



THE UNIVERSITY OF
SYDNEY

COPYRIGHT AND USE OF THIS THESIS

This thesis must be used in accordance with the provisions of the Copyright Act 1968.

Reproduction of material protected by copyright may be an infringement of copyright and copyright owners may be entitled to take legal action against persons who infringe their copyright.

Section 51 (2) of the Copyright Act permits an authorized officer of a university library or archives to provide a copy (by communication or otherwise) of an unpublished thesis kept in the library or archives, to a person who satisfies the authorized officer that he or she requires the reproduction for the purposes of research or study.

The Copyright Act grants the creator of a work a number of moral rights, specifically the right of attribution, the right against false attribution and the right of integrity.

You may infringe the author's moral rights if you:

- fail to acknowledge the author of this thesis if you quote sections from the work
- attribute this thesis to another author
- subject this thesis to derogatory treatment which may prejudice the author's reputation

For further information contact the University's Copyright Service.

sydney.edu.au/copyright

see also at 617.65
TAI

TAIREA, T
Dental Health Care Plan for children
aged 5 to 12 years for the Islands of
Rarotonga, Aitutaki and Penrhyn

CONTROL OF DENTAL CARIES IN CHILDREN
IN THE COOK ISLANDS

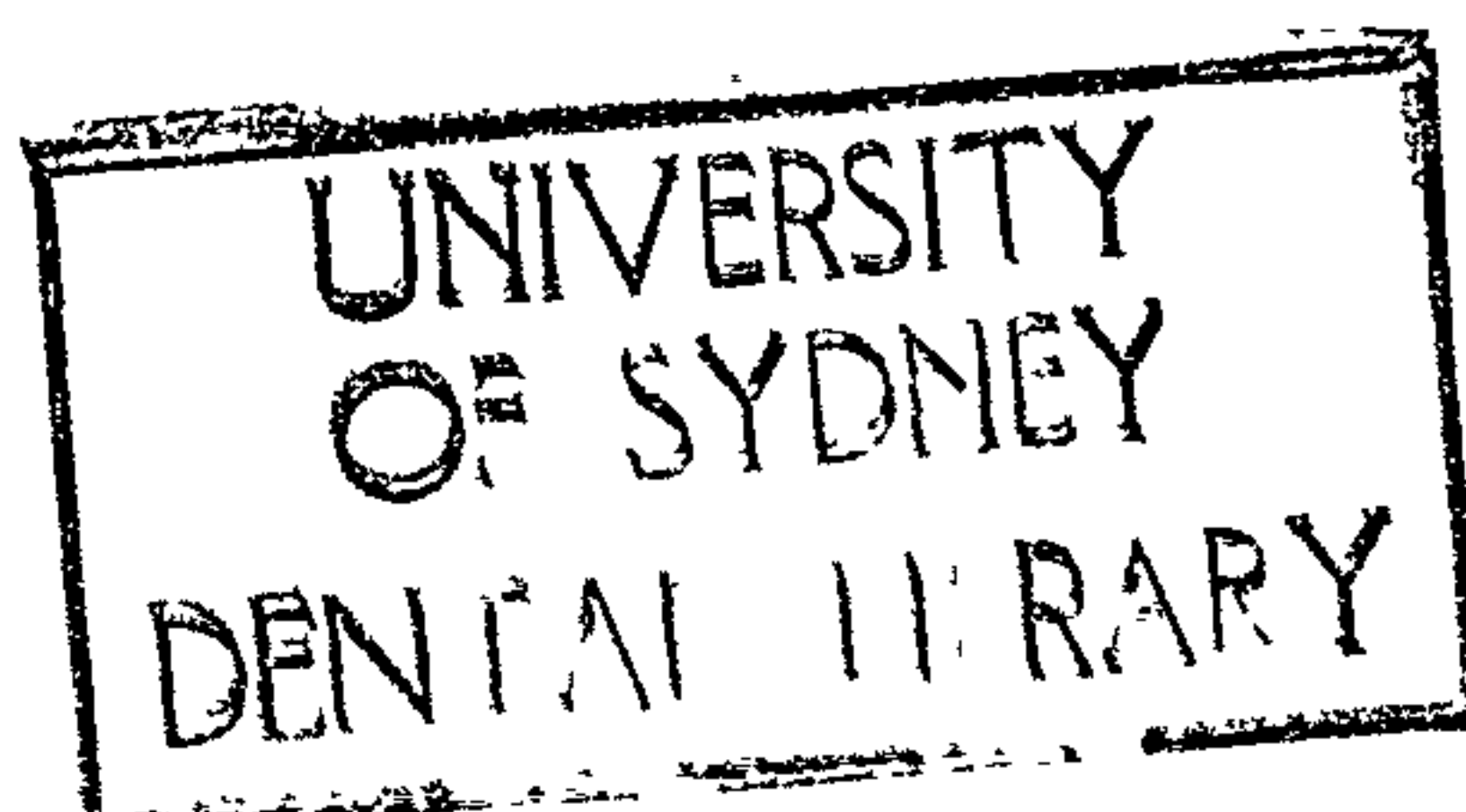
TEREPAI TAIREA
DSD (FIJI), DPHDENT (SYDNEY)



A treatise submitted in partial requirement
for the degree of
MASTER OF DENTAL SCIENCE

Department of Preventive Dentistry
Faculty of Dentistry
University of Sydney

1986



SUMMARY

This treatise deals with the review of the literature on the actions of silver and tin compounds, especially silver nitrate, silver fluoride and stannous fluoride for their role in the arresting and slowing of the progression of dental caries.

Survey data showed that all school children in the Cook Islands were affected by dental caries at the age of 13 years. Because of the arresting and slowing of progression of dental caries, by the above mentioned compounds, the writer feels that this treatment procedure can be adopted for controlling dental caries in children in the Cook Islands.

Dental caries is the most common of oral diseases, and its prevalence can only be reduced by prevention. The two major methods of preventing dental caries from an ecological point of view are:

1. By increasing the resistance of tooth enamel to external attack through systemic and topical administration of fluoride.
2. By reducing the intensity of the attacking agents through reducing consumption of sugars and regular efficient system of tooth cleansing to remove plaque.

Dental caries progression is of interest both in clinical practice and research. Caries is an initially reversible, dynamic process with episodes of destructive activity, quiescent periods, and periods of remineral-

ization: Remineralization occurs naturally during the formation of a carious lesion in human enamel. The presence of fluoride ions enhances greatly the degree of remineralization and reduces the time period for the mechanism to occur.

Silver nitrate has been used in the past as an agent to arrest or slow the progression of carious lesions. It has also been used in the profession as a remedy for hypersensitivity of dentin, erosion and pyorrhoea, and as a sterilizing agent and caries inhibition in deciduous as well as permanent teeth.

Craig's silver fluoride treatment procedure is the one considered in this thesis for the arresting of dental caries. In this procedure, the area to be treated is isolated with cotton rolls. In the case of lower area isolation, Junior Garmers Clamps are mandatory. A 40% silver fluoride solution on a pellet of cotton wool is applied to the region occlusal to the contact area and then moulded lingually and buccally of this contact area. The pellet is left in the buccal region and moved gently in and out for three minutes. A spot application of 10% stannous fluoride paste is applied to the regions occlusal, lingual and buccal to the contact area. A black stain would appear in the treated lesion after application of SnF_2 spotting paste. A small piece of stomahesive wafer (approximately 5 mm wide by 10 mm long) is carefully adapted over the treated site. This treatment procedure takes approximately 5 minutes per lesion. The patient is

asked not to eat for one hour and also informed that the wafer will slowly dissolve in the mouth fluids. At recall examinations, a continuous black mat associated with caries arrestment remained in the treated lesion (Craig, Powell, Cooper 1981). The rate of caries progression obtained by Craig et al. 1981 using this treatment procedure was slow.

The cost benefit and cost effectiveness of the silver fluoride and stannous fluoride therapy is not dramatic compared to water fluoridation or some self-applied topical fluoride therapies because the technique requires dentists and therapists to carry out the treatment procedures. However in countries such as New Zealand, Australia, and the Cook Islands where clinical facilities, dentists and auxiliaries are already providing dental care to the majority of the school children, the additional time taken is less than if full restoration had been carried out.

The Cook Islands consist of 15 tiny scattered islands, of which Rarotonga is the largest. Almost half of the 20,000 people of the Cook Islands live in Rarotonga. Reports of the few surveys carried out in the Cook Islands indicate that dental caries is the most prevalent of all diseases. Survey data published in 1970 and 1976 showed that all school children on Rarotonga were affected by the age of 13 years (no other survey has been carried out since 1976).

The school children on Rarotonga had a DMFT of 10 at 14 years of age. On Mangaia, 90% of 13 year olds were

similarly affected. One survey carried out in one of the northern group islands (Pukapuka - Davies 1956) revealed a low DMFT in the school children. The difference in the oral health status of the children in Pukapuka compared to that of Rarotonga can be explained by the fact that Rarotonga is the island of call for trading vessels - hence the greater availability of refined sugar.

In conclusion, it is emphasised that because primary teeth have a finite life-span the slowing or the progression of carious lesions may allow deciduous teeth to reach exfoliation without the need for conventional intervention. Secondly, priority can be given to the larger cavities with little risk of the remaining lesions developing too rapidly. There should be more clinical studies carried out on the use of tin and silver in their arresting and slowing of carious lesions since there are strong indications showing its potential as a practical preventive measure.

ACKNOWLEDGEMENTS

I would like to express my sincere thanks and gratitude to Professor Barnard, Associate Professor of Preventive Dentistry, University of Sydney and Director of Preventive Dentistry, Westmead Dental Clinic School for his expert guidance and invaluable assistance during the preparation of this treatise. His encouragement, patience and understanding are very much appreciated.

My thanks also go to the following persons and organizations who have greatly assisted me.

1. Professor Craig of the Preventive Dentistry Department, University of Sydney, for his assistance and expert guidance in the area studied for this treatise.
2. Australian Development Aid Bureau (ADAB) for funding this Master of Dental Science Degree Course (Dental Public Health) in the Preventive Dentistry Department, University of Sydney.
3. The Cook Islands Government for their support and confidence in my taking this MDSc course.
4. Mr. George Udall, the Principal Dental Officer of the Western Metropolitan Region for granting me permission to carry out a survey into the use of silver fluoride in some schools in the Western Region of New South Wales.
5. My wife Mehau and our children, Maria, Bernard and Cyprian for their moral support and encouragement.

TABLE OF CONTENTS

	<u>Page</u>
SUMMARY	i
ACKNOWLEDGEMENTS	v
TABLE OF CONTENTS	vi
LIST OF TABLES	ix
LIST OF FIGURES	x
 1. INTRODUCTION	 1
1.1 Aims	8
 2. REVIEW ORAL HEALTH STATUS	 9
2.1 The Cook Islands - Demography	9
2.2 Dental Services in the Cook Islands	17
2.3 Oral Health Status in the Cook Islands	22
2.4 Factors Influencing Changes in Oral Health Status	28
2.4.1 Changes in food consumption	28
2.4.2 Water supply	28
2.4.3 The education system	29
 3. TIN AND SILVER COMPOUNDS	 30
3.1 History	30
3.2 The Effect of Heavy Metals on Dental Caries (Theory)	38
3.3 The Effect of Tin (Sn) on Dental Caries	39
3.4 The Effect of Silver (Ag) on Dental Caries	40

	<u>Page</u>
3.5 The Effect of Fluoride on Remineralization	41
3.5 Remineralization	42
4. DENTAL CARIES	45
4.1 Etiology	46
4.1.1 Bacteria	46
4.1.2 Acid dissolution	48
4.1.3 Carbohydrates	51
4.2 Caries Progression	55
4.2.1 Studies which have monitored progression	58
4.2.2 Results of studies on caries progression	61
4.3 Changing Patterns of Dental Caries Prevalence	63
5. APPROPRIATENESS OF THE USE OF THE COMPOUNDS FOR THE COOK ISLANDS	71
5.1 Possible Mode of Action of Silver Fluoride (Hypothesis)	73
5.2 Clinical Effectiveness	75
5.3 Cost-Analysis	75
5.4 Cost-Effectiveness	78
5.5 Advantages of the AgF Treatment	79
5.6 Disadvantages of AgF Treatment	79

	<u>Page</u>
6. DISCUSSION	80
6.1 Introduction	80
6.2 Usefulness of the Treatment Procedure in the Cook Islands	81
6.3 Silver Compounds	83
6.4 Dental Caries	85
6.5 Changing Patterns of Dental Caries Prevalence	88
6.6 Remineralization	88
6.7 Appropriateness of the Use of the Compounds	89
6.8 Treatment Procedures	89
6.9 Clinical Effectiveness	90
6.10 Cost-Analysis	90
7. CONCLUSIONS	92
8. REFERENCES	95
APPENDICES	104
APPENDIX 1 - Summary of Survey	104
APPENDIX 2 - Craig's Treatment Procedures and Some Basic Rules	109
APPENDIX 3 - Cost of Materials	122

LIST OF TABLES

<u>Table</u>	<u>Page</u>
1. Mean DMFT of Rarotonga - Pre-1970 and 1976 (Source: Speake 1976)	23
2. Family Budget Sample Survey - 1976 Cook Islands (Source: Speake 1976)	27
3. Summary of Papers Which Have Monitored Progression of Approximal Carious Lesions by Studying Apparent Depth of Lesion Using Bitewing Radiographs (Source: Pitts 1983)	60
4. Caries Prevalence Trends in Highly Industrialized Countries, DMFT in Children Aged 12 Years (Source: Renson 1984(a))	66
5. Caries Prevalence Trends in Developing Countries, DMFT in Children Aged 12 Years (Source: Renson 1984(a))	68
6. Estimates from the Literature of Average Duration of Lesions in the Enamel Based on the Assumption that Duration of Time in any State Follows an Exponential Distribution (Source: Shwartz et al. 1984)	86

LIST OF FIGURES

<u>Figure</u>		<u>Page</u>
1.	Map of Rarotonga and the Cook Islands (Source: Survey Department, Rarotonga 1980)	10
2.	Monthly Return Form - January to August, 1976 (Source: Speake 1976)	21
3.	Mean Number of DMF Teeth per Subject/Age (Source: Hanson 1970)	26
4.	The Major Etiologic Factors Involved in the Dental Caries Process (Source: Newbrun 1983)	47
5.	pH Recordings at the Plaque-Enamel Interface of Upper Incisor Teeth Following a Glucose Rinse (Source: Stephan 1944)	50
6.	Observations on the Effects of Sugar Intake on Dental Caries Incidence (Source: Gustaffson 1953)	54

INTRODUCTION

Dentistry is concerned with the prevention and treatment of oral diseases and their sequelae. Many of the oral diseases can be prevented or controlled if proper measures are taken to minimise the known causative agents. The most widely known of these diseases is dental caries which is highly prevalent in our modern societies. Dental caries begins at an early age and affects most children. It is a chronic disease which, unless its progress is interrupted by treatment, eventually leads to the loss of the affected teeth. The prevalence of the disease is not affected by treatment since restorative dentistry treats the consequences of dental caries and not the cause. The prevalence of the disease can only be reduced by prevention.

The need for the prevention of dental caries must be obvious to all. Whether we consider the cost to the nation, the pain and misery to the sufferer, the loss in time to industry or the shortage of dentists; or whether we are interested only in the status of the dental profession, it is quite clear that we as a profession should be taking the problem of prevention much more seriously than we do at present.

There is little doubt that dental caries is primarily a local process occurring at the tooth surface and invading the tooth tissues. So far as can be shown, systemic factors can modify the tooth structure during development, but are most unlikely to cause any major change in enamel structure after eruption. It is also

known that other systemic factors may influence the process of caries through the saliva, but it seems most unlikely that they do more than slightly modify it. Thus the main factors involved in the production of caries are local.

In 1890 Miller described his experiments and gave his theory of the causation of caries and, today, we can still accept this theory though with some modifications. We believe that caries is caused by bacterial fermentation of refined carbohydrates in stagnation areas of plaque around the teeth, and that this fermentation produces the cariogenic agent which attacks the enamel. The nature of the cariogenic agent is not finally proven but is still thought of by many as an acid. The one big change to our views since Miller's day has been the increasing emphasis placed on the tooth structure.

From an ecological point of view, there are two major methods of preventing dental caries. The first is by increasing the resistance of tooth enamel to external attack. The second is to reduce the intensity of the attacking agents. The most important factors in promoting the resistance of teeth to dental caries are the systemic and topical application of fluoride. The most important factors in reducing the intensity of the attacking agents are restricting the frequency with which sugar in a sticky form is consumed, and the establishment of a regular and efficient system of tooth cleansing to reduce the accumulation of bacterial plaque on the surface of teeth

(Bunting 1935, Becks & Jensen 1948).

The need for additional methods of caries prevention is obvious. Improved knowledge on the details of the biological and clinical processes involved has strengthened the early belief in the dominating importance of food breakdown on the teeth in caries causation. Conversely, it has weakened the belief that the nutritional value of ingested foods has any measurable effect on caries. Diets that produce abundant caries when eaten normally do not cause caries if fed by stomach tube. The reason the caries attack is so little influenced by general nutrition or body physiology can be found in the fact that the tooth enamel is an essential inert substance that does not detectably reflect the changing states of the host. It seems that host influences can be brought to bear on the process of caries only through the saliva of which the best known effects are its ability to remineralize early caries lesions and supply absorbable proteins for pellicle formation. There is no evidence to show that these are influenced by body metabolism (Bibby 1978, Darling 1959).

The common belief that some teeth are highly resistant to dental decay and others highly susceptible lacks adequate proof (Bibby 1978). It seems that the developing dentition has first call on the body's supply of calcium and phosphorus and except in extreme conditions, the teeth are little affected by nutritional deficiencies. With the possible exception of the fluoride content, structural or chemical differences have not been shown to

exist between caries-resistant and caries-susceptible teeth. Thus, while the possible effects of tooth structure cannot be ignored, it seems that the human body is not capable of building a tooth that is resistant enough to withstand increasing strengths of caries attack to which it may be subjected in modern times.

From the foregoing it seems that the character of the food, of the mouth bacteria, of tooth structure or the saliva, all to some degree, influence the nature of the caries attack. It follows therefore, that progress towards caries prevention could be achieved by modifying any of these factors in the desired direction. The prospects are bright in the advanced western countries because of their use of fluoride in many ways. In the less advanced lands, the situation is worse. From dental examinations in several African countries it appears, as happened with the Polynesians and Eskimos (Bibby 1978) that an upsurge of caries is occurring among the people who have begun to use western foods. This problem of the high prevalence of dental caries is very common in the Cook Islands. Reports of oral health status surveys conducted in the Cook Islands since 1956 (Davies 1956, Hanson 1970, and Speake 1976) indicated that although data are not available for all islands, the indications are that in Rarotonga at least, dental caries constitutes the most prevalent of all diseases. Survey data published in 1970 showed that all school children were affected by the age of 13 years (Hanson 1970). On the average, it was found that school

children on Rarotonga at 14 years old had 10.4 decayed, missing or filled permanent teeth (DMFT). This caries explosion cannot be aborted by water fluoridation in the Cook Islands because the necessary piped water supplies or trained personnel do not exist.

The home use of fluoride dentifrices has expanded rapidly, and it is now a readily available preventive measure in many countries of the world. Similarly, topical fluoride application (operator applied) has also been shown to be very effective in increasing the caries resistance of sound fissures; and slowed or arrested the progression or development of any incipient lesions present (Craig 1975, Craig & Powell 1979).

Fluoride tablet programs have shown great efficacy when conducted in a well-controlled fashion, either on an individual or community basis; however such tablets are not always readily available and supplies are not constant in many developing countries (Driscoll 1974).

The opposition and slow implementation of fluoridation prompted the development of alternative approaches to the use of fluorides in developed countries, more specifically, the use of topical fluorides in the form of professionally applied topical application or group applied mouthrinses and toothpastes. Solutions of varying strengths and composition have been utilized to bring fluoride compounds into contact with the tooth surface.

At the present time the results of these approaches are being given considerable attention and credit for the

reduction in caries incidence being identified in certain developed countries. Whereas it is worth noting that, apart from water fluoridation, mass applied mouthrinsing for children has been the most readily implemented caries preventive programme in developing countries (Mellberg & Ripa 1983).

Preventive programmes in developed countries have utilized manufactured products such as toothpaste that are relatively expensive and professional and auxiliary dental manpower that may not be readily available in developing countries.

This treatise looks into a more recent development of topical fluoride therapy which has been developed by Professor G. Craig of the Preventive Dentistry Department, University of Sydney (Craig & Dunn 1971, Craig 1981). The therapy includes the application of 40% AgF solution to a carious lesion for 3 minutes, then, followed by 10% SnF₂ spotting paste.

A useful aspect of topical treatments with AgF followed by SnF₂ was that this combination turned the surface of active lesions black. Preliminary studies indicated that the presence of a continuous black mat on a lesion surface appeared to be associated with caries arrestment. Any lightening of the lesion was suggestive of caries development. This phenomenon was regarded as being of sufficient value that it was used as a guide to the status of the treated lesions (Craig, Powell & Cooper 1981).

The aim of the present treatise is to review the current literature of the studies and research that have been carried out on the use of silver and tin compounds for the arrestment of dental caries. Emphasis will be placed on determining the appropriateness of the use of the compounds and to determine areas of future development of the compounds.

It is considered that by reviewing the literature of the history of the silver compounds, by reviewing the studies carried out on caries progression and remineralization and by reviewing the appropriateness of the use of the compounds, appropriate conclusions can be drawn to suggest that such a preventive programme can be very effective on the Cook Islands. This is of special importance for the Northern Group islands of the Cook Islands where there is a very high incidence of dental caries, a large backlog of unmet treatment needs with limited dental personnel to provide the reparative and preventive services required.

In this presentation, the author presents some of the variations of use of silver compounds in the private clinics, hospital clinics and in school clinics noted while carrying out an opinion survey among dentists and dental therapists who are working in these clinics. The findings of this survey are summarized in the Appendix Section of this treatise.

AIMS OF TREATISE

1. To review the history of silver and tin compounds in the arrestment of carious lesions.
2. To determine the usefulness of silver and tin compounds in the arresting of dental caries for the Cook Islands.

