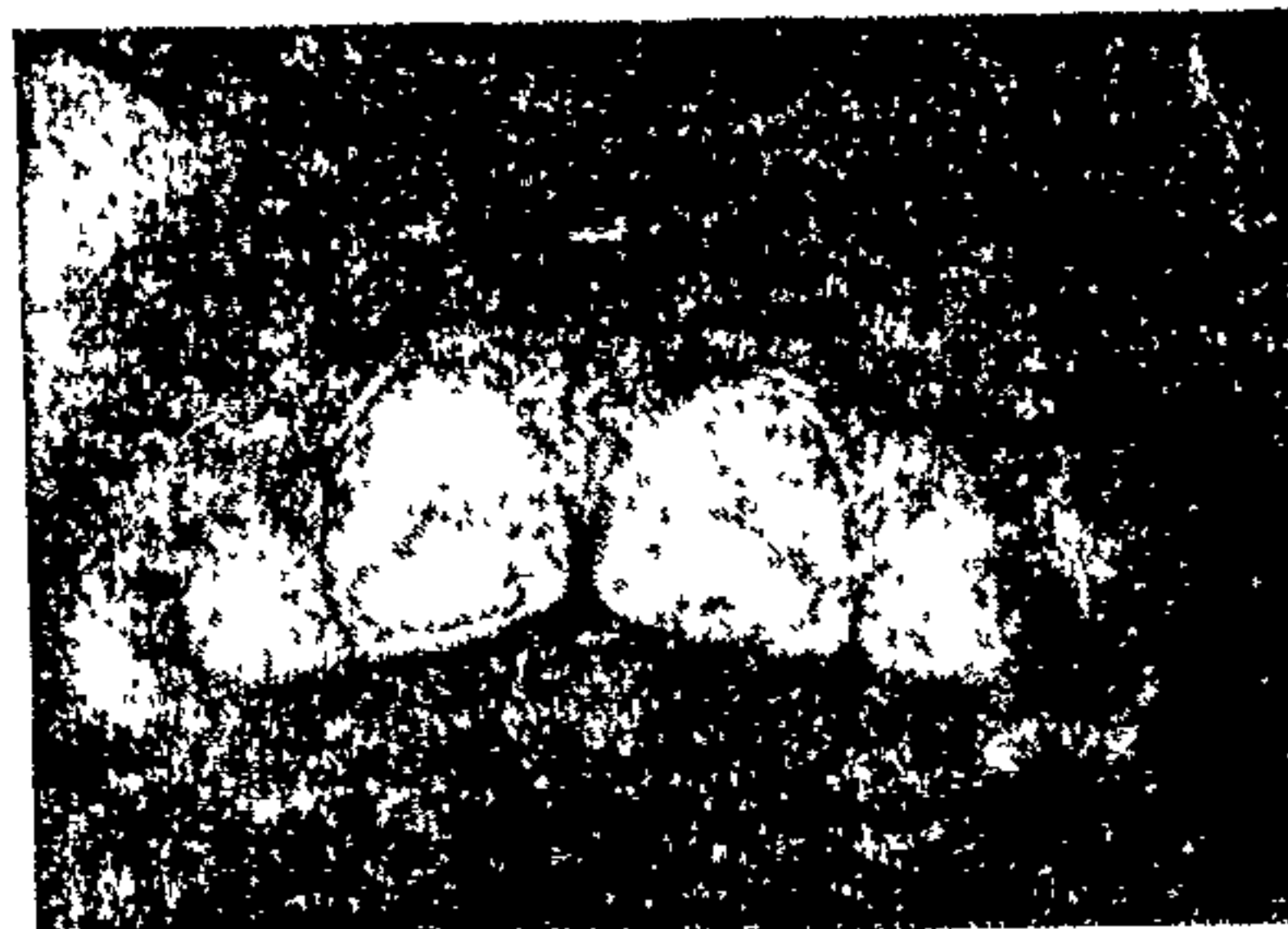


Fig. 1.—A photograph of the upper central incisor teeth on which there are large opaque white areas.

Furthermore, Black refers (loc. cit.) to white spots in the enamel. This deformity is paper white, "not very frequent and many of them are passed over without observation." This is a rather loose expression, if not conflicting.

McKay, writing in Black's work (op. cit.) refers to the

"constant misconception in the understanding and use of the term mottled; hence it is important to point out that this term applies exclusively to the grayish white or blotched appearance of the enamel and even includes the dead white phase and does not refer to the brown discolouration nor the corroded type."

McKay does not adhere to his definition, for many examples illustrated as mottled enamel demonstrate dark discolouration and corroded areas.

METHOD OF OBSERVATION.

The population group is not restricted to any one social strata since numbers of patients observed in the Department of Preventive Dentistry have been referred for consultation by private practitioners. On the other hand, the sample may not be truly representative of the child population of the Sydney Metropolitan Area, since the material was confined to those patients actually attending the Department for treatment. With this limitation in mind, this brief report is presented.

In the cases studied, there appears to be no definite pattern as to the teeth involved. Some show the defect on corresponding teeth in the one arch, others have the defect in opposing arches. Yet another modification observed is that of bilaterally opposed teeth such as an upper left molar and a lower right molar being affected. All cases recorded have been observed for more than two years and no disappearance of the zones have been noted in any discolouration.

In the younger age groups the teeth were noted during the eruptive stages. Some unerupted teeth have been observed with a mottled type of defect (the teeth — upper third molars were removed prior to the insertion of dentures).

Since the material for the survey comes from mouths of children born and living in the non-fluoride Metropolitan Area of Sydney, and, further, since these teeth have a wide range of defect, it can be reasonably assumed that many factors must be operating (cf. Sarnat and Schour⁴), for none of these children with the condition had any marked history of illness different from those whose mouths were free of the defect. Some of the defects are aesthetically disturbing. An example of this is shown in Fig. 1. It will be appreciated that whilst in this example the opacity is white, in many of those observed it ranged through white, yellow to brown. The zone of opacity, when small, has the same dense glistening surface of normal enamel, is more intense centrally and is more or less clearly demarcated from the surrounding

TABLE I

TOTAL NUMBER OF TEETH IN 481 PATIENTS

Age	No. of Patients	Affected Patients	Missing Permanent Teeth	Existing Permanent Teeth	Total Teeth
6	52	21	—	194	194
7	62	17	3	561	564
8	44	23	26	403	429
9	60	20	21	882	903
10	57	20	40	969	1,009
11	63	29	47	1,385	1,432
12	48	8	54	1,170	1,224
13	51	20	75	1,241	1,316
14	44	18	122	1,120	1,242
	481	176	388	7,925	8,313

TABLE II

OPAQUE AREAS AND HYPOPLASIA

AGE	OPAQUE AREAS				RIDGED				PITTED				PITTED AND RIDGED ENAMEL DEFECTS				TOTAL
	Upper		Lower		Upper		Lower		Upper		Lower		Upper		Lower		
	Ant.	Post.	Ant.	Post.	Ant.	Post.	Ant.	Post.	Ant.	Post.	Ant.	Post.	Ant.	Post.	Ant.	Post.	
6	4	21	4 (1)	19	2	3	—	2	—	2	3(1)	2	—	—	—	—	62
7	15	13(2)	10	15	—	—	—	—	2	3(2)	—	—	—	—	—	—	53
8	11(1)	24	7	21	6(1)	4	3	—	3	3	—	—	2	2	3	3	92
9	14(2)	18	9	17	4	—	—	—	6(2)	—	—	—	—	—	—	—	68
10	10	14	2(2)	16	—	—	—	—	4	—	6(2)	—	—	—	—	—	52
11	21(3)	32	8	20	11(2)	—	8	—	4	—	4	—	1(1)	—	—	—	118
12	6	5	6	5	—	—	1	—	1	—	—	1	—	—	—	4	25
13	12	22	12	13	—	—	—	—	—	—	—	—	—	—	—	—	63
14	22(2)	30	12	27	6(2)	—	10	—	4	2	2	1	—	—	—	1	117
	115(3)	179(2)	70(3)	102	29	7	22	2	24	10	15	4	3	2	3	8	655