2. REVIEW ORAL HEALTH STATUS

2.1 THE COOK ISLANDS - DEMOGRAPHY (Survey Dept - Rarotonga)

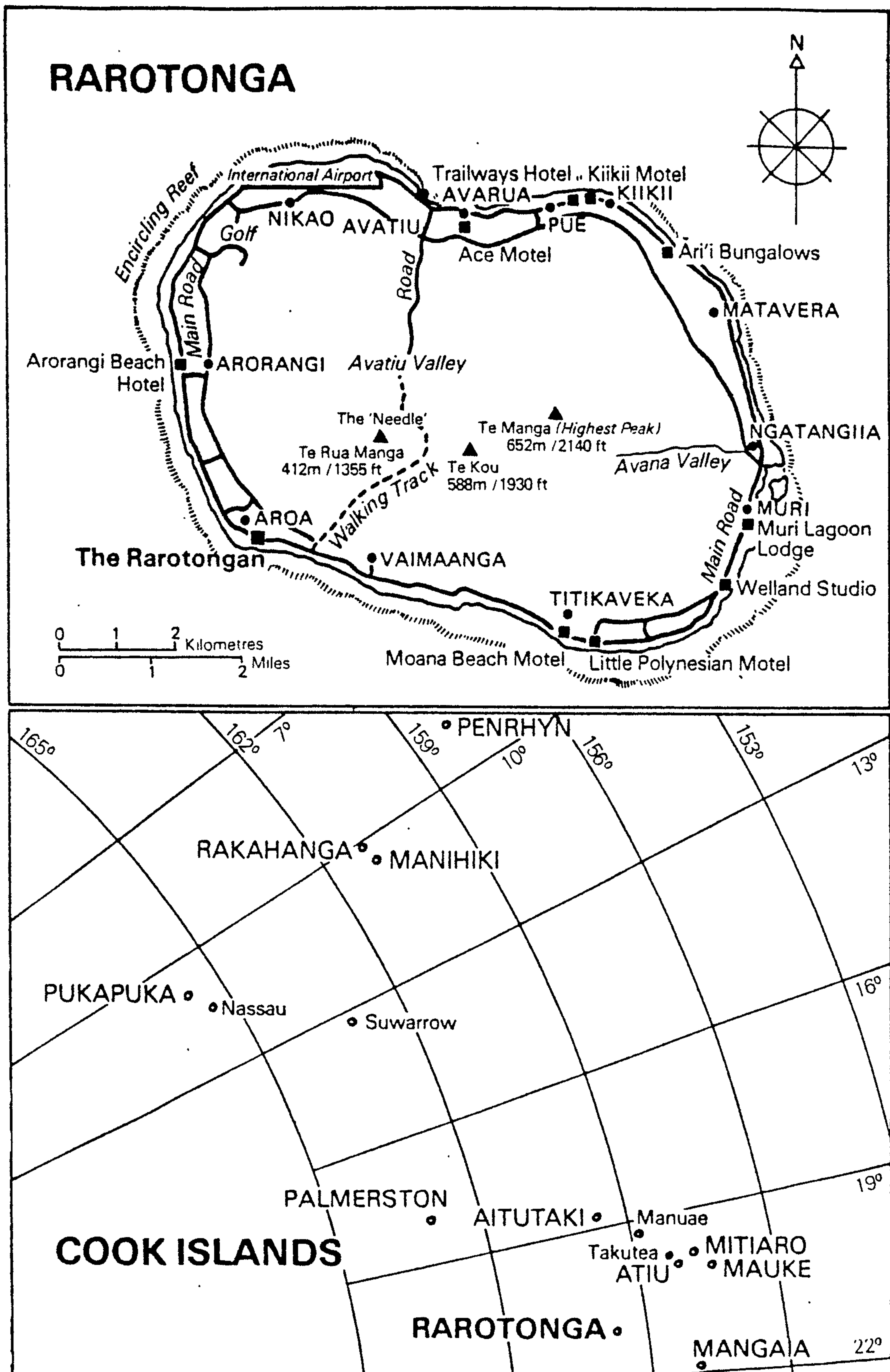
The fifteen islands that form the group known as the Cook Islands are spread over a very large area of the Pacific. Statutorily, the Cook Islands are defined as all the islands between 8 degrees and 23 degrees south latitude and 156 degrees and 167 degrees west longitude. This rectangle has an area of 751,000 square miles, but the total area of the islands within it is only 93 square miles, less than one hundredth of one per cent of the total.

Some 20,000 people, almost all of Polynesian origin, live on these scattered islands with half of the population living on the main island of Rarotonga.

On Rarotonga is the town of Avarua which is the capital of the island. Since 1965 the islands have been internally self-governing and an elected Assembly of 22 members meets annually in Rarotonga. A Cabinet with the Premier and five Ministers, responsible to this Assembly, governs the Islands.

The interests of New Zealand, the country responsible for the Defence and Foreign relations of the Cook Islands and the country that subsidises the financial income of the Islands are represented by a High Commissioner.

FIG. 1. MAP OF RAROTONGA AND THE COOK ISLANDS



(Source: Survey Department, Rarotonga 1980)

A brief note on each island of the group gives an idea of its diversity and of the communications and other problems faced by its Government.

RAROTONGA

Rarotonga is the largest, highest and most populous island of the group. In many ways it is also the most beautiful. Strangely enough it was also one of the last islands to be discovered by European navigators.

Rarotonga has an area of 17,000 acres. Te Manga, the highest peak reaches a height of 2140 feet.

Its 10,000 inhabitants live mainly on the coastal plain which stretches from the hills to the beach. Beyond this plain is a lagoon lying between the beach and the fringing reef. In places this lagoon is wide and deep, ideal for swimming and canoe sailing, but in others it is shallow and full of large coral rocks.

The island produces citrus fruit, bananas, tomatoes, pineapples and other tropical fruits. Fruit is both shipped to the New Zealand market and processed in a large modern cannery in Rarotonga.

The island has other secondary industries; two clothing factories and two factories producing curios.

From Rarotonga there has been considerable migration to New Zealand and the Cook Islands population in New Zealand is rapidly approaching that remaining in the Cook Islands.

The migration from Rarotonga does not reduce the population. A high birth rate plus considerable migration from outer islands to Rarotonga keeps the population on the increase.

AITUTAKI

Captain Bligh discovered the island of Aitutaki in 1789 just before the famous mutiny took place on his vessel, the 'Bounty'.

Aitutaki is part volcanic and part atoll. It has a large and beautiful lagoon, as well as hills, and over 2,000 acres of fertile soil.

The population of 2,500 is largely self supporting with a subsistence economy, a bountiful supply of fish, other sea foods, kumara, taro, arrowroot and other crops. At the same time they export oranges, bananas and tomatoes.

Aitutaki has two airstrips which were built by New Zealand workmen for the U.S. armed forces during World War II. A large U.S. force was stationed in Aitutaki at that time. Aitutaki was a stopping point on the coral route from New Zealand to Tahiti, until flying boats went out of service.

MANGAIA

Mangaia, discovered by Captain Cook in 1777, is the second largest island of the group and is also the closest to Rarotonga. Mangaia has a hilly inland reaching a height

of 554 feet. This inland part comes down to plains at almost sea level but this whole island is surrounded by a huge raised coral wall or Makatea. The Makatea reaches 230 feet in height and is in places over a mile wide.

The Makatea is riddled with huge caves and the whole island is of great geographical interest.

The 2,000 people who live on the island enjoy an almost complete subsistence agriculture and for a cash crop grow pineapples, most of which are processed in the cannery at Rarotonga.

Traditionally united are the islands jointly referred to as Nga-Pu-Toru. These are the inhabited islands of Atiu, Mauke and Mitiaro.

ATIU

Atiu with a population of 1,300 rises only to a height of 230 feet. The main village is on the top of the island. It was discovered by Captain Cook in 1777 and a very full account of the island and its people followed his visit.

The people of Atiu are very industrious and have in recent years made a spectacular change in the appearance of their village. Small modern concrete houses have replaced the older type of inadequate housing. The island has good plantations of citrus fruit which is shipped to the canning factory at Rarotonga.

MAUKE

With a population of 700, Mauke is a similar island to Atiu rising to 85 feet. It was discovered by Rev. John Williams in 1823.

The island produces copra and has tried many crops. Citrus, peanuts, ginger and livestock production have all been the subjects of Government directed agricultural experiments in recent years. Expansion of livestock industry is probable and new efforts are being made to develop the citrus industry there.

MITIARO

One of the poorest islands in the group today is Mitiaro. Its 300 inhabitants have practically no cash income.

The majority of its young people migrate to Rarotonga for the final stages of their education and to obtain work. Many have later gone from there to New Zealand.

The island is a low one with a large lake.

PALMERSTON ISLAND

Palmerston island is an atoll situated about 250 miles North-West of Raotonga. Its 86 inhabitants are descendants of an Englishman, William Marsters, who settled the island in 1862.

THE NORTHERN GROUP ISLANDS

Far to the north of the islands previously described lie the islands of the Northern Group. These are Manihiki, Rakahanga, Penrhyn, Pukapuka, Nassau and Suvarrow. All are low islands and all except Nassau are atolls.

MANIHIKI

Manihiki is best known for the high quality pearl-shell produced there. Its 600 people obtain an income from this pearlshell trade and from the making of copra.

Only 26 miles from it lies RAKAHANGA, a smaller atoll which supports just under 400 people. Its only cash income is from copra.

The islands of MANIHIKI and RAKAHANGA have always worked together. There has been frequent sailing of the short distance between them and in the past the whole population would leave their village in Rakahanga and stay in Manihiki for a few months.

Presumably the short voyage has often been a dangerous one. During the last 25 years on three occasions boats have been lost when attempting to make the 26 mile crossing. One with seven people was never seen again. Another finished its journey in Pukapuka and a third sailed to the New Hebrides in an epic and tragic small boat journey described in the book "The Man who Refused to Die".

Rakahanga was the landfall of the famous raft

Tahiti-Nui III after its crossing of the Pacific. Its famous captain, Eric de Bishop, died on the reef in Rakahanga on landing. This trip is described in the book "From Raft to Raft".

Biggest of all the Northern Group atolls is PENRHYN. Penrhyn surrounds a very large lagoon and has a 10,000 feet airstrip which was built during World War II when the island was an American base.

Penrhyn's 550 people also depend on copra although some shell is also exported. Penrhyn is the most distant island from Rarotonga, being over 850 statute miles to the north.

Right at the western extremity of the group lie PUKAPUKA and NASSAU. The small island of Nassau is worked for copra by the Pukapukans. Together the two islands have a population of 850. Copra gives these islands their only income.

The whole population of Pukapuka works on a communal basis, to a much greater extent than on the other islands of the group.

SUWARROW

A large atoll with little land, has had a strange history. It has not been permanently inhabited but has frequently been visited by pearl shell divers. Murder and piracy are part of Suvarrow's story as are rumours of buried treasure.

In recent years it has served as a hermit hideout. Alternatively New Zealander Tom Neale, and Englishman, Michael Swift, have lived there as hermits. Currently it is inhabited by Tom Neale whose story can be read in the book "An Island to Oneself".

It is believed that at least two of the Northern Group islands were visited by the early Spanish navigators, Mendana and Quiros in 1595 and 1606. But reports as to which islands were visited are somewhat vague due to the limited accuracy of navigation at that period and similarity in appearance of all small atolls.

2.2 DENTAL SERVICES IN THE COOK ISLANDS

The Dental Services in the Cook Islands are staffed by nine Dental Officers, who are Graduates of the Fiji School of Medicine and seven New Zealand trained school dental nurses. The first Australian trained dental therapist graduated from Tasmania in 1983 and is now in the service in the Cook Islands while a second therapist completed her training at the end of 1985.

There are two technicians and six locally trained hygienists whose instructions have included some elements of health education, prophylaxis and the extraction of deciduous teeth as well as chairside assisting. In addition to these hygienists, there are five chairside assistants who do not only assist the dentists but provide health education instructions. Overall, a total of twenty six persons are employed by the Dental Service giving an

overall national patient to operator ratio of 1277:1.

Five of the Dental Officers are stationed in Rarotonga and the other four are posted each to one of the islands of the Southern Group; namely Aitutaki, Mangaia, Atiu and Mauke.

Six Dental Nurses attend to all the pre-school and school children of Rarotonga. They are responsible for the conservative treatment of these children. When a more complicated case is encountered in the schools, the patient is referred to the Main Clinic where a Dental Officer sees the patient. The Dental Officers discuss the case so that the best treatment plan can be offered to the patient.

In Aitutaki, there is one Dental Officer and one Dental Nurse responsible for the population.

In the Northern Group, there are no resident Dentists on the islands. A Dental Officer from Rarotonga makes a visit to any of these islands, after a request has been approved by Cabinet following a submission through the Cabinet Minister responsible for that island. The visiting Dental Officer is primarily concerned with conservative treatment of the pre-school and school children. If the period of stay on any island is extended, the Dentist allocates his time to conservative work on the children in the mornings, and in the afternoon, to extraction and prosthetic work for the adult population. The conservative work on the adult population is done only after the children are completed, and, if there is sufficient time before the boat arrives for his return to Rarotonga. When a Technician

accompanies the Dental Officer, all the prosthetic work is undertaken by the Technician, thereby allowing the Dental Officer to concentrate on restorative work for both the children and adult population. To date, this has been the only possible manner of presenting dental services to the people of the Northern Group, but for long term maintenance of dental health, it is accepted that it leaves much to be desired.

In the absence of a Dental Officer on any of these islands, extractions to relieve pain and infection, are done by the resident Medical Dresser or Doctor.

At present in the Cook Islands the presentation of Dental Services is not well balanced. In Rarotonga there is a concentration of Dental personnel, in the Southern Group where there are permanent Dental Officers, but the Northern Group is not so favourably placed.

Because the Cook Islands Group encompasses such a vast area, distances will always impose great difficulties upon the Dental Health Services. If the aim is to preserve the dental health in the whole population, then because of the area to be covered and the limitations imposed by manpower and economics, dental education and prevention appear to offer the best solution.

There is one main clinic on Rarotonga where most adult patients are seen. The treatment provided ranges from conservative work to prosthetic, endodontic, extractions and preventive treatment. Referral patients from

school clinics on Rarotonga are treated at this clinic.

In addition to the main clinic on Rarotonga, there are eight other clinics situated in schools on the island. There is one mobile clinic which treats mostly pre-schools. There is a total of eight clinics on the outer islands and these are situated mainly in schools.

Equipment used on Rarotonga is of reasonable standard especially after recent completion of the installation of more modern chairs and units.

At the end of each month, a monthly report is submitted by dental officers and dental nurses of the services carried out. An example of such a report is made available from a summary of all monthly reports submitted by dental officers and dental nurses of the service for the period January to August 1976 (Figure 2, page 21). Although there are certain dangers inherent in trying to read too much into such activity reports, certain general conclusions may be drawn from the monthly report document (Figure 2, Speake 1976).

1. ADULTS

A fairly wide range of treatment is provided for adults on demand; including amalgam, gold and synthetic fillings, pulp treatments, oral surgery and acrylic dentures. Services are concentrated to extractions and fillings rather than to preventive procedures. The ratio of extractions to fillings during the eight month period was 1.2 to 1 and some 285 full and partial dentures were

(Source: Speake 1976)

constructed. Amongst this group, payment for services are on a fee for treatment basis.

2. CHILDREN

The ratio of permanent extractions to fillings is much more encouraging at 0.078 to 1 amongst this group, which is covered by the free School Dental Service. There is evidence that in recent years the average number of extractions per patient has been rising in the Cook Islands School Dental Service, from 0.3 during the period 1969-72 to 0.4 in 1973 and to 0.5 in 1974. A similar figure of 0.5 is apparent for the first eight months of 1976. During this same period, some 3859 were examined and of these 3227 or 84% had been completed. However, despite the space provided on the monthly reporting form, no record had been made of any preventive activity including dental health education. It would therefore seem reasonable to conclude that the emphasis in the School's Service is on the detection and treatment of caries through fillings but that in recent years there has been a slight increase in the average number of teeth extracted per patient.

2.3 ORAL HEALTH STATUS IN THE COOK ISLANDS

Reports of oral health status surveys conducted in the Cook Islands since 1956 (Davies 1956, Hanson 1970, Speake 1976) indicate that, although data are not available for all islands, the indications are that in Rarotonga at least, dental caries constitutes the most prevalent of all

diseases. Survey data published by Hanson in 1970 showed that all school children were affected by the age of 13 years. On Mangaia 89.6% of 13 year-olds were similarly affected. On an individual basis, it was found that, on the average, school children on Rarotonga had 10.4 decayed, missing or filled permanent teeth (DMFT) by 14 years of age. It must be stressed that although this information was published in 1970, the actual data were collected some years earlier and should therefore now be regarded as being about fifteen years out-of-date.

The DMFT data collected by J.D. Speake during his survey (October 1976) is limited to Rarotonga and confined to eight and eleven-year-olds. When comparisons are made between the same age groups in the pre-1970 survey and the 1976 samples, an increase in dental caries rates which is statistically significant can only be demonstrated between the two eight-year-old age groups (see Table 1).

TABLE 1. MEAN DMFT OF RAROTONGA - PRE-1970 and 1976

	<u>RAROTONGA</u>	
	MEAN DMFT PRE-1970	MEAN DMFT 1976
8 years	1.7 ± 2.05 (± SD)	2.7 ± 1.7
11 years	5.7 ± 2.56	5.9 ± 4.1

(Source: Speake 1976)

The difference between the two 11 year old groups is not statistically significant. Such a comparison is based on the assumption that the same criteria were used for the diagnosis of caries in both the pre-1970 and 1976 studies.

NOTE: The data published in 1970 gave means and standard errors which have been converted back by the writer to standard deviations to match 1976 data.

There can be little doubt that the caries rates in the Cook Islands are high. This is particularly so in Rarotonga and probably Aitutaki which are the two main concentrations of population. The situation in the outer islands remains obscure because of the lack of survey data. The usual pattern in the Pacific has been for the amount of caries to decrease with distance from the main centres and it is believed that this is related directly to access to imported refined foodstuffs.

There are indications that caries rates have risen during the past ten years. These are shown by:

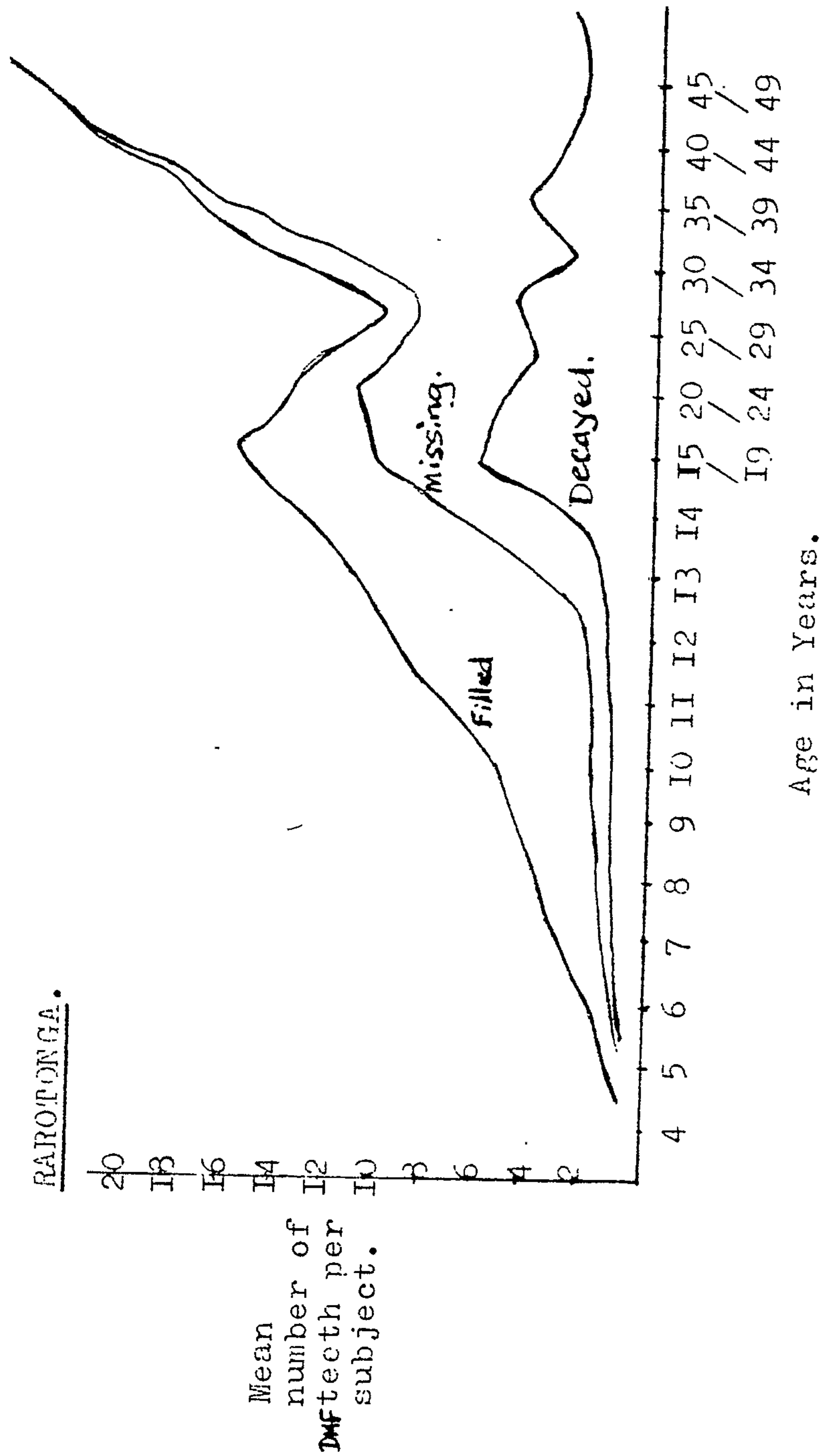
1. The increase in consumption of cariogenic foodstuffs
(See Table 2 on Family Budget Survey, page 27.
Source: Speake 1976).
2. The increase in caries rates amongst eight year old school children.
3. The increase in the average number of extractions per patient in the School Dental Service.

There are numerous approaches to the problem of dental caries. The first is the one already being employed by the Cook Islands Dental Service, which is the detection of caries and the restoration of teeth (fillings) through regular inspection and treatment in the schools. Such a service already exists in Rarotonga, where the school dental nurses are based, and to a certain extent in other islands with dental personnel. Visits to islands without dental personnel are, however, very infrequent. The disadvantages of this restorative approach by itself, particularly in high caries situations, are that benefits of treatment do not last long even under regular care. New fillings are required and old fillings break down and need replacement. By the time the individual reaches late teens or early adulthood little advantage has been gained.

Figure 3 (page 26) on Mean Number of DMF teeth per subject/age in years which has been taken from the pre-1970 survey (Hanson 1970) illustrated what happens on Rarotonga. There was a rapid increase in the number of missing teeth from the age of approximately 20 years onwards.

The second approach to the caries problem is through prevention measures; that is, taking steps to increase the resistance of the teeth to decay. In practical terms, this means increasing the fluoride content of the enamel and this may be achieved by fluoridating the water supply where practical and by using topical fluoride on an individual or group basis.