N.B. Number in brackets indicates teeth with one or more defects, so that thirteen teeth in the opacities group also had some other defect.

structures; when large, the surface was sometimes found to be pitted centrally. Such conditions have been listed separately (cf. Hurme⁵).

Frisch⁶ records in Salida, Colorado (where fluorine exists naturally at 2 ppm.) that the incidence of fluorosis was high — 90.3 per cent. but in a fluoride-free area, Union Grove, Wisconsin, 15.2 per cent. showed some form of discolouration. Hurme, in his New Haven Study (op. cit.) gives a figure of 83.5 per cent.

Care has been taken to delete from the group some twenty patients who may be classified as examples of odontogenesis imperfecta, in which each patient was affected with a generalised disturbance of the enamel. In these cases there was a marked quantitative deficiency of the enamel and in seven, familial relationships were apparent. Cases A and B were a brother and sister, cases C and D brother and sister, cases E and F brothers, case G had normal brothers and sisters, but hereditary factors were found as far back as great-grandmother and grand aunts and aunts. No examples comparable with those recorded by Gustafson et alii⁷ have been observed. Since these cases do not form part

of this survey, no further discussion will be devoted to them.

MATERIAL.

The observations were made in the mouth in good natural light, the teeth having first been cleaned. The interval between the first and second observation was at least two years and no record was obtained where any discoloured area had disappeared.

The recorded observations are listed in Tables I and II.

It will be noted from Table I that the numbers of patients examined in this survey and missing teeth have been listed as well as existing permanent teeth. It cannot be assumed that any of the missing teeth were afflicted in any way, although this may have occurred since the bulk of the missing teeth are upper and lower molars.

The number of persons affected with one or more opaque areas is 176, giving a prevalence of 36.5 per cent. If the existing teeth are taken alone, then the total number of affected teeth — 642 — from Table II shows a prevalence of the affliction as 8.1 per cent. of all teeth observed in this group.

Analysis of Table II gives the following figures:

OPAQUE AREAS

	Upper		Lower		Total
	Ant.	Post.	Ant.	Post.	
Teeth	115	179	70	162	526
Percentage of affected teeth	17.9	26.3	10.9	25.3	81.9

HYPOPLASIA

(Ridged, Pitted and Combined)

	Upper		Lower		Total
	Ant.	Post.	Ant.	Post.	
Teeth	48	17	37	14	116
Percentage of affected teeth	7.4	2.6	5.7	2.1	17.7

COMMENTS.

Considerably more defects appear in the refractive index of the enamel ("opacities") than in any quantitative loss. In one case with rather thin upper incisors, a lingual discolouration, approximately conforming to the pattern of the labial areas, was present. This was noted by reflected light and in order to prevent error, the lip was kept over the labial of the teeth.

In view of the possibility of fluoridation of community drinking water as a means of reduction of dental caries and of the known effects of waters where the concentration of fluorides is such that it has been demonstrated as causing an observable effect, it is felt that this report is useful in showing that a form of opacity and mild hypoplasia can be demonstrated in a given community where there is no fluoride in that community's drinking water.

It seems essential in any preliminary survey where fluoridation is proposed, that this factor be assessed in considerably greater detail than in this report.

SUMMARY.

Observations as to mild hypoplasia in the teeth of 481 children in the age group 6-14 years, born and living in a fluoride-free drinking water area, demonstrated that 36.5 per cent. have some form of opacity and mild enamel defects.