FIG. 3. MEAN NUMBER OF DMF TEETH PER SUBJECT/AGE



(Source: Hanson 1970)

Another approach to the caries problem in the Cook Islands is by reducing the sugar intake in the diet (Abraham 1947, Faine & Hercus 1951). Again, the high consumption of sugar in the Cook Islands in the last ten years is clearly shown by Table 2 taken from the Family Budget Survey (Speake 1976).

TABLE 2. FAMILY BUDGET SAMPLE SURVEY - COOK ISLANDS

ITEM: DRINKS, SWEETS, ETC. (By weight)

	1967	1976	CHANGE BY WEIGHT	% CHANGE
Sweet item	.45	1.49	+ 1.04	+ 231%
Chewing gum	.05	.3	+ .25	+ 500%
Lemonade tinned	0	1.51	+ 1.51	+ 00%
Ice cream	.47	8.9	+ 8.43	+ 1793%
Orange Juice tinned	.02	2.2	+ 2.18	+ 1090%
White sugar	2.2	2.43	+ 0.23	+ 10.45%

(Source: Speake 1976)

2.4 FACTORS INFLUENCING CHANGES IN ORAL HEALTH STATUS

2.4.1 Changes in Food Consumption Patterns

A report of a Family Budget Sample Survey in 1976 (Table 2, page 27 - Speake 1976) was compared with the results of a similar survey conducted nine years previously. It is apparent that there have been marked increases in the consumption of many potentially cariogenic foodstuffs.

2.4.2 Water Supply

The present reticulated water supply system on Rarotonga employs a ring main which runs around the island and which is fed by a total of seven sources located at Avatiu, Takuvaine, Turangi, Avana, Titikaveka, Papua and Arorangi. Plans are however in hand to reduce the number of sources to five (Avatiu, Takuvaine, Turangi, Avana and Papua) and to introduce chlorination plants located at easily accessible places along the feeding mains down from the intakes. However, at the time of the presentation of this treatise, no changes have taken place to the water supply system on Rarotonga as planned.

Typical analysis regularly undertaken by the government analyst in Auckland, New Zealand, since 1970 have shown that one source (Avatiu) produces water with a fluoride content of 0.2 parts per million fluoride while the others contain 0.1 ppm fluoride. Neither of these concentrations is sufficient to afford any appreciable protection against dental caries (Videnov 1975).

2.4.3 The Education System

Education between the ages of 6 and 16 is compulsory and therefore virtually universal in the Cook Islands. This is of great advantage to the Dental Services in its efforts to provide services because it allows ready access to the children from the time when their first molar teeth erupt right through to the eruption of the second and in some cases third permanent molars. Particularly therefore in Rarotonga, and perhaps to a lesser extent in some outer islands, the opportunity exists not only to provide restorative services but also to strengthen the teeth against dental caries.

Information supplied by the Education Department (Speake 1976) showed that there were approximately 6488 children attending school in the Cook Islands, in 1976. Of these, 3373 (52%) were attending schools in Rarotonga whilst the remainder - 3115 (48%) were located in schools on the outer islands.

If we assume that there are approximately nine months during the year when children are at school and available for dental attention, it would seem probable that the Dental Service working at its present pace (Speake 1976) would expect to complete about 3630 children, which amounts to about 60% of the total school population in one year.

3. TIN AND SILVER COMPOUNDS - HISTORY

3.1 HISTORY

In dentistry, tin has been extensively studied because of its widespread use as stannous fluoride (SnF_2) as a topical fluoride agent. However, it is not clear whether tin (Sn) has any effect on caries by itself, as a separate entity from the effect of fluoride.

The binding of tin to enamel can be considerable. Meckel (1965) suggested that this was due to uptake in "enriched" areas of enamel. The study by Hoerman, Klima and Birho (1966) showed that tin could be released slowly with time. This slow release of the tin ions could have an inhibitory effect on oral bacteria. The studies by Attramadal and Svatum (1980) showed that SnF_2 as a mouth rinse inhibits acid formation in plaque and also lessens the formation of plaque.

Compounds of silver have been used in dental therapy for more than a century. The attention of the profession has, for the most part, centred upon silver amalgam, then silver nitrate, and more recently silver fluoride. The value of silver nitrate as an aid in controlling caries was recognized as early as 1846 (Der Zahnartz 1846 - cited by Seltzer & Werther 1941), although very little study was given to this important property until late in the nineteenth century and early in the twentieth. Clinical observations following treatment with silver nitrate were first reported by Stebbins (1891) and

Frank (1891), according to Miller (1905), were the first to recommend the use of silver nitrate prophylactically; and Bryan (1907) pointed out its value in preventive dentistry.

Before the twentieth century, silver nitrate was firmly entrenched in the profession as a remedy for hypersensitivity of dentin, erosion and pyorrhoea and as a sterilizing agent and caries inhibitor in deciduous as well as permanent teeth (Seltzer & Werther 1941).

Up to this time, silver nitrate was employed empirically in whatever concentrations and by whatever method the particular author recommended. Seltzer and Werther (1941) reported in their summary that the compound was used: in powdered form pressed into place by gutta-percha (Holmes 1892); in powdered form applied with wood points (Stebbins 1891); in 50 per cent solution (Hinman 1892); in saturated solution (Parker 1894, Arrington 1893); in pure crystal form (Prinz 1905); by inserting a silver wire in nitric acid (Holmes 1892); with a blotting pad saturated with silver nitrate. Pierce (1894) later modified to an asbestos felt pad. Szabo (1902), in the first scientific investigation of silver nitrate solutions, clearly showed that the results were the same regardless of the concentration or method used.

In 1905 Miller showed experimentally that silver nitrate would protect the dentin from decalcification and laid down the chemotherapeutic principles of its action. He noticed that the precipitates of silver in the super-

layers of the dentin may form a barrier more or less impermeable to acids. He stated that it may be the coagulation of the contents of the dentinal tubules which protected the dentin against the action of acids.

J.M. Howe (1900) sounded a warning note about the likelihood of pulp irritation following the use of silver nitrate and, in 1906, G.V. Black further cautioned the profession against the use of silver nitrate in deep cavities. In the same publication, it was pointed out that silver nitrate should be reduced by exposure to sunlight.

When Percy R. Howe brought his ammoniacal solution of silver nitrate before the profession, in 1917, a renaissance in the clinical use of silver nitrate began. Howe's solution consisted of silver nitrate crystals dissolved in distilled water, to which was added ammonia water, which threw down a black precipitate of silver oxide. This precipitate disappeared when excess ammonia was added. Howe recommended this solution for the sterilization of the disintegrated dentin overlying pulps, as in large cavities of carious first molars. He reduced the silver from the ammoniated solution with formalin. Howe stated that his solution penetrated all affected dentin, but did not penetrate the sound tissue of the teeth. The use of Howe's silver nitrate solution was indicated in small fissure cavities in posterior deciduous teeth, as well as permanent teeth, where the enamel is not undermined with decay. It was also recommended in small fissure

and approximal cavities in deciduous teeth, instead of fillings by Nesson (1924). First, cut away the approximal decay and smooth with sandpaper discs - then apply the Howe's solution. The silver nitrate reduction process was also indicated in all deep cavities in posterior teeth. All root-canal treatments in posterior teeth call for the silver nitrate reduction process.

Prime (1935) discussed most of the phases of silver nitrate from the clinical standpoint but stressed its value in the treatment of incipient caries and its use as a disclosing solution. He showed the lack of value when the reducing agent was applied first.

Ireland (1939) suggested that since silver nitrate was such a good sterilizing agent of infected dentin, it could be left thereby reducing the incidence of carious pulpal exposure.

Seltzer and Werther (1941) in their studies showed that silver nitrate cannot be held responsible for pulp devitalization. It aided in the retention of the tooth by giving pulp tissue time to lay down secondary dentine.

Graham (1941) presented the value of silver nitrate by its ability to sterilize disintegrating dentinal structure and to neutralize the acid reaction of dental caries. He also referred to "Howe's Solution" of silver nitrate being an alkaline solution (consisting of a saturated aqueous solution of silver nitrate to which was added strong ammonia water (37%) and a 25 per cent solution of formalin as a reducing agent); differing from the

ordinary silver nitrate solution. Silver nitrate, in its ordinary state, is an acid salt. Graham (1941) noticed that the sterilization resulted from the action of the metallic silver upon the proteins of the bacteria in the decayed tooth structure, which are precipitated and thus prevented from further action. Neutralization of the acid reaction of dental caries was accomplished by the alkaline reaction of Howe's solution, having a pH of 8.5 to 9.5.

Graham's technique of application was very simple and was the same in deciduous and permanent teeth. His treatment programme with Howe's solution in his practice (Graham 1941) was presented as follows:

"Let us take for example a large, deep cavity in a lower first permanent molar in a patient 12 years of age. As yet, the patient has had no ache in this particular tooth and, on the use of excavators and large round burs for the removal of the carious material, we found that we were rapidly approaching the pulp. If continued removal of decay would expose the pulp, it would be advisable to take a roentgenogram of the tooth to determine the condition of the peridental membrane at the apex of the roots, and also the proximity of the caries to the pulp. If the roentgenogram showed no involvement of the root end, then in this particular case the following treatment is indicated. The area is blocked off with cotton rolls and the cavity dried completely with pellets of absorbent cotton and warm air.

When the cavity had been completely dried, Howe's solution was introduced into it by means of a pellet of cotton or a platinum loop, and the entire base and side walls of the cavity were saturated. The solution was allowed to penetrate for from one to two minutes and then the excess solution was removed with a dry cotton pellet. Immediately, a cotton pellet saturated with eugenol (U.S.P.) was applied to the cavity, and in from thirty to fifty seconds a dark stain would appear at various points in the cavity. The eugenol was used as a reducing agent for the ammoniacal silver nitrate solution and the reducing

action was permitted to continue from one to one and onehalf minutes.

Upon again drying the cavity, we found certain spots where there was an intense black stain and other spots where there seemed to have been no reaction. This was explained by the fact that some carious tooth structure which had no mineral content, was left intentionally in the base of the cavity. The silver nitrate solution had penetrated this particular material to its base, and when the reducing action of eugenol was introduced, all the dentinal tubules in the carious structure were literally filled with myriads of microscopic silver fillings. Where there was no dark stain, no penetration had resulted; which proved that Howe's Solution was self-limiting in its penetration action. This self-limiting penetrative power was best explained by the fact that in normal, healthy, sound dentin, each tubule contains a tiny fibril which was a protein. In as much as proteins were precipitated by the salts of heavy metals, we found that penetration of the silver nitrate solution to the depth where a vital fibril was encountered had produced a silver proteinate.

After treatment in the foregoing manner, it was usually advisable to seal the cavity with either temporary stopping or cement for two or three days to allow the reduction of the silver nitrate solution to become complete.

At the next visit, a large round bur was used to remove the upper and more superficial portions of black stained tooth structure in the base of the cavity. Contact with the bur would show that the previously carious material had assumed a hardness and resistance to cutting entirely different from that found at the previous sitting. If penetration and reduction had been properly carried out, the cavity will exhibit little or no sensitiveness to cutting by the bur. After the softer and looser portions of the stained area had been removed, the cavity was again blocked off with cotton rolls and thoroughly dried, and another application of silver nitrate solution was made and reduced with eugenol. As a rule, precipitation of the solution at this sitting would result in a more or less "mirror effect" in the area; which indicated complete and total saturation of the previously decayed tooth structure with metallic silver. When this point was reached, examination with an explorer would reveal that the area was now hard and firm and that the pulp was no longer sensitive to

thermal changes such as result from the blast from an air syringe."

The technique just outlined was particularly applicable to deciduous teeth presenting large cavities in which any attempt to remove the decay almost invariably resulted in pulp exposure. The "mirror effect" was necessary for the best results from this treatment.

Zander (1941) presented an excellent paper on the history of the scientific data and research on silver and he concluded that:

1. Silver nitrate should be used routinely in all cavities where appearance was not important.
2. Silver nitrate should not be depended upon to prevent an exposure, and that, all caries should be removed.
3. The use of silver nitrate as a disclosing agent for superficial decalcifications of enamel was of great value; apparently it arrested caries for some time.

Zander and Smith (1945) found that silver nitrate penetrated sound dentine - maybe carried by a tissue fluid after penetration. This further penetration does not injure the pulp nor irritate it.

Despite the many enthusiastic claims that had been made for ammoniacal silver nitrate as a sterilizing agent and caries inhibitor, a certain amount of antipathy had existed in the dental profession towards this medicament, because:

1. the use of silver nitrate in deep cavities was often painful;
2. it discolours the tooth, and
3. there was danger of devitalization (Ireland 1939).

However, it was believed that ammoniacal silver nitrate, when judiciously applied could be the means of saving many doubtful teeth that were on the borderline between pulp conservation and pulp exposure; in other words, cases in which the pulp would most certainly be exposed if all carious material were excavated (Graham 1941).

This method of treatment was in direct violation of Black's fundamental principle of operative dentistry, which stated that all carious dentine should be removed from a cavity before a filling is inserted, but past experience had shown that the foregoing treatment is the exception that proved that rule (Graham 1941).

Meticulous care should be exercised in using silver nitrate solution in the mouth because contact with the membranes would produce serious burns (eschars) (Graham 1941). Should an eschar occur, a saturated solution of sodium chloride, or table salt, should be immediately applied to the injured parts, followed by the application of oil of eucalyptus.

Should some of Howe's solution be accidentally spilled on the hand or clothing, a stain could be prevented by immediate application of a saturated solution of potassium iodide (Graham 1941).

Prime (1935) in his article had quoted the following:

"Percy Howe's Solution was more than a contribution to dental literature; it was a stroke of genius. The profession has been slow in recognizing its value. If we may judge by our experience, it will mean the saving of millions of teeth which, without it, would be lost. It will mean health for thousands of people otherwise doomed to sickness."

3.2 THE EFFECT OF HEAVY METALS ON DENTAL CARIES

Caries is not an irreversible process. It can be arrested. For decay to occur sucrose has to diffuse through plaque to the micro-organism living in and under the plaque. Acid end products must diffuse from micro-organism to the tooth surface. End products of demineralization, i.e. Ca and PO_4 ions must diffuse out before caries can occur. If the diffusion process is stopped caries is stopped.

The heavy metals may form a diffusion barrier in the plaque by precipitating proteins from micro-organisms and also by being very strong anti-microbial agents. They react by cross-linking with plaque constituents, e.g. carboxyls and sulphydil ions ($-\text{COOH}$, $-\text{SH}$) (Hoerman 1966, Leverett, McHugh & Jensen 1981). Experiments with toluene dye show that there is a barrier formed (Leverett et al. 1981).

Fluoride is needed in the plaque mass to precipitate Ca and PO_4 ions. The end product would be a calcified plaque mass almost like calculus.

3.3 THE EFFECT OF TIN (Sn) ON DENTAL CARIES

In dentistry, tin (Sn) has been extensively studied because of its widespread use as SnF_2 as a topical fluoride agent. However, it is not clear whether tin has any effect on caries by itself, as a separate entity from the effect of fluoride. Muhler, Spear, Bixler and Stookey (1967) showed that stannous chloride (SnCl_2) did not reduce caries but that stannous fluoride (SnF_2) was better than sodium fluoride (NaF) in inhibiting caries. This effect was greater than would be expected from the fluoride alone.

Binding of tin (Sn) to enamel can be considerable; it was suggested by Meckel (1965) as due to uptake in "enriched" areas of enamel. Other studies showed that tin could be slowly released with time (Hoerman et al. 1966). Such a slow release of tin ions (Sn^{++}) could have an inhibitory effect on oral bacteria. Lilienthal (1956) noted that stannous fluoride inhibited acid production in vitro more than sodium fluoride, proportional to the tin content rather than to the fluoride content.

Further studies have shown stannous fluoride, as a mouth rinse, inhibits acid formation in plaque (Attramadal & Svatum 1980); and also lessens the formation of plaque (Attramadal et al. 1980). The major effect was thought to be due to tin. Bacteria such as *S. mutans* show a rapid uptake of tin that has been shown to be greater than other metal cations (Attramadal et al. 1980). The presence of tin ions may have an inhibitory effect on growth rate

and metabolism of bacteria which could account for the indicated effect of tin on caries.

A study on the effect of tin has shown that a 0.4% stannous fluoride (SnF_2) experimental toothpaste significantly reduced both plaque and gingivitis (Bay & Rolla 1980). However, in longer term trials the plaque inhibition effect of stannous fluoride has been shown by Leverett et al. (1981) to be only of short duration (less than four months). It has also been demonstrated, in a preliminary report, that prerinsing with metal ions, zinc (Zn) and tin (Sn), reduced the antiplaque effect of chlorohexidine (Wales & Rolla 1980).

The dental literature on tin is much more extensive because of the many studies that have been carried out on stannous fluoride. However, data on the effect of tin itself are still limited, although there has been considerable interest recently. If this trend continues, such valuable information will be available on the relationship of tin to dental disease.

3.4 THE EFFECT OF SILVER (Ag) ON DENTAL CARIES

Silver is a major restorative material in dentistry through its role in silver amalgam. The dental literature contains an extensive list of references on the use of this metal for fillings. However, silver has been used as a possible anticaries agent for a long time as silver nitrate (AgNO_3). For many years, silver nitrate solutions were used on carious dentin, particularly in deciduous teeth,

to arrest or prevent dental decay. Studies to demonstrate this effect were carried out by Klein and Knutson (1942). Kuroiwa (1979) has suggested the use of ammonium silver fluoride for the prevention of caries. There is a lack of in vivo studies done on the effect of silver on dental caries.

Recently, studies on the therapeutic effects of silver in dentistry have concentrated on antibacterial and cytotoxic actions. Sterilization of carious dentin had been proposed by Rose (1976) by using silver nitrate; Lan (1977) used ammoniacal silver nitrate to sterilize root canals. Another approach to the use of silver for sterilization has been to use the generation of silver ions by low intensity direct current (Thibodeau, Handelman & Marquis 1978). This has the effect of inhibiting or killing of oral bacteria, and has been tried with some success for sterilizing root canals. Leirskar (1974) showed that silver and copper amalgams, by the release of free ions, were cytotoxic in cell cultures.

3.5 THE EFFECT OF FLUORIDE ON REMINERALIZATION

The majority of experimental work on the remineralization of acid damaged enamel has been assessed by changes in the microhardness of surface enamel. In such studies, the original enamel surface is ground and polished flat so that surface hardness measurements can be carried out (Lenz 1968). Such studies concentrate on the softening and subsequent rehardening of the outer 5 microns of the enamel

surface. Since the small carious lesions in human enamel is positioned deep to an intact and well-mineralized surface layer which is approximately 30 microns in depth, it may be difficult to extrapolate results of surface microhardness studies to the mechanisms involved in initiation and repair of the small enamel lesion.

Previous histological studies on the small enamel lesion and its remineralization in vitro have been well documented (Silverstone 1973). One factor common to many of the results obtained on the remineralization of either enamel caries, artificial caries, or acid etched enamel is that the presence of fluoride ions enhances greatly the degree of remineralization achieved, and reduces the time period for this mechanism to occur (Frank 1968).

3.6 REMINERALIZATION

Remineralization occurs naturally during the formation of a carious lesion in human enamel. Backer-Dirks (1966) and other authors (Darling 1968, Wei Shy 1967) have recorded reversals in clinical diagnosis whereby lesions detected clinically and by bitewing radiography apparently disappeared when examined at a later date. Experimental studies have shown that acid-softened enamel surfaces can reharder in vitro after the application of a calcifying fluid to bring about remineralization (Koulourides, Feagin & Pigman 1968). Exposure of small artificial caries-like lesions to a calcifying fluid in vitro produced a significant degree of remineralization in a

study by Silverstone (1977). After his experiment, lesions showed histological features which indicated that they were at a much earlier stage in development than prior to exposure to the calcifying fluid.

Koulourides and co-workers (1968) made a major contribution to support the contention that acid-softened enamel surfaces can be rehardened. Although such rehardening experiments are believed to be comparable to remineralization, modification of an outer softened layer of enamel at the surface differs from the situation found in the intact surface of a carious lesion. In the carious lesion, the outer layer of enamel, to a depth of approximately 30 microns, appears not only intact, but also relatively unaffected by the attack compared to the immediate subsurface region. This layer is the surface zone of the enamel lesion and it is through this well calcified barrier that remineralization of the enamel lesion occurs.

Other experiments on the remineralization in vitro of both natural and artificial lesions have shown that both types of lesions can be reduced in size and porosity (Silverstone 1977). This occurred by the deposition of mineral into the damaged enamel after exposure to a calcifying fluid. Recent work (Silverstone 1982 & 1983) has concentrated on the remineralizing effect of using different concentrations of various ions in a synthetic calcifying fluid on enamel lesions in vitro (Silverstone 1982 & 1983). Artificial lesions have been used in preference to natural ones since the lesions can be created

having predetermined characteristics. In addition, by creating an artificial lesion over a relatively wide test area, there is sufficient lesion surface to not only have adequate controls, but also to test different remineralization regimens on the single lesion. Examination of control sections from a lesion using a range of inhibition media in conjunction with the polarizing microscope makes it possible to study indirectly the ultrastructure of the tissue. Thus variations in pore volume in test and control regions can be assessed accurately as well as the total amount of oriented mineral in a particular region of a lesion (Silverstone 1982 and Silverstone 1983).

4. DENTAL CARIES

Dental caries is fundamentally a microbial disease which affects the calcified tissues of teeth, beginning first with a localized dissolution of the inorganic structures of a given tooth surface by acids of bacterial origin, and leading to a disintegration of the organic matrix. It is normally a progressive disease and, if unchecked, the lesion will expand in size and progress pulpally which may result in pain and pulpal inflammation. Ultimately, pulpal necrosis and loss of tooth vitality results (Darling 1956, Miller & Massler 1962).

Dental caries can be seen as a multifactorial disease. Bacterial mediation occurs through the production of organic acids by oral microorganisms which utilize locally available carbohydrates as substrates. The diet of the host provides the chief source of such carbohydrates, so that diet may be viewed as a primary factor in determining susceptibility to the disease. A number of factors indigenous to the host also determine susceptibility and severity of the disease. These include form, arch alignment, and the physicochemical nature of the tooth surface. The latter can be influenced by the intake of various trace elements in the diet, or through a surface effect by elements such as fluoride. Finally, the composition of the bacterial plaque is a primary factor. A number of different oral bacteria have been implicated in the caries process. Therefore, oral hygiene procedures which effectively remove plaque or which alter plaque metabolism tend

to inhibit dental caries. It is the combination of all these factors, superimposed upon the basic mechanism of bacterial acid dissolution of the tooth surface, that determines susceptibility to dental caries and the ultimate course of the disease (Meckel 1965).