Acknowledgment is made to the Superintendent, United Dental Hospital of Sydney, C. C. Croker, B.D.S., for permission to use the material in this report.

REFERENCES.

1. Jones, Pamela B.—Fluorine content and other chemical characteristics of potable waters in New South Wales: D.J. of Australia, 21:231 (May), 1949.
2. Reid, R. L. and Martin, N. D.—M.J. of Australia, July 27, 1946, p. 121.
3. Black, G. V.—Operative dentistry: 7 ed., Chicago, Medical Publishing Co., 1936, Vol. 3, p. 214.
4. Sarnat, B. G. and Schour, I.—Enamel hypoplasia (chronologic enamel aplasia) in relation to systemic disease: a chronologic, morphologic and etiologic classification: J.A.D.A., 29:67 (Jan.), 1942.
5. Hurme, V. O.—Am.Jrnl.Dis.Child., 77:1, 61, 1949.
6. Frisch, J. G.—The year book of dentistry: Chicago, The Year Book Publishers, 1949, p. 175.
7. Gustafson, G., Lystrom, P., and Stelling, E.—Odont. Tidsk, 55.

UNIVERSITY
OF SYDNEY
DENTAL LIBRARY

The biology of the children of Hopewood House,
Bowral, N.S.W.

II. Observations extending over five years
(1952-1956 inclusive)

Institute of Dental Research
United Dental Hospital, Sydney

2. Observations on Oral Conditions

H. R. Sullivan* and R. Harrist†

Introduction

Of great interest in the study of these children has been the observation of their caries experience because a characteristic of the dietary regime at Hopewood House is the avoidance of refined carbohydrates. Therefore the greater part of this paper is devoted to a discussion of the occurrence of caries in the Hopewood children. However, brief mention will be made of the condition of their gingivæ and of any other deviations from normal.

Method

All children have been examined at least once a year and the majority twice a year. A most careful examination by means of mirror, probe and radiography was made by the one observer (R.H.), who then conferred with a second observer (H.R.S.). All erupted teeth, carious or filled surfaces, stains on the teeth, gingivitis and developmental aberrations were recorded. The records for each child were kept on a specially designed sheet with space for the ten half-yearly examinations made during the five years. At the end of the five-year period the sheet was examined for discrepancies concerning carious lesions and these were corrected after the manner described in the report on the Vipeholm Dental Caries Study.^{(1)*} Because the majority of the lesions were extremely small and it was important (in relation to bacteriological studies) to know as precisely as possible when a lesion began, it was the custom to record a suspected lesion as carious until later examinations resolved the doubt.

* Institute of Dental Research.

† Department of Preventive Dentistry, United Dental Hospital of Sydney.

* If a lesion had been detected in, say, 1953-54 and in 1956, but not in 1955, the record was amended to show its presence in 1955; if a lesion had been recorded as present in 1953 but was not detected in 1954-55-56, it was considered not to have been present in 1953.

⁽¹⁾ Gustafson, B. E., *et al.*—The Vipeholm Dental Caries Study. *Acta Odont. Scandinavica*, 11: 195-388 (Sept.), 1954.

Results

Developmental Aberrations

A. *Missing permanent teeth.* Permanent teeth were missing in six children, and of these five lacked a lower second bicuspid (in one boy both second bicuspids); the sixth child lacked an upper lateral incisor.

B. *Opaque areas.* A total of 36 teeth in nine boys and ten girls showed white or brown opaque areas: 31 teeth had white spots and five brown.

C. *Hypoplasia of enamel.* For present purposes hypoplasia was accepted as being a defect in morphology of the enamel, whether it were an aberration of the total structure, a linear depression or a series of punctate areas. Five children (three boys, two girls) had 31 affected teeth and one of the girls had a linear form of hypoplasia affecting all 24 permanent teeth.