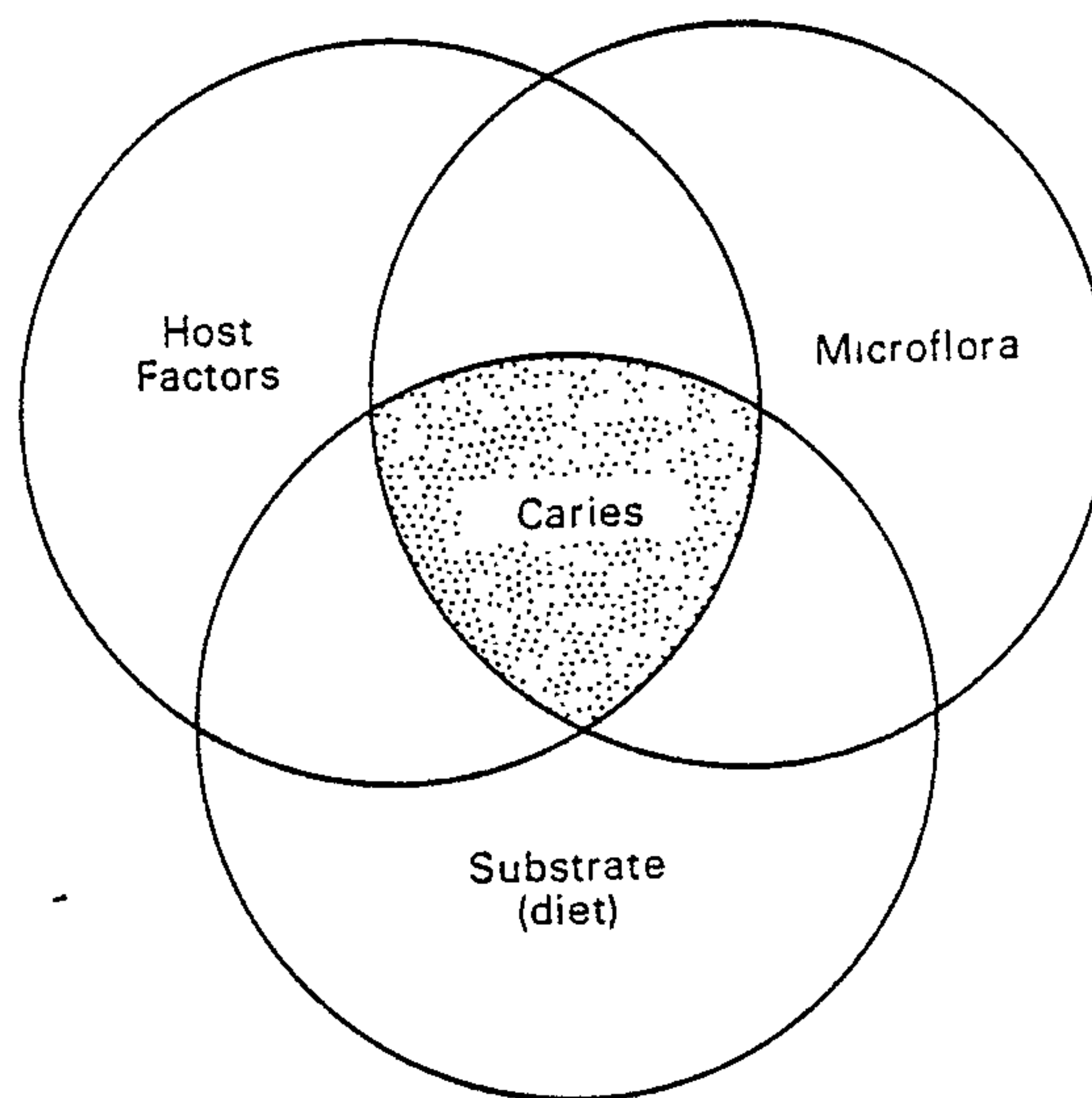
4.1 ETIOLOGY

Dental caries is a multifactorial disease. Numerous authors have recognized and described the caries process as one which is dependent upon the interrelationships of three main groups of factors. These groupings involve microbial, substrate, and host factors. It should be noted that for the disease process to be initiated, all three factors must exist simultaneously (see Fig. 4, page 47).

4.1.1 Bacteria

Definitive evidence for regarding dental caries as a bacterially mediated disease process has come from gnotobiotics studies. Orland, in 1955, conducted an experiment involving three groups of young, caries susceptible rats. One group was maintained under germfree conditions on a cariogenic diet. No caries resulted. A second group, which included littermates of those in the first group, was also maintained under germfree conditions, but these were infected with a strain of enterococci in conjunction with the cariogenic diet. Caries developed. The third group involved a control group raised in a normal environment with the same diet. Caries occurred in this group as well.

FIG. 4. THE MAJOR ETIOLOGIC FACTORS INVOLVED
IN THE DENTAL CARIES PROCESS



(Source: Newbrun 1983)

It seemed clear that specific strains of bacteria were, in fact, involved in the caries process and that their cariogenic potential was interrelated with dietary factors. This same type of experiment has been repeated on numerous occasions in various other laboratories. From such experiments as those of Orland the role of certain oral streptococci in dental caries has been more clearly defined, as well as the cariogenic activity of other groups of bacteria such as the lactobacilli and actinomyces (Orland, Blayney, Harrison, Ervin, Reyniers, Trexler, Gordon, Wagner & Morris 1955).

More recently, attention has focused on the role of dental plaque in the initiation of the caries lesion at specific sites on the tooth. By definition, plaque is an adherent bacterial mass which preferentially develops on the tooth surface. It is easily stainable by using certain disclosing agents and it is not removable by rinsing or a water spray. Plaque generates bacterial metabolites. These, along with certain exogenous materials, are concentrated within plaque. It is this bacterial ecosystem which enables the destructive influences of cariogenic bacteria to be focused on specific tooth surface sites.

4.1.2 Acid Dissolution

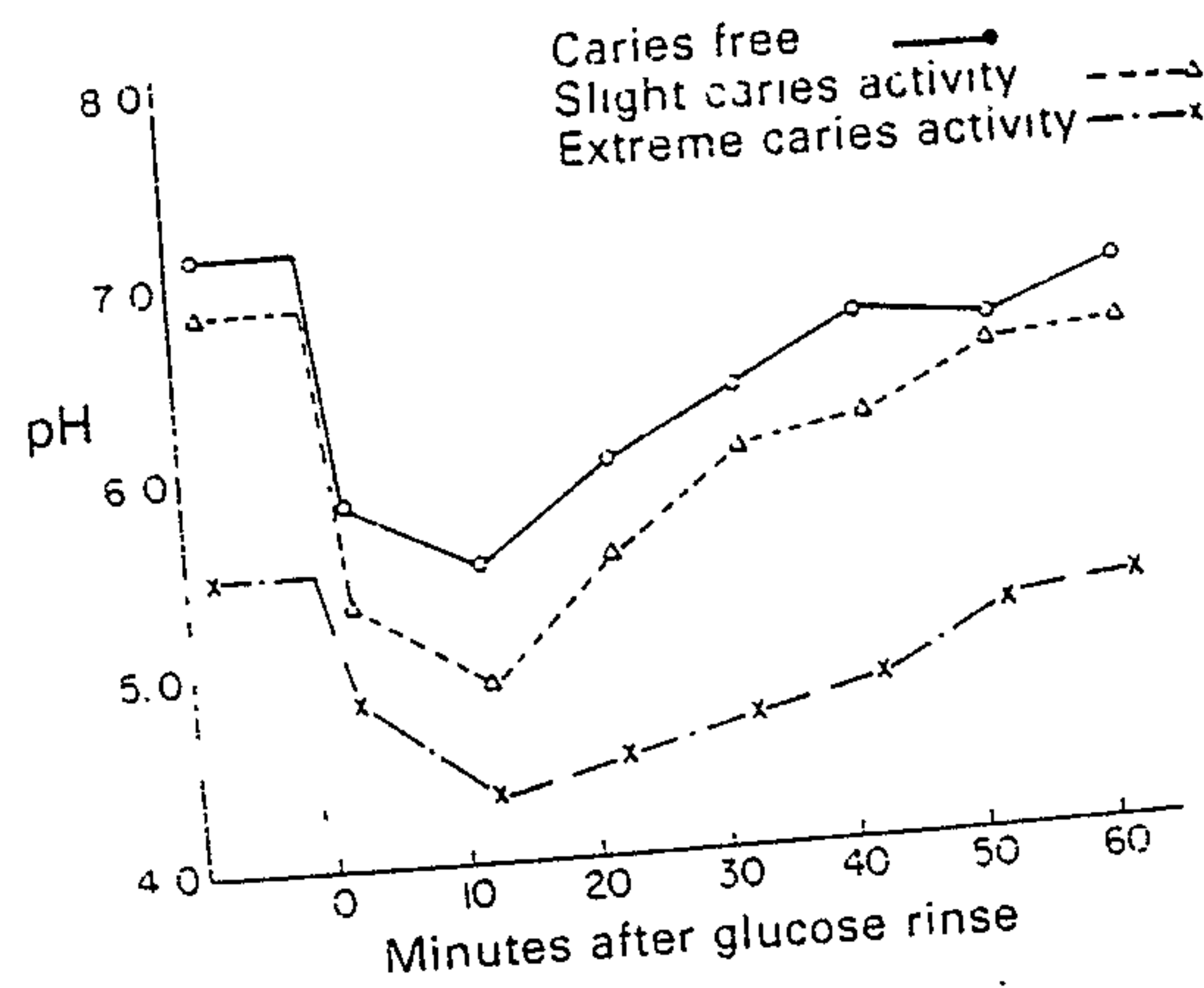
The fact that teeth are decalcified in the presence of acid is well established. A number of in vitro experiments have shown that dental enamel will demineralize when suspended in a test tube containing acid buffered

at a pH of 4.0 to 5.5. Calcium and phosphate from the dissolved tooth can be recovered in the buffer solution.

The acid producing (acidogenic) properties of a number of oral bacteria have been documented. These organisms are also aciduric in that most can grow and multiply best in an acid environment. These bacteria create an acid environment by converting carbohydrates to organic end products such as lactic, pyruvic, acetic, butyric, and propionic acids. The organic acids produced by plaque bacteria are capable of lowering the pH at the plaque-enamel interface to levels at which demineralization can take place. This has been confirmed experimentally (Stephan 1944). Microelectrodes have been placed at the tooth surface beneath a layer of plaque in human subjects and, following rinsing with a sugar solution, pH ranges of 4.0 to 5.5 and below have been recorded. Recordings of the pH were much lower beneath the plaque of extremely caries active subjects as compared to caries free individuals, presumably reflecting differences in composition of the bacterial plaques (see Figure 5, page 50). These low pH levels have been shown to persist for periods of over one hour. The plaque layer also shields the tooth surface from the washing and buffering action of saliva.

Recent research indicates that when plaque is systematically removed from the tooth surface by closely controlled oral hygiene procedures and biweekly professional prophylaxis caries incidence can be markedly reduced to a level approaching zero.

FIG. 5. pH RECORDINGS AT THE PLAQUE-ENAMEL INTERFACE OF UPPER INCISOR TEETH FOLLOWING A GLUCOSE RINSE



(Source: Stephan 1944)

All of these pieces of evidence fit together logically to portray the role of bacterial acids as prime mediators of the caries process.

4.1.3 Carbohydrates

For centuries it has been recognized that persons who ingest diets containing appreciable quantities of sugars and starches tend to experience tooth decay. Populations such as the Eskimos, who subsisted on diets heavy in fats but low in carbohydrates, historically have tended to have little or no tooth decay (Renson 1984(a)). Similarly, the natives of Tristan de Cunha with a simple diet low in fermentable carbohydrates historically exhibited only minimal caries experience (Fisher 1968). With the recent encroachment of Western customs and a diet rich in fermentable carbohydrates, caries incidence in both of the above populations has increased. This strong direct correlation between intake of fermentable carbohydrates and caries seems to prevail on a worldwide basis.

The need for carbohydrates, especially sugars, in the caries process has been shown experimentally through a number of mutually supportive observations. First, as previously noted, oral bacteria which have been shown to be cariogenic in gnotobiotic experiments have also been shown to utilize fermentable carbohydrates preferentially as an energy source in vitro. Secondly, animal experiments involving caries susceptible rats inoculated with known cariogenic organisms fail to show the development of dental

caries unless sugar is added to the diet.

A number of other formal experiments have illustrated the role of fermentable carbohydrates in the caries process, a role which is primarily a local one occurring at the plaque tooth surface interface (Frank & Brendell 1966). The first of these experiments involved an animal study utilizing two groups of caries susceptible rats (Orland et al. 1955). Each group was fed a high carbohydrate diet, except that the test group was fed by stomach tube so that the dietary carbohydrate was excluded from the oral cavity. The control group was fed in the normal manner. Results showed that the thirteen rats in the test group developed no caries lesions during the course of the study as compared to an average of 6.7 lesions in the control group. This illustrates that the influence of carbohydrates is due primarily to the local effects during passage through the oral cavity.

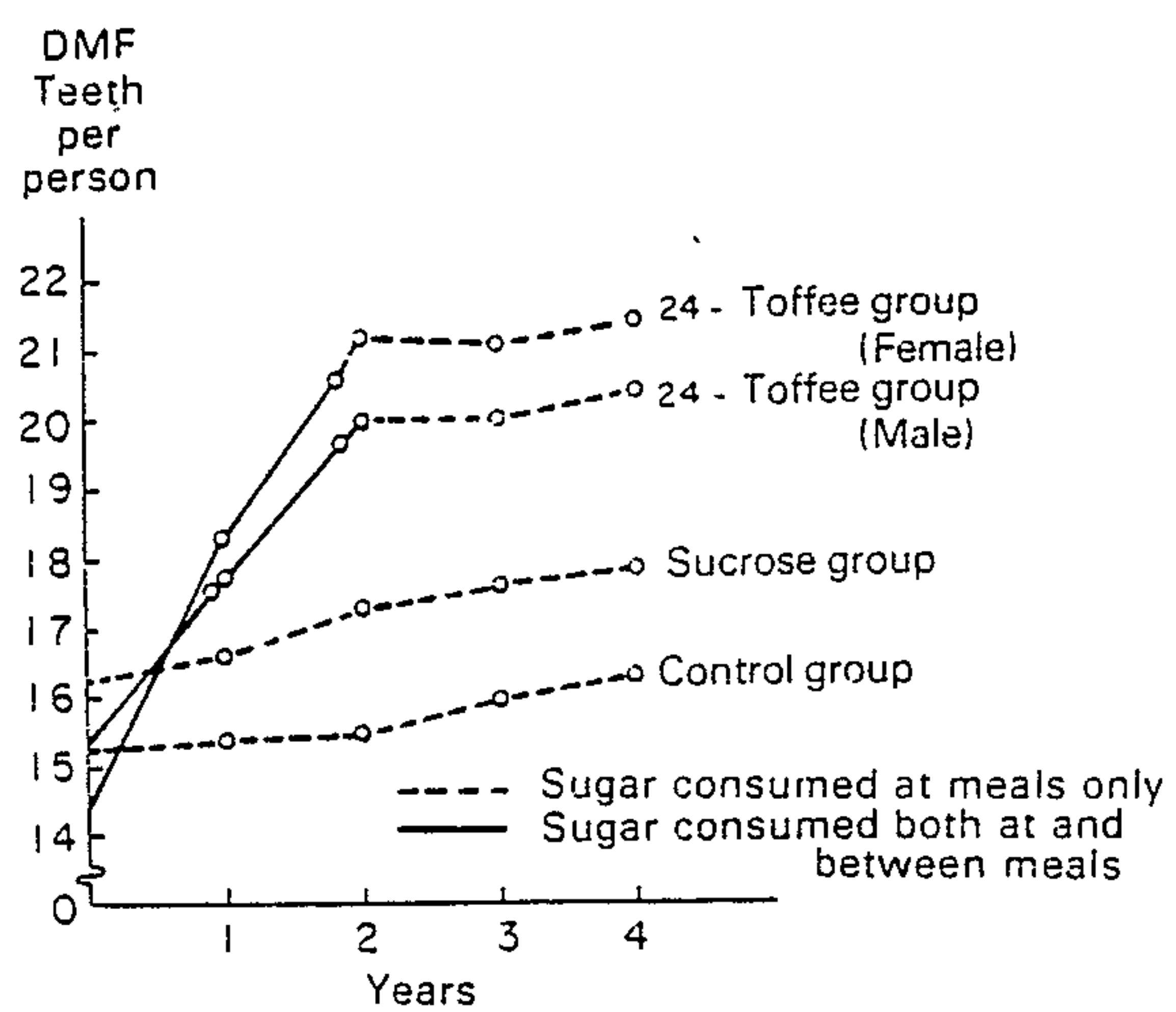
Another classic experiment, this time involving human subjects, was the Vipeholm dental caries study, a comprehensive investigation which involved 436 adult patients housed in a mental institution in Sweden (Gustafson 1953). Basically, this population was divided into several groups, each receiving a different diet. The control groups was given a basic diet relatively low in refined carbohydrates for two years (Carbohydrate Study I). During the final two years the diet of the control group more closely resembled the typical Swedish diet (Carbohydrate Study II). A second group was given the same basic

diet with the addition of sugar in solution at mealtime during the four year period (sucrose group). A third received the basic diet plus twenty four pieces of chewy toffee which was eaten at various times throughout the day for two years. As can be noted the caries incidence of patients in the sucrose group exceeded that of patients in the control group. The caries experience of patients in the toffee group, expressed in DMF-teeth per person, greatly exceeded that of patients in the sucrose and control groups. In fact, patients in the control group exhibited less than 10 per cent of the caries incidence noted in the toffee group over a two year period. Thus the importance of the frequency of sugar intake as well as consistency of the sugar containing foods was underscored (see Fig. 6, page 54).

Another set of observations, recorded in Australia, involved the orphaned children at Hopewood House. These children were raised on a diet which was essentially vegetarian and low in fermentable carbohydrates. Thirteen year old children raised on this dietary regime exhibited approximately 90 per cent fewer carious teeth (DMF) than did similar aged children attending state schools in Australia. Viewed from a different perspective, for each ten carious teeth that a typical thirteen year old child would have, his counterpart at Hopewood House would have only one (Goldsworthy 1958).

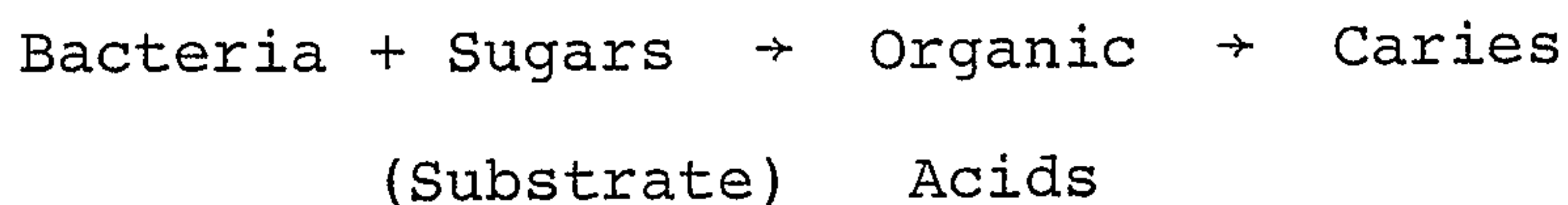
The common conclusion of these studies is to underscore the vital role that fermentable carbohydrates,

FIG. 6. OBSERVATIONS ON THE EFFECTS OF SUGAR
INTAKE ON DENTAL CARIES INCIDENCE



(Source: Gustaffson 1953)

especially sugars, play in the etiology of dental caries. The etiology of dental caries involves an interplay between oral bacteria, local carbohydrates, and the tooth surface that may be shown diagrammatically as follows:



4.2 CARIES PROGRESSION

The length of time a cavity takes to develop is of interest both in clinical practice and in research. It is important to know how often clinical examinations should be made in order to avoid serious damage by caries and how long an experiment must continue to ensure that the findings show an alteration in the number of new cavities appearing over a certain period of time and not merely an alteration in their rate of formation.

Caries activity may be expressed by the rate at which new caries develops (caries increment) and old caries progresses (caries progression). Increment may be expressed by the DMF index applied to a subject's teeth or surfaces, whereas caries progression which describes the rate at which an untreated lesion increases in volume is applied usually to surfaces or lesions. It is important to know how rapidly human dental caries progresses. Although the extent of an area of radiolucency in dentine does not accurately indicate the extent of a lesion (Marthaler & Germann 1970, Rugg-Gunn 1972); measurement of the rate of progression radiographically is the only

feasible in vivo method available. Further, as treatment of approximal lesions is usually based on radiographic appearance, it is important to know the rate at which radiolucencies progress.

The studies of Mathaler and Weissner (1973, as cited by Schwartz et al. 1984) estimated the average time that lesions remain in different parts of the enamel; Schwartz et al. (1984) showed that by ignoring filled lesions and non-progressing lesions, these studies may have overestimated the rate of progression.

Most studies of caries progression are based on an analysis of the number of lesions detected in a certain carious state (e.g. in the outer half of the enamel) that are still in that state at a subsequent examination (Backer Dirks 1966, Hollender & Koch 1969, Berman & Slack 1973, Hyde 1973, Murray & Majid 1978, Craig, Powell & Cooper 1981). These studies rarely provide sufficient data to estimate the probability distribution.

Caries progression describes the rate at which an untreated lesion increases in volume, which applies to surfaces or lesions. Boyd and his co-workers in the U.S.A. in a series of papers (Boyd & Wessels 1951, Boyd, Cannon & Leighton 1952) studied the progression of dental caries in the mouths of 212 teenaged children. The subjects lived in a state-controlled custodial institution where minimal amounts of dental care had been supplied in the past. Only emergency treatment was given during the course of the study. The findings indicated that one third of the

group had consecutive periods of at least one year during which caries did not advance in detectable degree. No difference was detectable between the group with the most, the median duration of non-advance was the same for both groups - 11 months. More specifically these workers observed the progression rates of dental caries in the occlusal surfaces of second permanent molars. This tooth was chosen for study because of its ready availability for clinical examination. The study extended over a 4-year period, but not all the teeth were present for that period of time. Carious lesions were diagnosed on half the occlusal surfaces within a year after tooth eruption and on 75 per cent of the occlusal surfaces within 18 months. Once the initial lesion had been diagnosed as carious, long periods usually elapsed before the involvement had spread notably. The medium length of time for progression to a definite enamel lesion was 20 months. To reach mild dentinal involvement took 3 years for the median cavity. Many of the lesions present when the study started did not advance at all. Boyd and his workers concluded that progression towards severe caries involvement is not rapid in the great majority of cases, but by contrast the appearance of an initial enamel lesion is prompt.