D. *Supernumerary teeth.* Only one child had a supernumerary tooth in the permanent dentition. It was situated in the upper anterior region.

Surface Stains

Because of difficulties in supervision, no organized system of tooth-brushing had been

TABLE 1.
Distribution and duration of stains on the teeth.

Number of years over which stain was seen.	Boys.	Girls.
4	8	6
3	13	16
2	7	7
1	10	6
0	3	6
	41	41

introduced and despite the rather fibrous nature of the diet the oral hygiene of the children, as a whole, was poor. This is reflected in the extremely small number of children whose teeth were entirely free from some form of brown, green or yellow exogenous stains (Table 1).

Stains seen in the mouths of three girls in 1953 and 1954 had disappeared at subsequent examinations.

Calculus

Calculus was detected in six girls and six boys, the lower incisor area being the most frequent site. However, the amount was small and subgingival calculus was not observed at all. In two boys and two girls the deposits subsequently disappeared, and in three girls the presence of calculus was observed only at the last observation.

Gingivitis

Despite the absence of organized tooth-brushing 13 girls were free of gingivitis, nine of these being ten years of age or less. Seven boys were also free of gingivitis, four of these being ten years of age or less.

Gingivitis was always absent in seven boys and thirteen girls.

TABLE 2.
Distribution and site of gingivitis.

Boys.	Girls.	Area.
13	9	Lower incisor
2	5	Upper incisor
—	—	Lower posterior
—	—	Upper posterior
1	—	Upper and lower posterior
12	8	Upper and lower incisor
6	6	All quadrants
34	28	

Caries Experience

For the purpose of compiling the following tables only the results of the five annual examinations made at the end of each calendar year have been used. Because it is impracticable to present the serial results for individual children, the data have been processed in different ways.

Method 1. All children were born in the years 1942-49 and the examinations reported in this paper were made in the years 1952-56, hence children attained ages from 3 to 14 years during that period. It was found that sufficient children attained ages of 5 to 13 years to justify the grouping of the results of their examinations according to age. For example, the results of examination of a child born in 1942 have been used in the 10, 11, 12 and 13 year age groups (1952-55 examinations) and those of the child born in 1949 have been

used in the 5, 6 and 7 year age groups (1954-56 examinations) (Table 3). Table 4 sets out the mean number of d.e.f.* and D.M.F.† teeth per child at the ages 5 to 13 years.

For purposes of comparison groups of children of similar age and living under approximately the same socio-economic conditions, but

TABLE 3

Year of Birth	Age in					Number
	1952	1953	1954	1955	1956	
1942	10	11	12	13	14	6
1943	9	10	11	12	13	32
1944	8	9	10	11	12	53
1945	7	8	9	10	11	59
1946	6	7	8	9	10	68
1947	5	6	7	8	9	74
1948	4	5	6	7	8	49
1949	3	4	5	6	7	29
Number	1	2	14	23	29	

not subject to the same dietary regime, such as were included in the survey of Barnard,⁽²⁾ are ideal. He examined children attending State schools in New South Wales by the same methods as we used, but because he examined permanent teeth only, Tables 5 and 6 include comparative data for permanent teeth.

Method 2. It is also of interest to know the rate at which lesions have developed, their size and other data. Therefore results of the five annual examinations are presented in Tables 8, 9 and 10. The children were placed in three arbitrary groups according to age so that three approximately equal groups were obtained. Thus the results of those born in 1942 and 1943 were pooled; likewise, of those born in 1944 and 1945 and also of those born in 1946, 1947, 1948 and 1949. By this means three groups of children numbering 32, 27 and 23 were obtained. Their distribution is shown in Table 7 and the following tables set out relevant data for these groups.

Discussion

Observations on the prevalence of various oral conditions reveal that this group of

* "d.e.f. teeth" means those temporary teeth which were decayed, extracted because of caries or filled.

† "D.M.F. teeth" means those permanent teeth which were decayed, extracted because of caries or filled.

⁽²⁾ Barnard, P. D.—Dental Survey of State School Children in New South Wales, N.H.M.R.C. Special Report Series, No. 8, Canberra, 1956.

TABLE 4.
Mean number of def and DMF teeth per child according to age.

Age in years ..	5	6	7	8	9	10	11	12	13
Number in groups	14	23	29	49	74	68	59	53	32
def teeth ..	0.14	0.61	0.86	0.96	1.11	0.88	0.83	0.79	
S.D.	0.52	1.34	1.30	1.34	1.64	1.23	1.16	1.08	
DMF teeth ..		0	0.14	0.24	0.41	0.38	0.61	1.08	1.06
S.D.		0	0.46	0.66	0.84	0.82	1.28	1.90	1.78

TABLE 5.
Comparison of the mean number of DMF teeth per Hopewood child with that per child of similar age attending State schools in N.S.W.

Age in years ..	6	7	8	9	10	11	12	13
Hopewood child..	0	0.14	0.24	0.41	0.38	0.61	1.08	1.06
S.D.	0	0.46	0.66	0.84	0.82	1.28	1.90	1.78
State school child	0.99	2.31	3.22	4.44	5.28	6.98	9.32	10.70
S.D.	1.38	1.70	1.91	2.32	3.08	4.10	5.33	5.15

TABLE 6.
Percentage of Hopewood children having caries-free permanent teeth compared with that of children attending State schools in N.S.W. according to age.

Age in years ..	6	7	8	9	10	11	12	13
Hopewood children	100	89.6	85.7	78.4	79.4	74.6	62.3	53.1
State school children	58.7	23.5	10.5	4.1	4.6	2.5	1.0	0.4

TABLE 7.
Arbitrary grouping of 82 children according to age.

Year of birth ..	Group I.		Group II.		Group III.			
	1942	1943	1944	1945	1946	1947	1948	1949
Boys	2	15	14	1	3	4	1	1
	17		15		9			
Girls	4	11	7	5	6	8		
	15		12		14			
Total	32		27		23			

children is affected by developmental aberrations such as are encountered in the community at large. The frequency with which lower second bicuspid teeth were missing is noteworthy.

The relative infrequency of calculus conforms to the pattern seen in mouths that are actively used for mastication of fibrous food, a pattern which can be expected more particularly in children.⁽³⁾ Calculus was not related to any particular age in the girls, but in the boys it tended to occur in those aged over ten years. The presence of stains upon the teeth in 73 of the children demonstrates that oral hygiene was not good and supports the observation made in the introductory paper that it appeared to play little or no part in determining the caries experience.

The presence of areas of white or brown opacity in the enamel could not be attributed to fluorosis, because the water supply in the area is free of fluorides. The staining of the teeth in this group varied considerably from a thin dark brown line just above the gingival margin to yellow or green areas on buccal and labial surfaces. The brown stain was associated with a structure which appeared to be tough and closely adherent to the tooth whilst the yellow and green stain in most cases was associated with heavy deposits of materia alba. All types of stain occurred in some mouths.

Table 2 shows that 75 per cent. of the children had gingivitis. This was determined by the presence of alterations in the colour and surface texture of the marginal gingivæ or interdental papillæ. Wherever these tissues had a red colour darker than the normal pink shade of the gingivæ or where they were oedematous and smooth, that area was recorded as affected by gingivitis. Two of the oldest girls had some ulceration, as indicated by bleeding from the gingival sulcus. It is interesting that the region of greatest prevalence is in the anterior part of the mouth; the reason for this has not been determined. Parfitt⁽³⁾ suggests that the commonest site for gingivitis in a child is in the upper buccal segments.