Parfitt (1956) has to a large degree confirmed these findings. The speed of development of carious lesions in the occlusal surfaces of 136 children in a mixed longitudinal survey was undertaken. It was seen that caries on occlusal surface of the teeth takes from

less than 3 months to over 48 months to progress through the incipient stage of dental caries.

Backer-Dirks (1966) carried out a study to investigate the post-eruptive changes in dental enamel. He described the criteria for assessing caries progression in fissures and pits, approximal surfaces and free smooth surfaces. Backer Dirks concluded that the carious process started first in pits and fissures and that lesions recorded in approximal surfaces did not progress rapidly. He also noticed that a small fraction of early lesion white spots progressed soon to a climax, but other white spots seemed to remain unchanged or even disappear altogether. Backer Dirks suggested that it seemed most likely that remineralization and/or recrystallization had taken place.

Berman and Slack (1973) in their studies also noticed that caries progression in posterior approximal surfaces appeared to be a slow process. Although the development of enamel lesions was prominent, the progression of cavities to a higher degree was not a marked phenomenon.

4.2.1 Studies Which Have Monitored Progression

A review of the literature has revealed nineteen studies in which the activity of small approximal carious lesions has been monitored in such a way that pertinent comments and results can be derived with respect to lesion progression (Pitts 1983). Before discussing their findings

it is essential to examine the nature of these reports and their sample populations to appreciate the many differences between them.

Table 3 reveals the sizes and age ranges of the study groups, and the study periods, of 13 of the series reported.

The majority of subjects examined were children (both the permanent and the primary dentitions have been studied). There is clearly a shortage of information relating to adults and elderly groups in which it might be expected that lesion progression would be slower.

Fluoride supplementation can of course be expected to exhibit a marked influence on the progression of approximal lesions, as can the level of fluoride present in the drinking water. Unfortunately most papers gave no indication of the water fluoride concentration, six recorded the levels as "very low" or "negligible" while no study reported findings for a fluoridated community. In 10 studies at least part of the study groups had received additional fluoride supplementation in some form at some time.

The initial caries prevalence of the different groups examined is also worthy of note; only three studies have dealt with low caries groups (i.e. an initial DMFS < 5), seven studies were concerned with an intermediate caries level (where DMFS = 5-20), three studies were conducted on groups with an initially high caries experience (DMFS > 20), whilst six gave no indication of

TABLE 3. Summary of Papers Which Have Monitored Progression of Approximal Carious Lesions by Studying Apparent Depth of Lesion Using Bitewing Radiographs

Authors	Age at start (yr)	n	Results 1 p *	Results 2 p †
A. At apparent F ⁻ supplementation				
ENGLISH	20	50	80% ±2.4 after 2 yr	ND
BAKER DIXON	7	100	m. surfaces of perm. 1st molars 50% ±7.6 after 4 yr 33% ±7.2 after 6 yr 26% ±6.7 after 8 yr	ND
HOLLENDER & KOCH	10	123	non-F ⁻ control groups 49.3% ±2.8 ‡ after 3 yr	ND
	10	85	50.4% ±3.7 ‡ after 3 yr	ND
BERMAN & SEWER	11-13	35	52.6% ±2.5 after 3 yr	ND
HUGGLOFEN & SACK	13-14	40	77.4% ±4.5 § after 1 yr	79.4% ±5.1 § after 1 yr
ZAMPA <i>et al.</i> 2 groups	20-24	51	77.9% ±4.2 " after 2 yr	81% ±4.7 " after 2 yr
	14-15			
GRONDAH <i>et al.</i>	16	158	46.9% ±1.4 " after 3 yr	64.4% ±1.7 " after 3 yr
GRONDAH & HOLLENDER	19	100	72.1% ±1.7 " after 3 yr	82.2% ±1.9 " after 3 yr
GRONDAH & HOLLENDER	16	100	35.6% ±1.8 " after 6 yr	48.5% ±2.3 " after 6 yr
GRANATH <i>et al.</i>	12-13	126	71.8% ±1.5 after 1 yr	86.4% ±1.5 after 1 yr
	12-13	126	56.8% ±2.5 after 2 yr	71.6% ±2.9 after 2 yr
POWELL <i>et al.</i>			non-F ⁻ control group	
	12-14	102	41% ±3.5 after 4 yr	ND
B. With F ⁻ supplementation				
HOLLENDER & KOCH			NaF dentifrice group	
	10	122	57.3% ±3.7 ‡ after 3 yr	ND
			NaF rinse group	
KOEHMAINEN & RYTOMAA	10	87	61.1% ±3.9 ‡ after 3 yr	ND
HOLLENDER & RONNERMAN	21-26	59	94.2% ±1.8 after 20 mth	ND
POWELL <i>et al.</i>	10-17	109	78.5% ±2.0 after 2 yr 2 mth	83.9% ±2.0 after 2 yr 2 mth
			dentifrice alone group	
	12-14	76	51% ±4.4 after 4 yr	ND
			dentifrice & topical group	
	12-14	71	71% ±3.7 after 4 yr	ND
CRAIG <i>et al.</i>			primary teeth	
	7	54	74% ±5.3 after 2 yr	ND

* % ±SD of lesions in the enamel at the start, still confined to enamel.
† % ±SD of lesions in the outer 1/2 of the enamel at the start, still confined to the enamel.
‡ SEM calculated by authors.
§ results from 2nd reading of radiographs.
" scoring code "DEJ" category added to "enamel" category.
" results for all approximal surfaces.

(Source: Pitts 1983)

the caries experience.

There are important factors relating to the study groups reported upon such as the level of oral hygiene, use of dental floss, dental awareness, motivation, and the availability and scope of preventive advice, which may all have influenced the reported rates of lesion progression. Unfortunately, most authors have given no detailed information of these factors. Other differences in methodology which might influence the results include the manner in which apparent lesion reversals were dealt with, whether or not the provision of restorative treatment was supervised, the number of people who "read" the radiographs, and whether readers had access to previous films.

Of the different approaches in recording scoring codes, eight studies recorded radiolucencies by subdividing the enamel into two halves, a further eight recorded only to the level of the full thickness of enamel whilst three studies used other scoring codes. As it appears that no penalty in terms of reproducibility is incurred by subdividing the enamel, this approach would seem preferable as it appears to offer more accurate evidence of progression.

4.2.2 Results of Studies on Caries Progression

Table 3 shows the results of the various studies as they pertain to the progression of serious lesions set out by the authors. The results of the investigations can

be expressed in the following way:

If p = the percentage of lesions in enamel at the start of the study, which remain confined to the enamel after a specific period, a series of values for p can be derived (either directly from the author's results or by calculation from the published data). The results obtainable in this way are summarised in the Results - 1 column of Table 3. The values of p exhibit a wide range from 94.2% (± 1.8) after 20 months to 26% (± 6.7) after 8 years. Scoring codes which enabled values for $p_{\frac{1}{2}}$ to be calculated (where $p_{\frac{1}{2}}$ = the percentage of lesions in the outer half of the enamel at the start of the study, which remain confined to the enamel after a specific period) were used in six reports. These values are given in the Results - 2 column of Table 3. The values of $p_{\frac{1}{2}}$ are consistently higher than the corresponding values of p for the same group; the range being from 86.4% (± 1.5) over 1 yr to 48.5% (± 2.3) over 6 years (Pitts 1983).

The remaining papers do not allow comparisons of their results by the above methods and are therefore considered individually. Backer Dirks (1961) was the first to undertake a systematic longitudinal study which allowed an assessment of progression to be made. For the 100 9-year-old children monitored over 6 years, he stated that in approximal surfaces "caries develops slowly, as it takes in the mean 3-4 yr for an incipient lesion to develop to a lesion affecting the dentine".

The fastest progression was reported by Murray and Majid (1978) for primary teeth in a group of 310 children between 5 and 7 yrs of age. Of the 71 "enamel" lesions studied, 69 had progressed to dentine 1 yr later. The authors noted that the rapid progression rate might be partly explained by the convention used that only those lesions entering the inner half of the enamel were considered. These results are in strong contrast to a report (Craig et al. 1981) that 74% of lesions remained in primary enamel after 2 yr; however, these 7-yr-olds did receive a fluoride application immediately prior to the study. The remaining studies have dealt with groups for whom faster progression rates could have been expected. The average time taken for a lesion to penetrate the outer half of the (permanent) enamel in 133 "caries active" children between the ages of 7.5 yr to 14.5 yr is reported as 1.71 years (Mathaler & Wiesner 1973 - cited by Pitts 1983). The accuracy of this time interval can be questioned as the data excluded all arrested lesions (15% of the total), and the authors (Marthaler & Wiesner) admit that unsupervised retoration of enamel lesions often occurred.

4.3 CHANGING PATTERNS OF DENTAL CARIES PREVALENCE

The problems presented by dental caries are not the same in all countries. There are wide geographical differences in its prevalence, and varying social and economic conditions modify what can be done about it.

From data which have accumulated at the WHO Global Data Bank in Geneva over a number of years two major trends in oral health status have been identified:

- deterioration for most of the developing countries;
- improvement for most of the industrialized countries

(Renson 1984(a))

As it is seen today, dental caries is most destructive in the teeth of children in whom an attack leads to the formation of cavities or in extreme cases the destruction of all the teeth. It was not always this way. Examination of teeth from archaeologic or historic sites show that in primitive man it was the old people and not children who were the principal sufferers. It was the roots of the teeth and not the crowns that were principally affected and the total amount of decay that occurred was little more than that found in some wild animals today. The teeth from subsequent times indicate relationships between caries prevalence and changing eating habits. For instance, an increase of caries in ancient Egypt has been associated with the adoption of wheat as a major food item; a doubling of the numbers of decayed teeth in ancient Greece during one millenium to the adoption of Roman cooking practices, and a rise and then a fall in decayed teeth in Britain to an adequate food supply during the Roman occupation and a shortage of food thereafter (Bibby 1978).

A dramatic time-related increase in caries appeared in western Europe over the past three centuries. During

this time sugar became plentiful and flour became more finely refined and the amount of dental decay multiplied several times. Now, instead of only 2 or 3% of the adults having carious teeth, 90-98% of the population are affected during childhood years (Bibby 1978).

An even more discouraging situation exists in the less developed countries of the world; particularly in urban centres. Until comparatively recent times, the majority of the islands in the Cook Islands were isolated from the outside world by their remoteness. Early western explorers, such as Cook and Bougainville, in the accounts of their voyages in the eighteenth century, specifically remarked on the excellence of the teeth of the inhabitants, but over the past twenty years or so, high dental caries rates have been reported from these islands. This deterioration in dental health is due to the increased availability of refined carbohydrate foods and especially the increased consumption of sugar (Speake, Cuttress & Ball 1979).

A comparison of recent studies (Renson 1984 (b)) with previous epidemiological measurements has shown that in many industrialized countries a significant reduction in dental caries prevalence has occurred (Table 4). Moreover, the decrease appears to be continuing to levels well below those expected in a number of these countries.

In developing countries, the prevalence of dental caries 15-20 years ago was low or very low. Recent studies in a number of these countries have shown a rapid increase

TABLE 4. Caries Prevalence Trends in Highly Industrialized Countries, DMFT in Children Aged 12 Years

Country	Highest	Year	Lowest	Year
Australia	9.3	1956	2.9	1982
Canada	7.4	1958-60	2.9*	1979
Finland	7.5	1975	5.7	1981
New Zealand	10.7*	1973	6.3*	1982
Norway	12.0	1940	4.5	1979
Sweden	7.8	1937	3.4	1979
Switzerland	9.6	1961-63	1.7	1980
USA	7.6	1946	2.0	1980

*Children aged 13-14 years.
Prepared by WHO in 1983.

(Source: Renson 1984(a))

in dental caries prevalence (Table 5) (Renson 1984(b)) and this is especially true in urban communities. It is not yet clear whether this trend will continue, how far it will go, or whether it will extend to other developing countries.

In the examination of data received from many different countries around the world, Renson (b) (1984) sought to establish the factors which were common to those countries which had achieved the greatest success in reducing prevalence figures for dental caries. The most probable factors underlying the improved dental situation among children in these countries are:

- (1) The widespread exposure of children to fluoride supplements and especially the daily home use of fluoridated dentifrices.
- (2) The ready availability of dental resources.
- (3) The provision of preventive oral health services, especially for children.
- (4) The enhancement of "dental awareness" by organized oral health education programmes.

(Renson (b) 1984)

The Influence of Declining Caries Prevalence on Dental Caries

It is difficult to assess the influence of the changing pattern of dental caries on future dental manpower needs because it is not possible to forecast the future

TABLE 5. Caries Prevalence Trends in Developing Countries, DMFT in Children Aged 12 Years

Country	Level	Year	Level	Year
Chile	2.8	1960	6.3	1978
Ethiopia	0.2	1958	1.5	1975
French Polynesia	6.5	1966	10.7	1977
Iran	2.4	1974	4.9	1976
Israel	2.4	1966	3.7	1976
Jordan	0.2	1962	2.7	1981
Kenya	1.7	1973-77	3.7	1982
Lebanon	1.2	1961	3.6	1974
Mexico	2.7	1972	5.3	1976
Morocco	2.6	1970	4.5	1980
Philippines	1.4	1967-68	2.9	1981
Thailand	2.7	1977	4.4	1982 (Bangkok)
Uganda	0.4	1966	1.5	1982
Zaire	0.1	1971	2.3	1982

Prepared by WHO in 1983.

(Source: Renson 1984 (a))

rate of change. It may well be that the full effect of the change has yet to be experienced or it may be that there will be a levelling off in the decline in prevalence figures. However it would appear likely that dental restorative needs in developed countries in coming generations will be considerably less than has been the case in the past. Because the demand for restorative dentistry has been the major consumer of dental manpower in the past, consideration must now be given to the need to monitor the future demand for such treatment (Renson 1984(b)). It is likely that there will be a change of emphasis in dental care. In children and young adults extensive restorative treatment will not be the norm, prevention and simple restorations will be the order of the day. Restorative care for increasing numbers of the elderly who retain their teeth will demand increased operative skills. Thus there will be a polarization of treatment needs: simple for the young and complex for the increasingly dentally aware middle-aged and elderly.

All countries should develop a goal-oriented national plan for oral health within the national health plan. The plan should be based upon a situation analysis and sound epidemiological data (Renson 1983). The planning process should be divided into short, medium and long term phases and regular reviews should be incorporated in the plans of activities. Measurable goals are essential and will relate to disease prevalence, manpower availability, economic resources and other relevant factors.

In most developing countries the shortage of suitable dental personnel is preventing the improvement of poor periodontal health and an increasing prevalence of dental caries. It is evident that oral health problems in developing countries are now at a stage when reasonable resources must be devoted to prevention by (Renson 1983):

- (1) An expansion of available dental manpower.
- (2) The setting up of preventive oral health services; particularly for children.
- (3) The introduction of oral health education programmes.
- (4) The provision of fluoride supplements and dentifrices.
- (5) Would obviate the need for a huge expenditure in the future for restorative and rehabilitative services.

The situation in industrialized countries complements the situation in developing countries, with declining disease prevalence and an oversupply of dental resources (Renson 1984(b)). In addition, developing countries should make use of established dental schools and other training establishments in overseas countries to place their students rather than to opt for costly dental schools in their own countries. It is in these and in similar ways that the most effective use can be made of available dental resources.

5. APPROPRIATENESS OF THE USE OF THE COMPOUNDS
FOR THE COOK ISLANDS

It has been reported that topical treatment with ammoniated silver fluoride ($\text{Ag}(\text{NH}_3)_2\text{F}$) prevented the lateral spread of established lesions in primary anterior teeth over a 30 months period (Nishino 1969).

One factor in the selection of silver fluoride for the treatment of arresting caries is that this salt possesses appreciable antimicrobial activity, a property of possible value in the treatment of active lesions. Low concentrations of silver fluoride have been shown to be effective in inhibiting the growth of *S. mutans* (Thibodeau et al. 1978) and in reducing the metabolic activity of dental plaque. The intended function of tin ions (Sn^{++}) from stannous fluoride (SnF_2) was to act as a reducing agent for silver (Ag^+) ions. However, it should be pointed out that stannous fluoride (SnF_2) itself also possesses definite antimicrobial properties.

A useful aspect of topical treatments with silver fluoride followed by stannous fluoride is that this combination turned the surface of active lesions black. Preliminary studies indicated that the presence of a continuous black mat on a lesion surface appeared to be associated with caries arrestment. Any lightening of the lesion was suggestive of caries development (Craig et al. 1981).

Since 1978, the most successful and accepted technique in the use of silver fluoride for the arrestment of dental caries is that adopted by Craig of the Department of Preventive Dentistry, University of Sydney (Craig & Powell 1979).

The use of silver fluoride has been to slow or arrest caries in both the incipient and open lesions. This action is most important in primary dentition because primary teeth have a finite life-span. The slowing of the progression of carious lesions may allow teeth to reach exfoliation without the need for operative intervention. Silver fluoride can also be used after cavity preparation with some caries remaining; and prior to replacement of G.I.C. (glass ionomer cement). Glass ionomer cement can be used in conjunction with silver fluoride under field conditions especially if there is further progression (Craig 1981). This minimal treatment procedure is designed for delivering dental care to young children without undue dental trauma; and also in situations where there is:

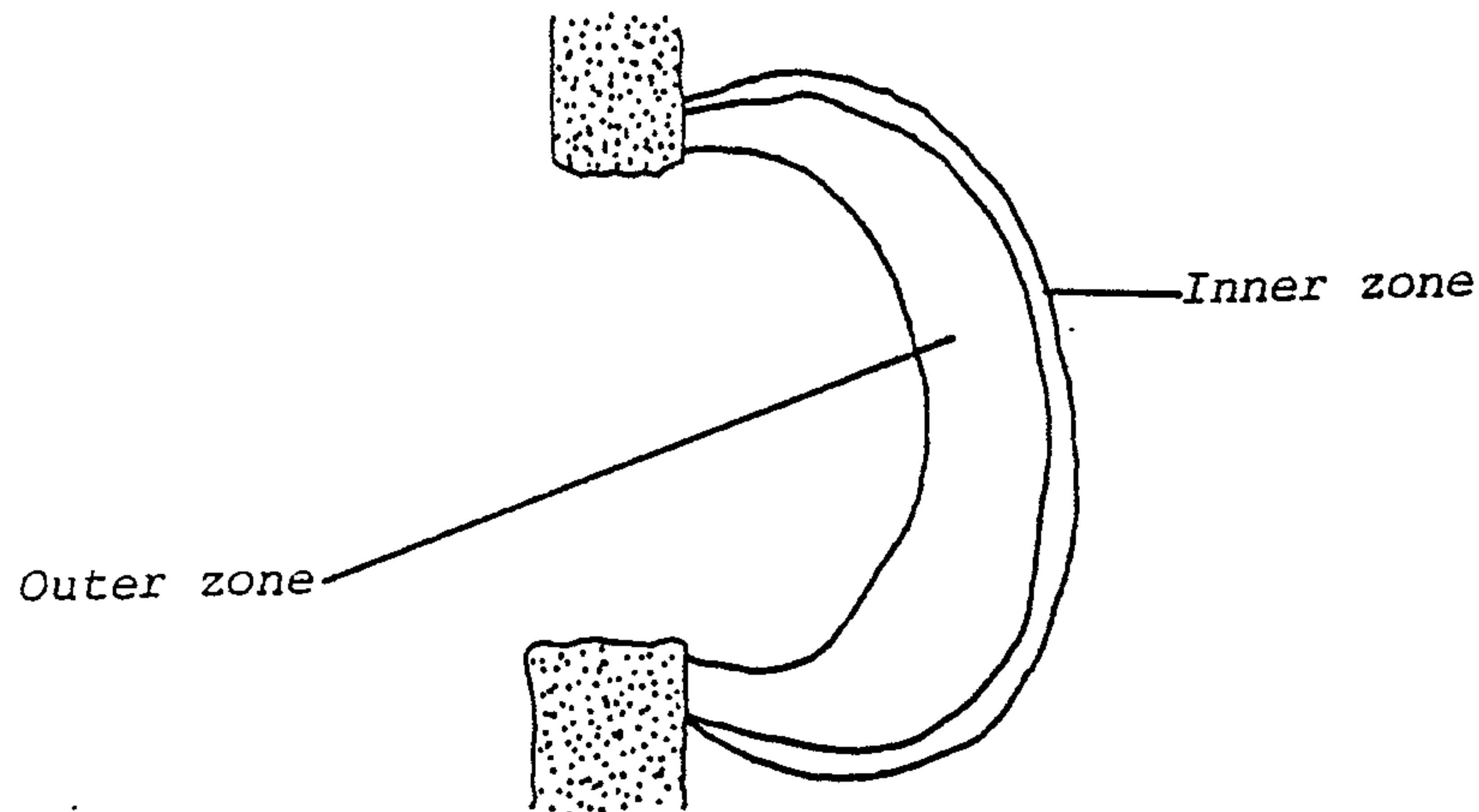
1. a high incidence of dental disease;
2. a large backlog of unmet treatment needs; and
3. limited patient co-operation during dental procedures.

The indications for the use of Craig's treatment procedures, materials to be used and some basic rules for a variety of presenting conditions are presented in the Appendix Chapter of this treatise.

5.1 POSSIBLE MODE OF ACTION OF SILVER FLUORIDE (HYPOTHESIS)

(a) An active carious lesion in dentine consists of two zones:

- (i) An outer zone containing bacteria, denatured collagen fibres and other dentine breakdown products;
- (ii) An inner zone which has been softened by acid attack but where the collagen fibres are still basically intact.