It will be seen from Table 4 that the rate of increase in caries in this group of children has remained extremely low. The mean number of d.e.f. teeth in children at the age of

five years was 0.14 and at twelve years 0.79. The highest mean number of d.e.f. teeth (1.11) occurred when children attained the age of nine years, indicating a slow rate of development of carious lesions and a retention of temporary teeth until their correct time of exfoliation. Children retained some of their temporary teeth up to the age of 12 years. These had often undergone extreme attrition, the occlusal surfaces at times being quite concave. However, when lesions occurred they were little different in their clinical appearance from those occurring in other mouths but often had dark, hard, carious dentine. Likewise, the rate of decay of permanent teeth has been very slow; none of the children at the age of six years had any decayed permanent teeth, and at the age of 13 the average number of D.M.F. teeth per child reached only 1.6. The rate of development of carious lesions apparently increased slightly from the age of 11 years onwards.

A comparison of these figures for permanent teeth with those for groups of children of similar age attending State schools in N.S.W. reveals a remarkable difference. Even at the age of six years children in State schools had an average of one D.M.F. tooth per child, and this number increased rapidly until at the age of 13 years the children had an average of 10.7 D.M.F. teeth per child. In all groups of children of similar age the amount of caries as expressed by the D.M.F. teeth was at least tenfold greater in the State school children. This contrast is also made obvious by comparing the percentage of children having caries-free permanent teeth on attaining the ages of 6 to 13 years. On attaining the age of 13 years 53% of the Hopewood House children still retained completely caries-free permanent teeth, a figure which is almost identical with that of the six-year age group of the children attending State schools. After the age of six years the percentage of caries-free children attending State schools dropped extremely rapidly so that in the 13-year age group only 0.4% retained caries-free permanent teeth. The Hopewood children did not show as great a rate of decrease as did the State school children, but the percentages did appear to decrease somewhat more rapidly from 11 years onwards.

From an examination of Tables 8, 9 and 10, where the results have been grouped according to date of birth of the child, it will be seen that the oldest age group included relatively

⁽³⁾ Parfitt, G. J.—Clinical Pedodontia. Philadelphia, W. B. Saunders Co., 1957 (p. 451).

TABLE 8.
The caries experience of 32 children included in Group I during the years 1952-1956.
Number in group: 17 boys, 15 girls.

Year.	Caries-free.			d.e.f. teeth.			d.e.f. surfaces.			D.M.F. teeth.			D.M.F. surfaces.			Restorations temporary teeth.			Restorations permanent teeth.				
	Boys.	Girls.	Total No.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.		
																						Mean.	Mean.
1952 ..	13	10	23	0.76	0.47	0.63	0.94	0.47	0.72	0	0.27	4	0.13	0	0.13	0.12	0	0.06	0	0.13	2	0.06	
1953 ..	11	6	17	0.65	0.80	0.72	0.94	0.93	0.94	0	0.40	6	0.19	0	0.22	0.18	0.13	5	0.16	0	0.20	3	0.09
1954 ..	12	7	19	0.41	0.47	0.44	0.53	0.67	0.59	0.06	1.0	16	0.50	0.06	0.50	0.12	0.07	3	0.09	0	0.20	3	0.09
1955 ..	12	7	19	0.29	0.07	0.19	0.35	0.07	0.22	0.18	1.47	25	0.78	0.18	0.88	0.06	0	1	0.03	0	0.20	3	0.09
1956 ..	10	6	16	0.12	0	0.06	0.26	0	0.13	0.94	2.93	60	1.88	1.0	3.67	72	2.25	0	0	0.06	0.33	6	0.19

Mean = mean per child.

TABLE 9.
The caries experience of 27 children included in Group II during the years 1952-1956.
Number in group: 15 boys, 12 girls.