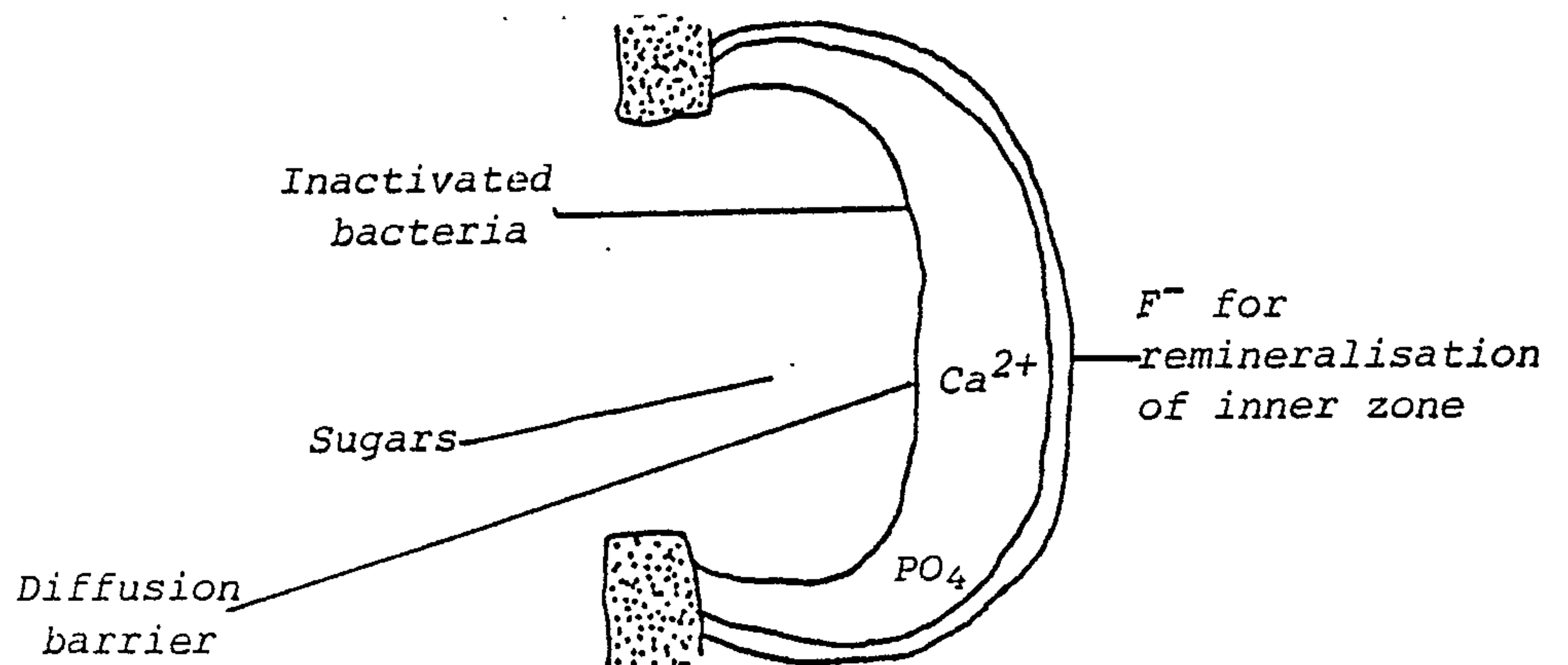
(b) The silver moiety from silver fluoride possibly has its main effect on the outer zone. Ag^+ ions have a very pronounced anti-bacterial action and thus inactive acid producing organisms. In addition, Ag^+ ions have an affinity for chemical groups found on bacterial cell walls and in proteins, e.g. $-\text{SH}$, $-\text{COOH}$, $-\text{PO}_4$ groups. This property may result in some degree of cross-linking occurring in the outer zone (a bit like tanning leather) which could mean that the outer layer becomes a diffusion barrier, either inhibiting

the entry of sugars into the lesion and/or the exit of calcium and phosphate.

- (c) The fluoride moiety from silver fluoride I believe to be necessary for the remineralization of the inner zone. Although the inner zone is softened as a result of acid attack, the fact that the collagen fibres are still intact means that they can act as a template for remineralization.

This is the basic difference between silver fluoride and the formerly used silver nitrate - silver nitrate lacks the crucial F^- ion.

After AgF Treatment



5.2 CLINICAL EFFECTIVENESS

The usefulness of Craig's treatment procedure in arresting dental caries is supported by the result of a survey which was carried out by the writer in the hospital clinics, school dental clinics, and private clinics in Sydney. The survey was done in February 1986 in the form of questionnaires directed to the operators using the treatment procedure (Appendix Chapter).

Although the sample taken in this survey (35 operators) is not a true representation of all operators in Sydney, the results obtained indicated that most operators favoured the treatment for the arrestment of dental caries.

The results showed that 23 operators (65%) used this treatment procedure specifically for arresting dental caries.

The treatment procedure was performed only by dentists and school dental therapists.

5.3 COST-ANALYSIS

An attempt is made by the writer to analyse the cost involved in the treatment procedure for arresting dental caries. In such an exercise, it is assumed that if the cavities are not treated with AgF, each would end up with amalgam restorations.

When a decision has to be made between treatment alternatives, especially for public health programmes,

cost-benefit and cost-effectiveness analyses can be used to assess the economic aspects. In cost-benefit analysis the benefit of a procedure is measured and is then related to the cost of achieving that benefit (Horowitz & Heifetz 1979). This form of analysis is useful in deciding between broad treatment strategies, for example whether to adopt a preventive approach or place restorations. Once a general treatment strategy has been decided upon, cost-effectiveness analysis provides a means of determining the most efficient or least expensive way of achieving the particular objective (Horowitz & Heifetz 1979). If such an objective is the prevention of dental caries, cost-effectiveness analysis can be used to help make a rational choice between the various preventive regimes available.

Because of the high cost of professional time, questions have been raised as to the economic viability of programmes where fluorides are professionally applied (Davies 1973, Heifetz 1978).

Since the silver fluoride therapy discussed in this presentation also requires professional application, an attempt is made by the author to obtain some acceptable data on the cost-benefit and cost-effectiveness of the procedure. Such data can be compared to that presented by Enno and Craig (1982) on the economic aspect of the prolonged fluoride application method.

The estimated cost of treatment per tooth with the PFA method is as follows:

	PFA METHOD	OCCLUSAL AMALGAM RESTORATION
Treatment time	1.5 min	-
* COST OF PROFESSIONAL TIME	\$1.51	-
Materials	4c	-
TOTAL COST	\$1.55	\$15.60 [†]

* Derived from recommended professional hourly rate of \$60.35

(A.D.A.-N.S.W. Branch, Nov. 1980)

[†] Recommended fee (A.D.A.-N.S.W. Branch, Nov. 1980)

(Source: Enno & Craig 1982)

The estimated cost of treating each tooth with AgF is presented as follows:

	AgF	OCCLUSAL AMALGAM RESTORATION
Treatment time	5 minutes	-
* Cost of professional time	\$9.67	-
Materials	.97	-
TOTAL COST	\$10.64	\$29.90 [†]

* Suggested minimum fee schedule of the professional hourly rate of \$116.00 per hour (A.D.A.-N.S.W. Branch, May 1986)

[†] Suggested minimum fee schedule (A.D.A.-N.S.W. Branch, May 1986).

Estimated Cost of One Treatment AgF/SnF₂/Stomahesive Wafer

	<u>\$</u>
Cost of 3 mls bottle of AgF	37.62
Approx. there are 60 drops in this bottle	
∴ 1 drop	<u>0.62</u>
Cost of Spot Application Paste	3.50
∴ 1 drop	<u>0.01</u>
Cost of Stomahesive Wafer	13.00 Box
There are about 500 pieces/box	
∴ Cost of 1 piece for 1 treatment	<u>0.03</u>
Cost of G.I.C. (50 capsules in kit)	16.30 kit
∴ Cost 1 capsule for 1 treatment	<u>0.33</u>
∴ Total cost for materials for 1 tooth	<u>\$0.97</u>

(See Appendix Chapter for Cost of Materials)

5.4 COST-EFFECTIVENESS

The cost-effectiveness of this treatment programme can be calculated by using the method of Heifetz (1978).

$$\text{i.e. Cost-effectiveness} = \frac{\text{Cost of AgF per child}}{\text{Mean number of surfaces saved per child}}$$

However, at the time of writing this treatise, the author is unable to present any data on the cost-effectiveness of this preventive programme as there is none available;

and there have been no studies conducted to estimate the number of surfaces saved by using the AgF treatment.

5.5 ADVANTAGES OF THE AgF TREATMENT

1. There is no pain involved in the treatment. Use of injections and high-speed drills are omitted.
2. As this treatment is not painful, the operator can accomplish the treatment because of the full patient co-operation.
3. It is a time-saving operation (5 minutes).
4. There is no G.A. required.
5. This treatment is cheaper than amalgam restorations.
6. Some practitioners have reported that they derive more personal satisfaction from this procedure.
7. The patients and parents can see something tangible as a result of the treatment (especially the black stain which is associated with arrestment of caries).

5.6 DISADVANTAGES OF THE AgF TREATMENT

1. The black stain when treating the anterior teeth is the only complaint from the parents.
2. Restlessness (if more than one quadrant is treated) during the treatment may occur in small children.

6. DISCUSSION

6.1 INTRODUCTION

This thesis is a review of the literature on the need for dental care, which is concerned with the treatment and prevention of oral diseases and their sequelae. Dental caries is the most common of these oral diseases, and its prevalence can only be reduced by prevention. The prevention procedure, which is the main subject of this review, is silver compounds - especially silver nitrate and silver fluoride.

The two major methods of preventing dental caries, from an ecological point of view are:

1. By increasing the resistance of tooth enamel to external attack. This can be achieved by the systemic and topical administration of fluoride.
2. By reducing the intensity of the attacking agents; through restricting the frequency with which sugar in a sticky form is consumed and the establishment of a regular and efficient system of tooth cleansing to reduce the accumulation of bacterial plaque on the surface of the teeth.

Proof that bacteria are necessary participants in the caries attack has been offered. Some types of bacteria can actually cause caries while some can participate in the initiation or expansion of carious lesions.

There is no doubt that fluoridation of the community water supplies is the most effective and least expensive way to prevent dental decay in large groups of

people. However, its implementation is limited to areas with central water supplies. Where community water fluoridation is not feasible, other methods of delivering fluorides have to be considered. The method described in this thesis is that of 40% silver fluoride solution followed by a thin layer of 10% stannous fluoride spot application paste (Craig's technique). Emphasis will be placed on the treatment (arresting) and prevention of dental caries in children and young adults by the use of the silver compounds. The therapy includes the application of 40% silver fluoride (AgF) solution to a carious lesion for 3 minutes, followed by 10% SnF_2 spotting paste. The treated lesion is then covered by a stomahesive wafer. In anterior teeth, glass ionomer cement (G.I.C.) is used instead of the stomahesive wafer. After the application of SnF_2 , there is a change of the carious lesion to black - a sign of caries arrest.

6.2 USEFULNESS OF THE TREATMENT PROCEDURE IN THE COOK ISLANDS

As described in the Cook Islands section of this presentation, dental caries constitutes the most prevalent of all diseases. Past survey data showed that all school children in the Cook Islands were affected by the disease at the age of 13 years. It was found that on the average, school children on Rarotonga had 10.4 decayed, missing or filled (DMFT) permanent teeth. In a situation as such, if it is to overcome this high caries rate, the emphasis

is based on the need to control and prevent dental caries. Other measures will not be successful; because, the resources are stretched both geographically and economically.

It is clear that the addition of fluoride to the public water system in the Cook Islands is not possible. Therefore, other alternatives to water fluoridation, especially topical fluoride therapy should be introduced to prevent this high prevalence of dental caries.

There should be a tight control by the government on the importation of cariogenic foodstuffs into the country. The Family Budget Sample Survey which was carried out in 1976 showed a marked increase in the consumption of many potentially cariogenic foodstuffs.

The author in this presentation believes that the slowing or arresting of dental caries by silver fluoride treatment will be one of the preventive programmes to be introduced for the Northern islands of the Cook Islands. Because there is no dentist stationed on any of these islands in the Northern group, this silver fluoride treatment programme seems to be ideal for these isolated islands because it has the potential to be used by auxiliaries in the field. A dentist's temporary visit to these islands can be expensive; and therefore in this preventive programme, an auxiliary from any of these Northern Group islands can be trained in this silver fluoride treatment procedures. This auxiliary, who is from these Northern Group islands can be stationed

permanently there. The visit by the dentist and technicians to these islands can be concentrated to other complicated services.

The education system in the Cook Islands can also have a big influence in the success of this silver fluoride treatment programme. Because the dental clinics are situated in the school compounds of these islands, and because the children in the Cook Islands spend nine months of the year at school, it is very likely that each child can be seen and treated once a year or even twice a year in smaller schools.

Besides other preventive programmes such as mouth-rinses at schools, AgF treatment can be introduced as part of the overall preventive programme. AgF treatment can be for lesions that may develop in deciduous teeth.

6.3 SILVER COMPOUNDS

Silver nitrate was firmly entrenched in the dental profession (in the 1930's-1940's) as a remedy for hypersensitivity of dentin, erosions and pyorrhoea, as well as a sterilizing agent and caries inhibitor in deciduous as well as permanent teeth.

Szabo (1902), who attempted to examine silver nitrate scientifically was also trying to answer the question as to how far silver nitrate actually penetrated the dentinal tubules. From his study, he found that the penetration of silver nitrate through carious matter into sound dentine was not further than 0.5 mm. Several

applications of the solution did not change the depth of penetration. He believed that silver nitrate combined with the albumin of the dental fibrils to form a silver protein-ate, which stopped further penetration of the silver nitrate solution.

Miller (1905) laid down the chemotherapeutic principles of the actions of silver nitrate. He noticed that the precipitates of silver in the superficial layers of the dentine may form a barrier more or less impermeable to acids. This may be due to the coagulation of the contents of the dentinal tubules which protected the dentin against the action of acids.

The introduction of Howe's silver nitrate solution (1917), being an alkaline solution, had the same effects on carious lesions as that seen in the silver nitrate study done by Szabo (in 1902). The sterilization action resulted from the action of the metallic silver upon the proteins of the bacteria in the tooth structure, which are precipitated, and thus prevented from further action.

Prime (1935), discussed the value of silver nitrate in the treatment of incipient caries and its use as a disclosing solution.

Ireland (1939) found that silver nitrate was a good sterilizing agent for infected dentin. This action reduced the incidence of carious pulpal exposure.

It was Seltzer and Werther (1941) during their studies who showed that silver nitrate cannot be held responsible for pulp devitalization. They discovered

that silver nitrate aided in the retention of the tooth by giving pulp tissue time to lay down secondary dentine.

Graham (1941) presented the value of silver nitrate by its ability to sterilize disintegrating dentinal structure and to neutralize the acid reaction of dental caries. Graham (1941) noticed that the sterilization resulted from the action of the metallic silver upon the proteins of the bacteria in the decayed tooth structure, which are precipitated and thus prevented from further action. The technique is particularly applicable to deciduous teeth presenting large cavities in which any attempt to remove the decay almost invariably would result in pulpal exposure.

6.4 DENTAL CARIES

Dental caries, being a microbial disease, affects the calcified tissues of teeth. It begins with a localized dissolution of the inorganic structures of a given tooth surface by acids of bacterial origin leading to disintegration of the organic matter.

Caries progression can be analysed by the number of lesions detected in some carious state that are still in that state at another examination given at a later date. Table 6 (page 86) shows estimates of the average time a lesion remains in the enamel, or in parts of the enamel. For permanent teeth, with the exception of Hyde (1973), where the average duration of an enamel lesion is between 14 and 33 months, and Granath, Kahlmeter, Mattson, Schroder

TABLE 6. Estimates from the Literature of Average Duration of Lesions in the Enamel Based on the Assumption that Duration of Time in any State Follows an Exponential Distribution

Author	Location	No. cases	Teeth	Average starting age	Length of follow up	Average duration (months)	Comments
Backer Dirks (1966)	The Netherlands	100	mesial surface, 1st permanent molar	7	8	70.5 in enamel	
Hollender and Koch (1969)	Malmö, Sweden	100	premolars	10	3	64.6	fluoride dentifrice
		100	1st permanent molar			51.3	placebo dentifrice
		100				73.1	fluoride rinse
		100				52.5	placebo rinse
Berman and Slack (1969)	Essex, England	353	premolars, 1st and 2nd permanent molar	11.8	3	in enamel 56.1 in enamel	
Hyde (1973)	Vancouver, Canada	100	1st permanent molar	within 2 yr of eruption	2	14.0	placebo
		96				20.6	silver nitrate
		92				33.5	phosphate-fluoride
		89				21.4	stannous-fluoride
Haugejorden and Slack (1975)		40 pairs radiographs		13-15	1	in enamel (26.9), (24.3)	outer half of enamel inner half of enamel total
Gröndahl and Hollender (1979)	Gothenburg, Sweden	100	all	16	6	51.2 in enamel (53.2), (36.6)	outer half of enamel inner half of enamel total
Granath <i>et al.</i> (1980)	Malmö, Sweden	126	premolars and molars	12-13	2	89.2 in enamel (19.4), (22.9)	outer half of enamel inner half of enamel total
Craig <i>et al.</i> (1981)	Bourke, Wales	54	primary molars		2	42.3 in enamel 79.7 in enamel	43.7 months using 1-year non-progression rate
Murray and Majid (1978)	Chesham, England		primary molars	5	1	3.4 months	inner half of enamel

(Source: Shwartz et al. 1984)

(1980), where the average duration is 42 months, estimates are all greater than 50 months, with a maximum of 89 months. Only two of the studies in Table 6 considered primary teeth. Estimates of the average duration of enamel lesions from Craig et al. (1981) are dependent on whether the one-year rate or the two-year rate is used to derive the estimate. The estimate of the average duration of enamel lesions using the two-year rate, 79 months seems unreasonable. The estimate using the one-year rate, 44 months, is longer than Shwartz (1984), estimates of between 20 and 31 months. The estimate of time taken to progress through the inner half of the enamel derived from Murray and Majid (1978), 3.4 months is extremely rapid.

It seems from the analysis of Shwartz et al. (1984), plus other data in the literature, that it takes at least an average of four years for a lesion to progress through the enamel of permanent teeth. Progression is slower for older individuals, particularly those with long-term exposure to fluorides.

Pitts (1983), after reviewing much of the literature shown in Table 6 (page 86), reaches similar conclusions. However, it is important to recognize that the duration of enamel lesions is extremely variable between individuals and between lesions in any one individual. This suggests that it may be more useful to focus on the distribution of time for which lesions remain in the enamel rather than on the average duration. Assuming that the distribution of time for which a lesion remains in each half of the

enamel is exponential with a mean of two years; the percentage of new lesions still in the enamel one, two, three and four years later is 91, 74, 55 and 41 per cent respectively (Shwartz 1984).

6.5 CHANGING PATTERNS OF DENTAL CARIES PREVALENCE

The problems presented by dental caries are not the same in all countries. Over a number of years, two major trends in oral health have been identified:

- deterioration for most of the developing countries
- improvement for most of the industrialized countries.

This changing pattern of dental caries affects the demand for future dental manpower needs. There will be an oversupply of dental resources in industrialized countries with declining disease prevalence. In most developing countries, the shortage of dental manpower is preventing the improvement of poor periodontal health and an increasing prevalence of dental caries.

6.6 REMINERALIZATION

Remineralization occurs naturally during the formation of a carious lesion. Experimental studies have shown that acid softened enamel surfaces can reharden in vitro after the application of a calcifying fluid to bring about mineralization. These studies also show the presence of fluoride ions enhances greatly the degree of remineralization achieved and reduces the time period for this mechanism to occur.

6.7 APPROPRIATENESS OF THE USE OF THE COMPOUNDS

The use of silver nitrate, and silver fluoride with stannous fluoride, in the arresting and prevention of dental caries, has been widely studied. The development of silver nitrate and its sterilizing effect on the problems of the bacteria in the tooth structure has already been discussed. The selection of silver fluoride for the treatment of arresting caries is important because this salt possesses antimicrobial activity properties which can be of great value in the treatment of active lesions. Low concentrations of AgF has been shown to be effective in inhibiting the growth of *S. mutans* and in reducing the metabolic activity of dental plaque. The intended function of Sn^{++} ions from SnF_2 was to act as a reducing agent for Ag^+ ions. Stannous fluoride itself also possessed definite antimicrobial properties. The combination of the treatment (AgF followed by SnF_2) turned the surface of active lesions black - associated with caries arrestment.

6.8 TREATMENT PROCEDURES

Absolute care must be observed when carrying out the treatment procedures. In common with other salts, e.g. AgNO_3 , this preparation will stain skin and clothing. Silver fluoride solution will produce burns (eschars) if it accidentally comes in contact with the soft membranes of the mouth. During the treatment, the following are avoided:

1. There is no use of high speed drills
2. There is no injection to perform in the patient's mouth
3. The use of matrix bands for compound cavities which can cause a lot of pain in young children's mouths is avoided.

6.9 CLINICAL EFFECTIVENESS

The review of literature done in the studies presented in this thesis showed encouraging results; especially by the number of operators using the AgF treatment for arresting caries. Although the sample (35 operators) surveyed by the author is not a true representation of all operators in Sydney, the results obtained showed that most operators (64.8%) favoured the treatment for the arrestment of dental caries.

6.10 COST-ANALYSIS

The cost-benefit and cost-effectiveness of the silver fluoride treatment can be less desirable or poor compared to other self-applied topical fluoride therapies. This is because the technique requires direct attention of the dental personnel to treat the patients. The greater cost of employing dental professionals is usually not balanced by a proportionate increase in the reduction of caries. As such, it is at a disadvantage in public health programmes when the benefits of topical application are sought for large numbers of children. It is only suitable for countries with an abundance of professional personnel

and an extensive programme of dental services in the schools. Davies (1973) has shown that the cost-benefit ratio obtained by employing dental auxiliaries to apply the topical fluoride is better than utilizing the dentist. Thus, in such countries such as New Zealand, Australia and the Cook Islands where clinical facilities, dentists and auxiliaries are already providing dental care to the majority of the school children, the additional time required to carry a silver fluoride treatment application is minimal. Taking this factor into consideration, and the fact that this treatment programme needs to be applied at 6 monthly intervals or when there are signs of recurrent caries, the incorporation of this technique as a preventive measure can be economically efficient.

This treatment procedure is well adapted for use in private practices. It is interesting to note that many private practitioners, and school and hospital clinics in Sydney are employing this treatment therapy in their preventive programmes.

7. CONCLUSIONS

The following conclusions can be drawn from this thesis:

1. There is now a choice open to operators whether to do a restoration or to carry out silver fluoride treatment procedure. There is no question that a good restoration is the best for any cavity, but in situations as in the Cook Islands where the prevalence of dental caries is very high; and where there is an imbalance of the distribution of dental personnel, this chemical treatment seems to be the treatment of choice.
2. Historically, it was seen that many borderline cases of deep-seated decay can be saved by the use of Howe's ammoniacal silver nitrate. There is an indication that silver fluoride topical application can increase caries resistance and may play a role in situations as those occurring in the Cook Islands. This treatment programme can slow or arrest the progression and the development of both the incipient and open carious lesions. This slowing or arresting of caries can be of distinct benefit in primary teeth because firstly, many of the teeth may exfoliate before restorative treatment is done; and secondly, priority can be given to the larger cavities with little risk of the remaining lesions developing too rapidly.