Year.	Caries-free.			d.e.f. teeth.			d.e.f. surfaces.			D.M.F. teeth.			D.M.F. surfaces.			Restorations temporary teeth.			Restorations permanent teeth.				
	Boys.	Girls.	Total No.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.		
																						Mean.	Mean.
1952 ..	9	8	17	0.67	1.0	0.81	0.67	1.25	25	0.93	0.13	0.08	3	0.04	0.11	0	0.17	2	0.07	0.13	0	2	0.07
1953 ..	6	7	13	0.93	1.58	1.22	1.20	2.08	43	1.59	0.33	0.88	15	0.55	0.59	0.13	0.25	5	1.85	0.13	0	2	0.07
1954 ..	6	5	11	0.87	1.58	1.19	1.20	2.17	44	1.63	0.33	1.08	18	0.67	0.78	0.13	0.17	4	0.15	0.13	0.17	4	0.15
1955 ..	5	5	10	0.87	0.42	0.67	1.0	0.58	22	0.81	0.53	1.08	21	0.78	0.93	0.07	0	1	0.04	0.13	0.17	4	0.15
1956 ..	3	6	9	0.93	0	0.52	1.80	0	27	1.0	1.07	2.5	46	1.70	2.07	0.06	0	1	0.04	0.13	0.58	9	0.33

Mean = mean per child.

TABLE 10.
The caries experience of 23 children included in Group III during the years 1952-1956.
 Number in group: 9 boys, 14 girls.

Year.	Caries-free.			d.e.f. teeth.			d.e.f. surfaces.			D.M.F. teeth.			D.M.F. surfaces.			Restorations temporary teeth.			Restorations permanent teeth.			
	Boys.	Girls.	Total No.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	Boys.	Girls.	Total.	
																						Mean.
1952 ..	7	14	21	0.33	0	3	0.33	0	3	0.13	0	0	0	0	0	0	0	0	0	0	0	0
1953 ..	5	10	15	0.89	0.5	15	0.89	0.5	15	0.66	0	1	0.07	0	1	0.04	0	0	0	0	0	0
1954 ..	5	7	12	1.0	0.79	20	1.0	0.79	20	0.87	0.22	5	0.22	0.21	5	0.22	0.21	5	0	0	0	0
1955 ..	4	7	11	1.56	1.0	28	1.56	1.0	28	1.22	0.22	6	0.22	0.29	6	0.22	0.29	6	0	0	0	0
1956 ..	2	5	7	1.89	1.29	35	2.33	1.43	41	1.78	0.56	10	0.43	0.36	12	0.78	0.36	12	0	0	0	0

Mean = mean per child.

more caries-free children than the other two groups at the last examination. The reason for this is not apparent.

It is interesting to compare the numbers of d.e.f. and D.M.F. teeth with the number of d.e.f. and D.M.F. surfaces at the various examinations. They will be seen to be almost identical, indicating that the majority of lesions remained small. When there is a difference it is often because a restoration had been inserted which required extension to another (usually caries-free) surface or because occasionally a carious temporary tooth had been extracted. Bodecker's method of scoring⁽⁴⁾ was used and thus an extracted tooth was counted as equivalent to three involved surfaces. The relatively slow extension of cavities is also indicated by the small number of restorations required. It was policy not to restore a tooth as soon as a cavity was detected

⁽⁴⁾ Bodecker, C. F.—The modified dental caries index. J.A.D.A., 26: 1453-1460 (Sept.), 1939.

but only when it was considered necessary for the maintenance of the tooth's vitality and function. Very occasionally, despite half-yearly examinations, an abscess developed on a deciduous tooth or a tooth was considered too carious to warrant a restoration, in which case it was extracted. At the end of the five-year period only 15 restorations had been inserted in permanent teeth.

Although the mean number of carious teeth is very small, considerable variation occurred between individuals. One child had 12 small lesions present at the last examination but none of these required restoration. Another child had ten lesions, another nine, one eight and one seven. On the other hand, 25 children remained caries-free for the five-year period.

Summary and Conclusions

The examination of the mouths of the Hope-wood children over a five-year period revealed that whilst their oral hygiene was poor the caries experience was extremely limited.