3. As a result of the observations and studies carried out on this AgF treatment in Australia, the same methods can be adopted in the Cook Islands.
4. More studies should be conducted on the economical aspects of operator applied silver fluoride and stannous fluoride therapy when used by dental auxiliaries in school dental clinics or as part of the comprehensive dental care programme.
5. More clinical studies should be conducted on the silver fluoride and stannous fluoride application procedure since there are strong indications showing its potential as a practical preventive measure. They also have the potential to be used by auxiliaries in the field.
6. The application procedure requires dentists and dental auxiliaries to carry out the treatment in the dental clinics. This resulted in the procedure being less cost-effective than any other self-applied topical fluoride therapy. However in countries where clinical facilities and dental personnel are already providing comprehensive dental care to school children, the additional time required to apply the treatment is minimal. This can then be more economically efficient than any other topical fluoride programmes.
7. This treatment procedure can be very useful for the northern islands of the Cook Islands where there are no dental personnel, to enable a greater number of

children to be seen, and lesions treated in short periods when visiting dental personnel are available.

8. The treatment with AgF has another potential advantage in that the treatment is straightforward and atraumatic.

8. REFERENCES

- ABRAHAM M (1947).
Report on food conditions in Rarotonga.
Department of Island Territories, New Zealand.
- ARRINGTON BF (1893).
Nitrate of silver as a remedy for treatment of teeth.
Southern Dent J XII, 143-147.
- ATRRAMADAL A, SVATUM B (1980).
Uptake and retention of tin by *S. mutans*.
Acta Odontol Scand 38:349-354.
- BACKER-DIRKS O (1961).
Post-eruptive changes in dental enamel.
J Dent Res 45:503-511.
- BACKER-DIRKS O (1966).
Post-eruptive changes in dental enamel.
J Dent Res 45:503-511
- BAY I, ROLLA G (1980).
Plaque inhibition and improved gingival condition by use
of a stannous fluoride tooth paste.
Scand J Dent Res 88(4):313-315.
- BECKS H, JENSEN A (1948).
Dental prevention service.
J Dent Res 27, 737.
(Abstract)
- BERMAN DS, SLACK GL (1973).
Caries progression and activity in approximal tooth
surfaces. A longitudinal study.
Br Dent J 134:51-57.
- BIBBY BG (1978).
Dental caries.
Caries Res 12 (Suppl 1) 3-6.
- BLACK GV (1906).
Application of silver nitrate.
D Rev 20:856.
- BOYD JD, WESSELS KE (1951).
Epidemiologic studies in dental caries. Variability of
progression rates of dental cavities in the occlusal
surfaces of second molar teeth.
J Dent Res 31:124.
- BOYD JD, CANNON JJ, LEIGHTON RE (1952).
Epidemiological studies in dental caries. Placement of
fillings as a source of statistical error of estimate as
to dental caries progression rates.
J Dent Res 31:354.

BRYAN LC (1907).
Silver nitrate and preventive dentistry.
D Cosmos 49:1090.

BUNTING RW (1935).
Diet and dental caries.
J Am Dent Assoc 22:114-122.

CRAIG GG (1975).
A useful method for the topical application of fluorides.
Dent Outlook 1:4-5.

CRAIG GG (1981).
Towards a rational approach to topical fluoride therapy
at the mix dentition stage.
Int Dent J 31(2):121-124.

CRAIG GG, DUNN (1971).
Multiple stannous fluoride therapy, surgery procedures in
"A guide to the practice of preventive dentistry."
Dental Health Education and Research Foundation. The
University of Sydney; 20-24.

CRAIG GG, POWELL KR (1979).
Dental caries in susceptible fissures after prolonged
fluoride application.
Comm Dent Oral Epidemiol 7:158-160.

CRAIG GG, POWELL KR, COOPER MH (1981).
Caries progression in primary molars: 24 months results
from a minimal treatment program.
Comm Dent Oral Epidemiol 9:260-265.

DARLING AI (1956).
Studies of the early lesions of enamel caries with trans-
mitted light, polarized light and radiography.
Br Dent J 101:289, 329.

DARLING AI (1968).
Ultrastructure of enamel in relation to mineralization,
demineralization and remineralization as revealed by micro-
radiography, polarized light and plane light microscopy.
Int Dent J 17:648.

DAVIES GN (1956).
Dental conditions among the Polynesians of Pukapuka
(Danger Island).
J Dent Res 350:115-131.

DAVIES GN (1973).
Fluoride in the prevention of dental caries. A tentative
cost benefit analysis.
Br Dent J 135:333-336.

DRISCOLL WS (1974).

The use of dietary fluoride supplements for the prevention of dental caries. Prepared for oral presentation at the "International Workshop on Fluorides and Dental Caries Reductions;" held April 28-May 1, 1974 in Baltimore, M.D. Mimeo.

ENNO A, CRAIG GG (1982).

Economic aspects of the prolonged application paste: Method.

Aust Dent J 27(2):91-93.

FAINE S, HERCUS (1951).

The nutritional status of Cook Islanders.

Br J Nutrition 5:327-343.

FISHER FJ (1968).

A field survey of dental caries, periodontal disease and enamel defects in Tristan de Canha.

Br Dent J 125(10):447-453.

FRANK RM, BRENDALL A (1966).

Ultrastructure of the approximal dental plaque and the underlying normal and carious enamel.

Arch Oral Biol 11:883.

FRANK RM (1968).

The ultrastructure of the tooth from the point of view of mineralization, demineralization and premineralization.

Int Dent J 17:661.

GOLDSWORTHY NE (1958).

The biology of the children of Hopewood House.

Aust Dent J 3(5):309-330.

GRAHAM FW Jr (1941).

Silver nitrate and zinc oxide in the treatment of children's teeth.

J Am Dent Assoc 28:124.

GRANATH L, KAHLMEYER A, MATTSON L, SCHRODER U (1980).

Progression of approximal enamel caries in early teens related to caries activity.

Acta Odontol Scand 38:247-252.

GRAY JA (1965).

Acid dissolution rate of sound and white spot enamel treated with Tin(II) and fluoride compounds.

J Dent Res:493-501.

GUSTAFFSON BE (1953).

Vipeholm (Sweden) dental caries study of the literature on carbohydrates and dental caries.

Acta Odontol Scand 11:206-231.

HANSON WW (1970).

Dental caries in the Cook Islands. South Pacific Commission, Noumea. Information Document No. 21.

HEIFETZ SB (1978).

Cost-effectiveness of topically applied fluoride. "In relative efficiency of methods of caries prevention in dental public health." Workshop proceedings, University of Michigan, June 5-8:69-104.

HINMAN TP (1892).

Causes, treatment and prevention of alveolar abscess. Southern Dent J X:348-353.

HOERMAN KC, KLIMA JE, BIRHO LS (1966).

Tin and fluoride uptake in human enamel in situ: Electron probe and chemical microfilm. J Am Dent Assoc 73:1301-1305.

HOLLENDER L, KOCH G (1969).

Influence of topical applications of fluoride on rate of progress of carious lesions in children: A long-term roentgenographic follow up. Odont Revy 20:37-41.

HOLMES AM (1892).

Advantages of nitrate of silver in dental practice. Am J Dent Sc 26:445.

HOROWITZ H, HEIFETZ S (1979).

Methods for assessing the cost effectiveness of caries preventive agents and procedures. Int Dent J 29:106-117.

HOWE JM (1900).

Notes on silver nitrate. Int Dent J 27:237.

HOWE PR (1917).

Method of sterilizing and at the same time impregnating with metal, affected dentinal tissue. Dent Cosmos 59:891.

HYDE EJ (1973).

Caries-inhibiting action of three different topically-applied agents on incipient lesions in newly erupted teeth: result after 24 months. J Canada Dent Assoc 39:189-193.

IRELAND RL (1939).

Ammoniacal silver nitrate as a sterilizing agent for deep seated decay in deciduous teeth. J Am Dent Assoc 26:871.

- KLEIN H, KNUTSON JW (1942).
Studies on dental caries. XIII. Effect of ammoniacal silver nitrate on caries in the first permanent molar.
J Am Dent Assoc 29:1420-1426.
- KOULOURLIDES T, FEAGIN F, PIGMAN W (1968).
Effect of pH, ionic strength, and cupric ions on the rehardening rate of buffer-softened human enamel.
Arch Oral Biol 13:335-341.
- KUROIWA M (1979).
Use of silver ammonia fluoride for the prevention of caries development for a long time-management of infant dental caries.
Dent Outlook 53(1):85-100.
- LAN WH (1977).
Efficacy of ammoniacal silver nitrate in root canal therapy.
Bull Tokyo Med Dent Univ 24(2):169-176.
- LENZ H (1968).
Ultrastructure of the tooth in respect to mineralization, demineralization and remineralization.
Int Dent J 17:693.
- LEIRSKAR J (1974).
On the mechanism of cytotoxicity of silver and copper amalgams in cell culture system.
Scand J Dent Res 82:74-81 (cited by M.E.J. Curzon, Postgraduate Dental Handbook).
- LEVERETT DH, MCHUGH WD, JENSEN DE (1981).
The effect of daily mouth rinsing with stannous fluoride on dental plaque and gingivitis. Four months' results.
J Dent Res 60:781-784.
- LILIENTHAL B (1956).
Inhibition of acid formation from carbohydrates by stannous fluoride and stannous chlorofluoride.
Aust Dent J 1:165-173.
- MARTHALER TM, GERMANN M (1970).
Radiographic and visual appearance of small, smooth surface caries lesions studied on extracted teeth.
Caries Res 4:224-242.
- MARTHALER TM, WIESNER V (1973).
Rapidly of penetration of radiolucent areas through mesial enamel of first permanent molars.
Helv Odont Acta 17:19-26.
- MECKEL AH (1965).
The formation and properties of organic films on teeth.
Arch Oral Biol 10:585-597.

- MELLBERG JR, RIPA LW (1983).
Fluoride in Preventive Dentistry.
Chicago, Illinois. Quintessence Publishing Co.
- MENAKER L, LEGLER DW (1980).
Etiology, epidemiology and clinical implications of
caries. In "The Biologic Basis of Dental Caries;"
Harper and Row Publishers, 2350 Virginia Avenue, Hagerstown,
Maryland. 21740.
- MILLER CH, KLEINMAN JL (1974).
Effect of microbial interactions on in vitro plaque
formation by *S. mutans*.
J Dent Res 53:427-434.
- MILLER WD (1890).
Micro-organisms of the human mouth.
(Philadelphia, S.S. White Dental Manufacturing Company).
- MILLER WD (1905).
Preventive treatment of teeth with special reference to
silver nitrate.
Dent Cosmos 47:913.
- MILLER WA, MASSLER M (1962).
Permeability and staining of active and arrested dentinal
caries.
J Dent Res 44:1389.
- MUHLER JC, SPEAR LB Jr, BIXLER D, STOOKEY GK (1967).
The arrestment of dental caries in adults after the use
of three different forms of SnF_2 therapy: Results after
30 months.
Am Dent Assoc:1402-1406.
- MURRAY JJ, MAJID ZA (1978).
The progression of approximal caries in the deciduous
dentition in British children.
Br Dent J 145:161-164.
- NESSON JH (1924).
Pedodontia with special reference to the use of Howe's
silver nitrate.
Dent Cosmos 66:62.
- NEWBRUN E (1983).
Cariology - Williams and Wilkins, Baltimore London.
2nd Edition.
- NISHINO M (1969).
Studies on the topical application of ammoniacal silver
fluorides for the arrestment of dental caries.
J Osaka Univ Dent Soc 14:1-14 cited from Oral Res Abstr
1970:5:395.

ORLAND FJ, BLAYNEY JR, HARRISON RW, ERVIN RF, REYNIERS JA, TREXLER PC, GORDON HA, WAGNER, MORRIS (1955).
Experimental caries in germfree rats inoculated with enterococci.
J Am Dent Assoc 50:259-272.

PARFITT GJ (1956).
The speed of development of the carious cavity.
Br Dent J 100:204.

PARKER JM (1894).
Nitrate of silver and its application in diseased conditions of the mouth and teeth.
Dent items of interest XVI:680-682.

PIERCE CW (1894).
New mode of using nitrate of silver. Ash's Quart. 20
cited by Seltzer S et al.
J Am Dent Assoc 28:1586-1594, 1941.

PITTS NB (1983).
Monitoring of caries progression in permanent and primary posterior-approximal enamel by bitewing radiography.
Comm Dent Oral Epidemiol 11:228-235.

POWELL KR, BARNARD PD, CRAIG GG (1981).
Effects of stannous fluoride treatment on the progression of initial lesions in approximal surfaces of permanent posterior teeth.
J Dent Res 60:1648-1654.

PRIME JM (1935).
Further extending of the use of Howe's ammoniacal silver nitrate in control of dental caries.
Dent Cosmos 77:1046.

PRINZ H (1905).
Silver nitrate - A Pharmaco-Therapeutic Study. (Treatment of children's teeth and caries).
Dent Digest XI:1198-1202.

RENSON CE (1983).
Interim report on data received from selected countries on caries, periodontal disease, preventive programmes, sugar consumption and fluoridated dentifrices.
FDI working paper, CORE Joint Working Group 5.

RENSON CE (1984a).
Changing global trends in dental caries and periodontal disease.
World Dental Congress, Helsinki, Finland.

RENSON CE (1984b).
Changing patterns and concepts of oral disease: the effects on future manpower needs.
Dentistry Today Journal. A review of the world literature.
Vol 1, 3:1-8.

- ROSE TP (1976).
Sterilization of carious dentine with silver nitrate.
Chron Omaha Dist Dent Soc 39(8):151.
- RUGG-GUNN AJ (1972).
Approximal carious lesions: A comparison of the radiological and clinical appearance.
Br Dent J 133:481-484.
- SELTZER S, WERTHER L (1941).
Conservative silver nitrate treatment of borderline cases of deep dental caries.
J Am Dent Assoc 28:1586-1594.
- SHWARTZ M, GRONDAHL HG, PLISKIN JF, BOFFA JS (1984).
A longitudinal analysis from bitewing radiographs of the rate of progression of approximal carious lesions through human dental enamel.
Arch Oral Biol 29, 7:529-536.
- SILVERSTONE LM (1973).
Oral Sciences Review.
No 3; Dental Enamel, Eds. AH Melcher and GA Zarb: 100-160, Munksgaard, Copenhagen.
- SILVERSTONE LM (1977).
Remineralization phenomena.
Caries Res 11:59-84.
- SILVERSTONE LM (1982).
The effect of oral fluid and synthetic calcifying fluids in vitro on the remineralization of enamel lesions.
Clin Prev Dent 4:13-22.
- SILVERSTONE LM (1983).
Remineralization and enamel caries: Significance of fluoride and effect on crystal diameters. In Demineralization and Remineralization of the Teeth Edited by SA Leach, WM Edgar, Oxford, England IRL Press Ltd.
- SPEAKE JD (1976).
Report on a visit to the Cook Islands.
South Pacific Commission, Noumea, New Caledonia.
- SPEAKE JD, CUTTRESS TW, BALL ME (1979).
The prevalence of dental caries and the concentration of fluoride in enamel of children in Pacific Islands.
South Pacific Commission, Noumea, New Caledonia.
- STEBBINS EA (1891).
Nitrate of silver as a therapeutic agent in dentistry.
Ash's Quarterly 271. Selected Inter Dent J XII:661-671.
- STEPHAN RM (1944).
Intra-oral hydrogen ion concentrations associated with dental caries activity.
J Dent Res:257-266.

SURVEY DEPARTMENT, RAROTONGA (1980).

Maps of the Cook Islands.

Published by the Department of Internal Affairs, Rarotonga, Cook Islands.

SZABO J (1902).

Method in which nitrate of silver affects carious dentin.

Dent Digest 8:573 cited by Seltzer et al.

J Am Dent Assoc 28:1586-1591 (1941).

THIBODEAU EA, HANDELMAN SL, MARQUIS RE (1978).

Inhibition and killing of oral bacteria by silver ions generated with low intensity direct current.

J Dent Res 57:922-926.

VIDENOV TL (1975).

Report on proposals for improvements to the Rarotonga water supply.

WHO Region of Western Pacific:135.

WALER SM, ROLLA G (1980).

Plaque inhibiting effect of combinations of chlorhexidine and the metal ions zinc and tin. A preliminary report.

Acta Odontol Scand 38(4):213-217.

ZANDER HA (1941).

Use of silver nitrate in the treatment of caries.

J Am Dent Assoc 28:1260-1267.

ZANDER HA, SMITH HW (1945).

Penetration of silver nitrate into dentin II.

J Dent Res 24:121-128.

APPENDICESSUMMARY OF SURVEY

- I. A survey concerning the use of silver compounds for the treatment or arresting of dental caries, and prevention of dental caries was carried out by the author. This was done in the form of questionnaires directed to the operators using the compounds in the clinics. There were 37 operators questioned; from hospital clinics, school dental clinics and private clinics in Sydney.
- II. The operators were asked:
- (a) if they use silver compounds for the treatment or arresting of dental caries, or for preventing dental caries.
 - (b) the methods they use.
 - (c) if they like using silver compounds for the treatment or arresting of dental caries, or for preventing dental caries.
 - (d) if they use the compounds because they have to (its usage is a formal procedure in the clinic) for arresting dental caries or for preventing dental caries.
 - (e) if they will continue to use the compound in future because of its beneficial effect for arresting dental caries or for preventing dental caries.
 - (f) for any general comments on the use of the method.

III. Number of operators who used silver compounds

For:

Arresting caries: YES: 65% (24) NO: 32% (12)

DID NOT ANSWER: 3% (1)

Preventing caries: YES: 16% (6) NO: 73% (27)

DID NOT ANSWER: 11% (4).

IV. Method of application used

(i) Remove loose caries and food debris, etc.

(ii) Dry tooth and isolate with cotton rolls.

(iii) Apply 40% AgF to treatment area for
3 minutes.

(iv) Then apply 10% SnF₂ spot application paste.

(v) Cover with stomahesive wafer.

The patient is informed not to eat for 1 hour
and also told that the wafer will dissolve in the
mouth.

The number of operators using the outlined method:

76% (28).

Those not using method: 5% (2)

Did not answer: 19% (7).

V. Number of operators who like using silver compounds for:

Arresting caries: YES: 65% (24)

NO: 30% (11)

DID NOT ANSWER: 5% (2).

Preventing caries: YES: 16% (6)
 NO: 65% (24)
 DID NOT ANSWER: 19% (7)

VI. Number of operators using silver compounds because they have to, for:

Arresting caries: YES: 24% (9)
 NO: 70% (26)
 DID NOT ANSWER: 6% (2).

Preventing caries: YES: 11% (4)
 NO: 73% (27)
 DID NOT ANSWER: 16% (6).

VII. Number of operators who would continue to use silver compounds in future for:

Arresting caries: YES: 68% (25)
 NO: 30% (11)
 DID NOT ANSWER: 2% (1).

Preventing caries: YES: 27% (10)
 NO: 54% (20)
 DID NOT ANSWER: 19% (7).

VIII. Operators favouring recommended treated procedure

- (i) 22% (8 operators) commented that the method was messy.
- (ii) 44% (16 operators) favoured the use of the method.

- (iii) 34% (13 operators) did not comment on the method.

CONCLUSION

Even if the sample questioned do not represent the total operators in Sydney, the observations clearly indicate that:

1. Silver compounds (AgF) are effective in arresting dental caries.
2. Most operators interviewed like using silver compounds for arresting dental caries.
3. The method of application was favoured by most operators who would continue to use the compound for arresting dental caries.

QUESTIONS CONCERNING USE OF SILVER COMPOUNDS
FOR PREVENTION OF DENTAL CARIES

T. TAIREA (MDS Graduate Student)

- | | | | |
|----|---|-----|----|
| 1. | Do you use silver compound(s) for
the treatment or arresting of dental caries? | YES | NO |
| | or for the prevention of dental caries? | YES | NO |

2. What method(s) do you use?

- | | | | |
|----|--|-----|----|
| 3. | Do you like using silver compound(s) for
arresting dental caries? or | YES | NO |
| | for preventing dental caries? | YES | NO |

Comments (expand)

4. Are you using the silver compound(s) because you have to?

(In other words, its usage is a formal procedure in the practice)

- | | | | |
|--|------------------------------|-----|----|
| | arresting dental caries? | YES | NO |
| | prevention of dental caries? | YES | NO |

5. Would you continue to use the compound in future because of its beneficial effect for

- | | | | |
|--|------------------------------|-----|----|
| | arresting dental caries? | YES | NO |
| | prevention of dental caries? | YES | NO |

6. Any general comments on the use of the method(s)?

CRAIGS TREATMENT PROCEDURES AND

SOME BASIC RULES

(Taken from the clinical notes
for Fourth and Fifth year
students).

- USE - Sharp excavators whenever possible to remove caries. With practice an operator can excavate right up to vital pulp tissue with negligible discomfort to the patient.
- USE - Bite-wing radiographs whenever possible to establish a logical treatment plan. A great deal of useful information can be obtained from good bite-wing radiographs including data on depth of lesions, furcation pathology, root length, position of permanent successors etc.
- USE - Junior Garmers Clamps for lower arch isolation. They are mandatory when silver fluoride is being used.
- MINIMISE - Use of high-speed handpiece. The noise and water spray can sometimes cause apprehension in a new patient.
- MINIMISE - Use of high volume suction apparatus with a new patient. When using the low volume type place tip in muco-buccal fold whenever possible.

Before Starting

1. Obtain an Adequate History

During history taking the patient and the parent should be asked separately about any symptoms of pain. It is worth remembering that the main causes of pain in the deciduous dentition, in decreasing order, are:

Food impaction

Tooth mobility caused either by imminent exfoliation or a chronic alveolar abscess

Pulpitis

Very early stages of a chronic alveolar abscess before the abscess has pointed

2. Plan the Treatment

Bite-wing radiographs are extremely useful in treatment planning. Apart from the aspects mentioned previously, good bite-wing radiographs enable one to assign approximal and, frequently, occlusal lesions to one of the following categories:

Category 1 - Lesions involving enamel and some dentine

Category 2 - Open lesions with no obvious pulpal involvement

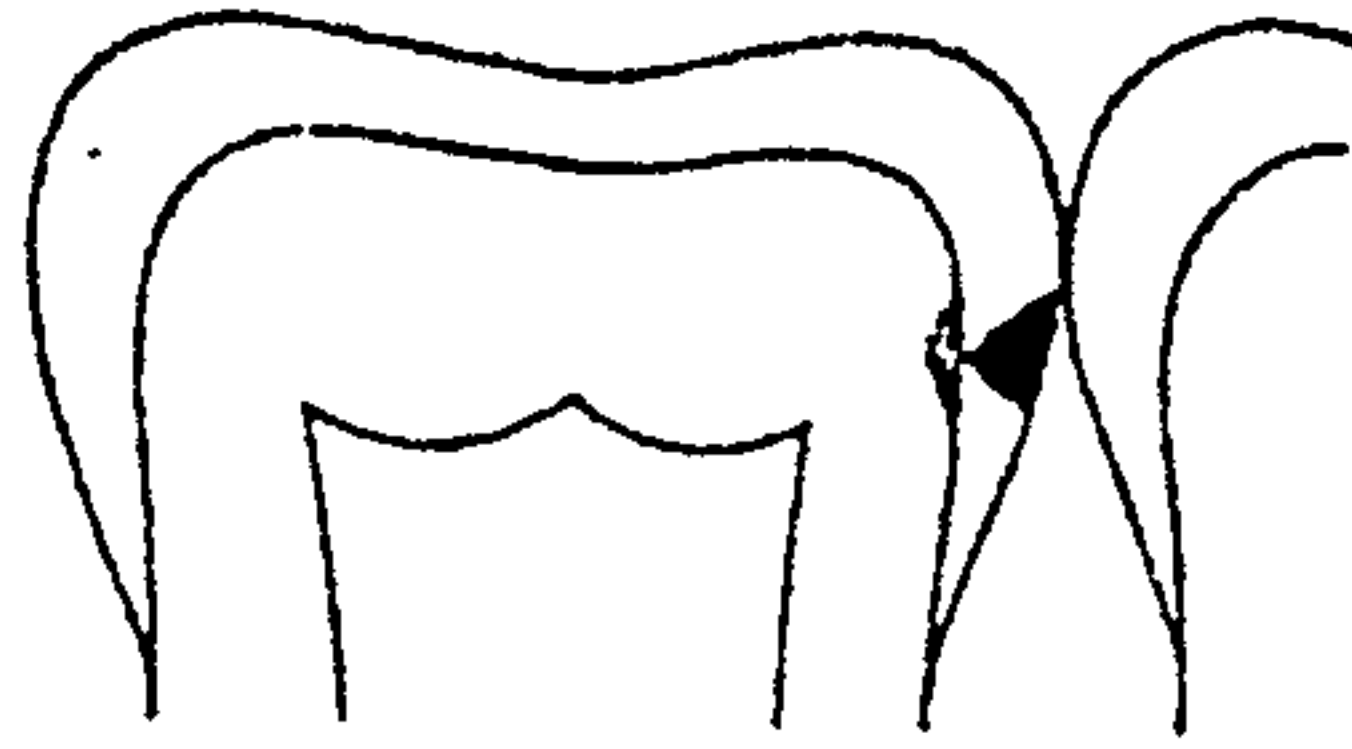
Category 3 - Obvious pulp exposure

TREATMENT PROCEDURES

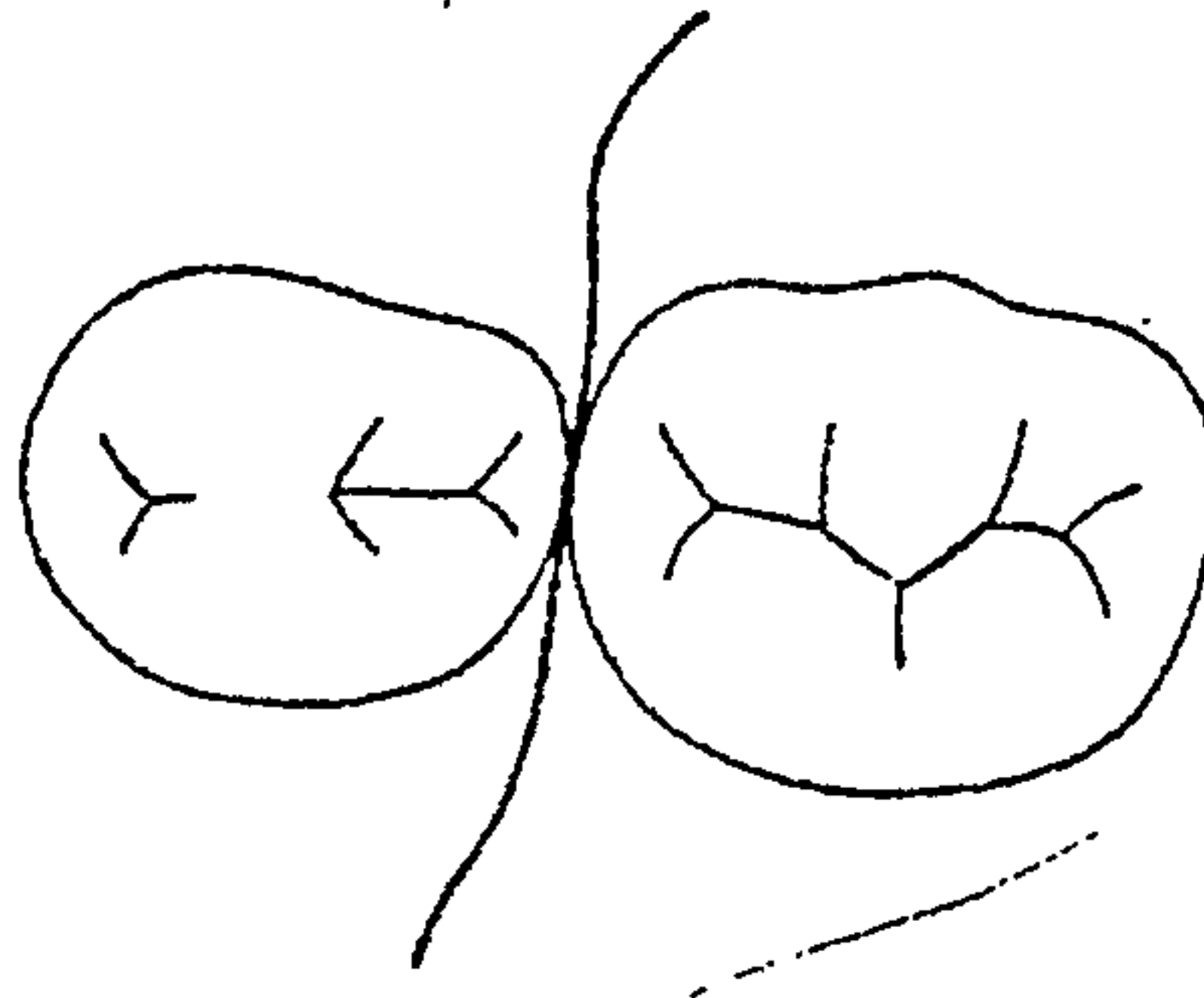
CATEGORY 1

Lesions involving enamel and some dentine

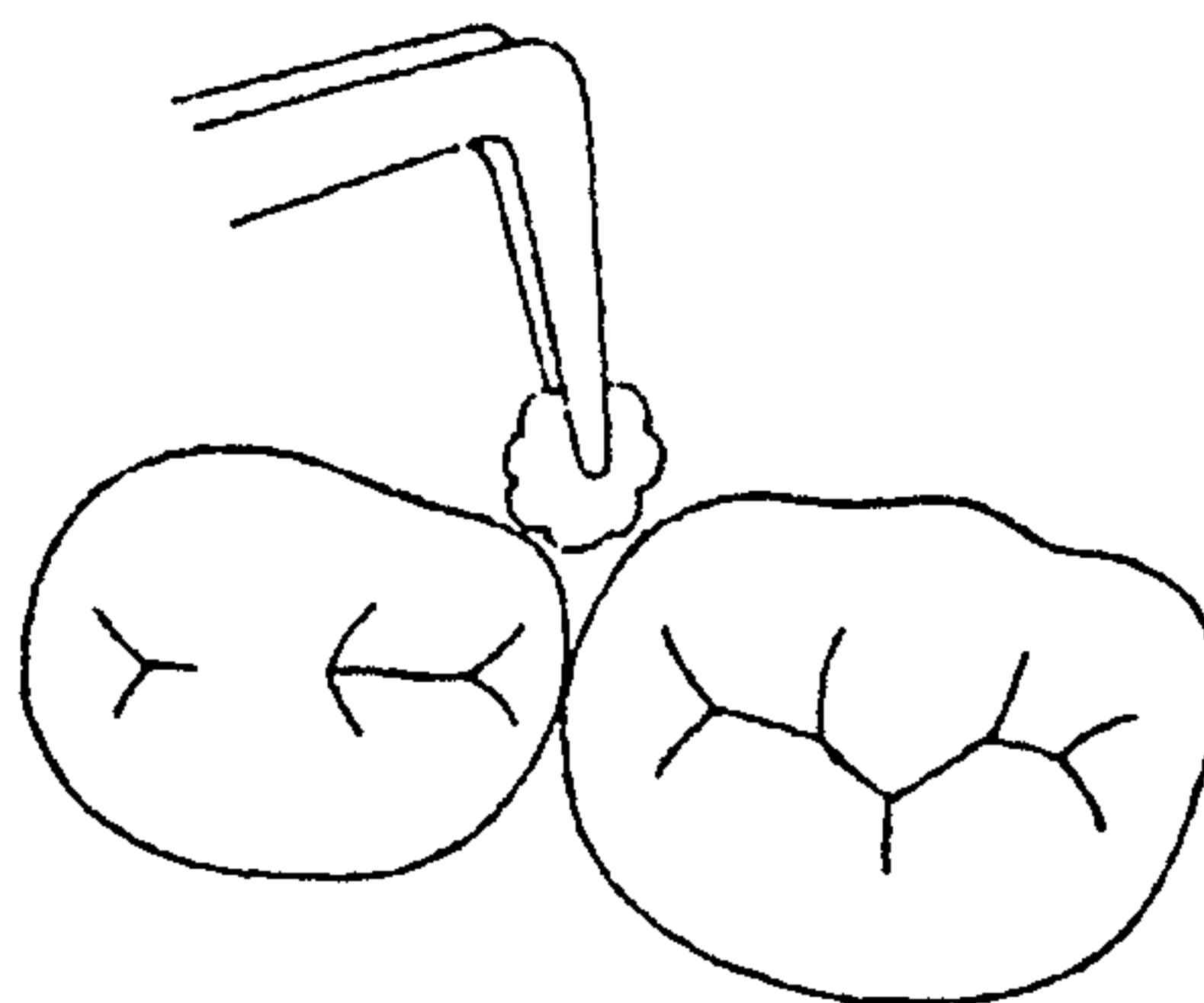
In the case of lesions in approximal surfaces, the overlying marginal ridge is intact.



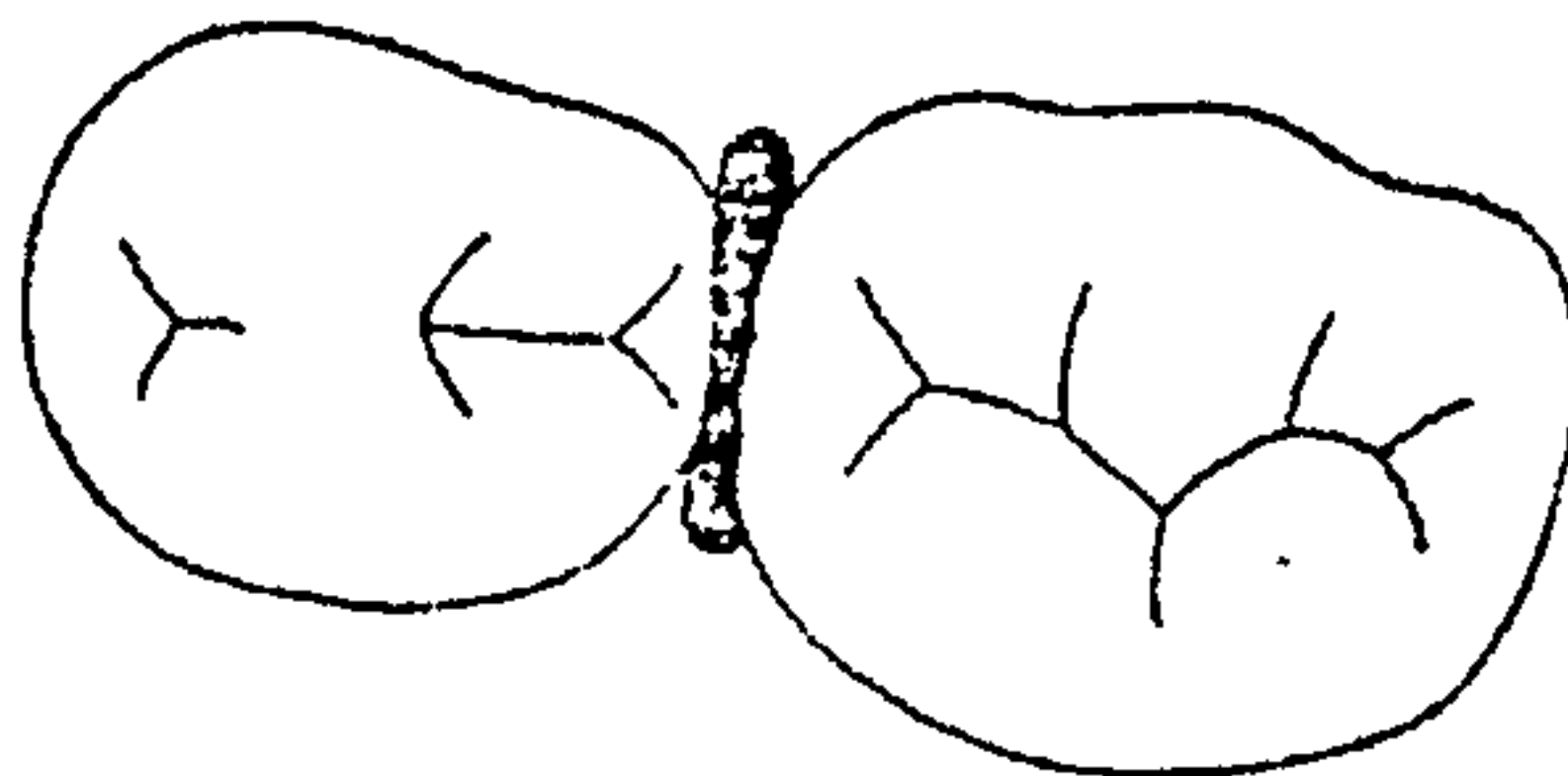
1. Remove plaque from interproximal region using dental floss.



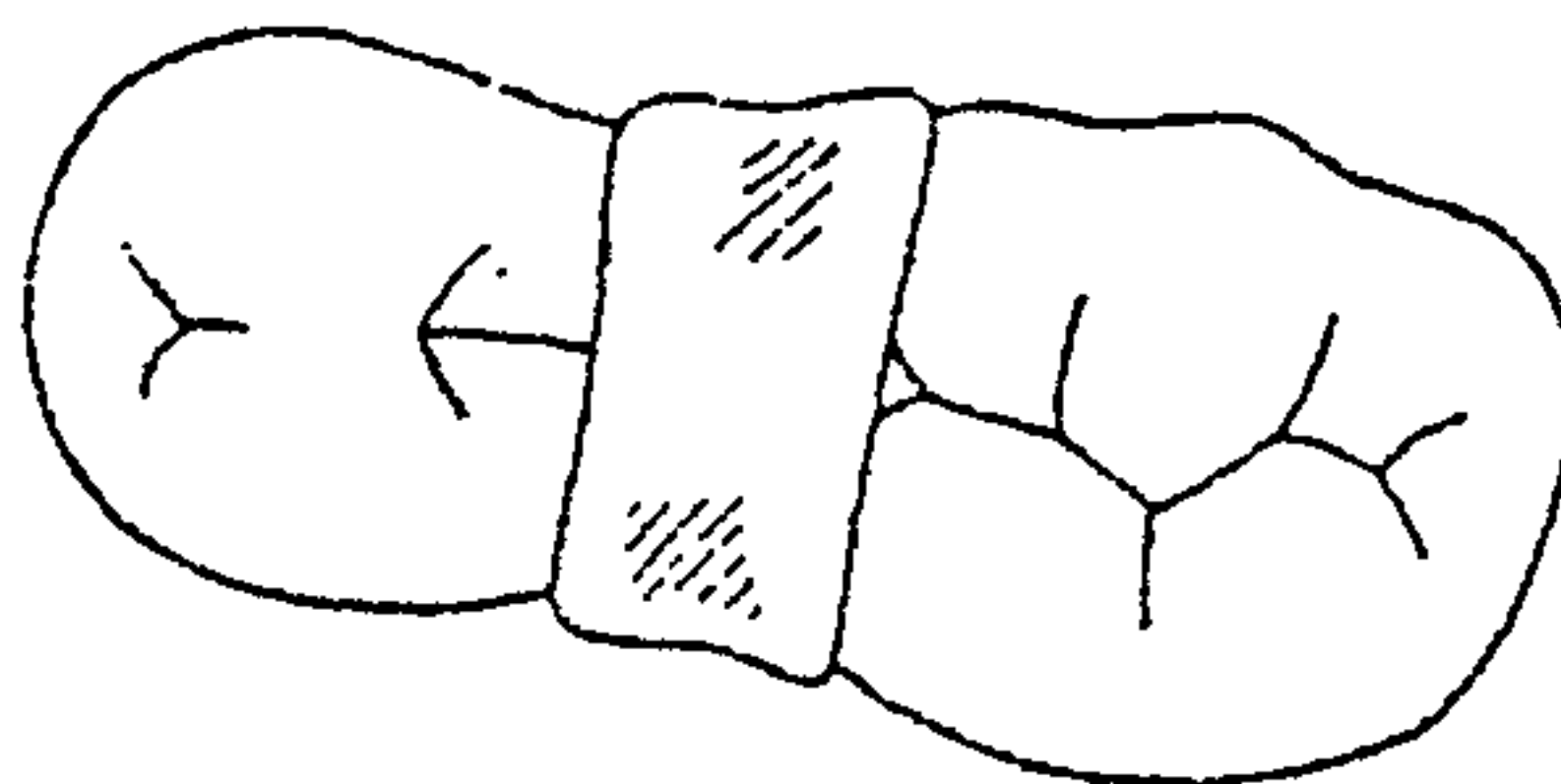
2. Isolate area to be treated with cotton rolls. In the case of lower arch isolation, Junior Garmers Clamps are mandatory.
3. Place one drop of A.G.F. brand silver fluoride on a waxed paper or plastic surface.
4. Apply silver fluoride on pellet of cotton wool to region occlusal to the contact area then move pellet to the regions lingual and buccal of the contact area. Leave pellet in buccal region and move it gently in and out for 3 minutes. Do not attempt to draw silver fluoride through a contact area with dental floss unless one is absolutely sure that meticulous isolation can be maintained throughout.



5. Apply a very thin layer of Floran Spot Application Paste to the regions occlusal, lingual and buccal to the contact area.

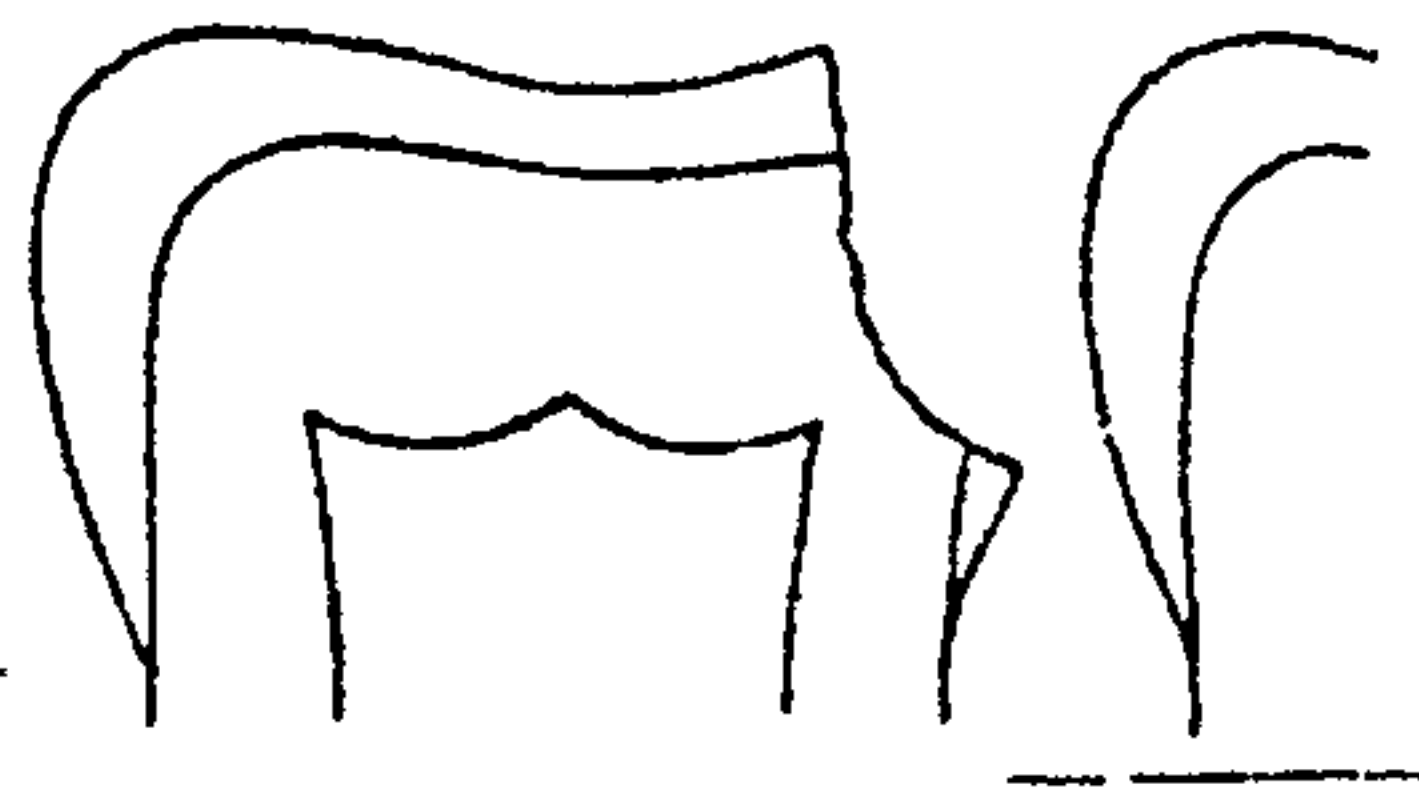


6. Cut out a small piece of Stomahesive Wafer (approximately 5 mm wide by 10 mm long) and carefully adapt over treated site. . Adaptation of the wafer is facilitated if it is warmed before use by either placing it near a sterilizer or asking the patient to hold it for a few minutes in his hand.

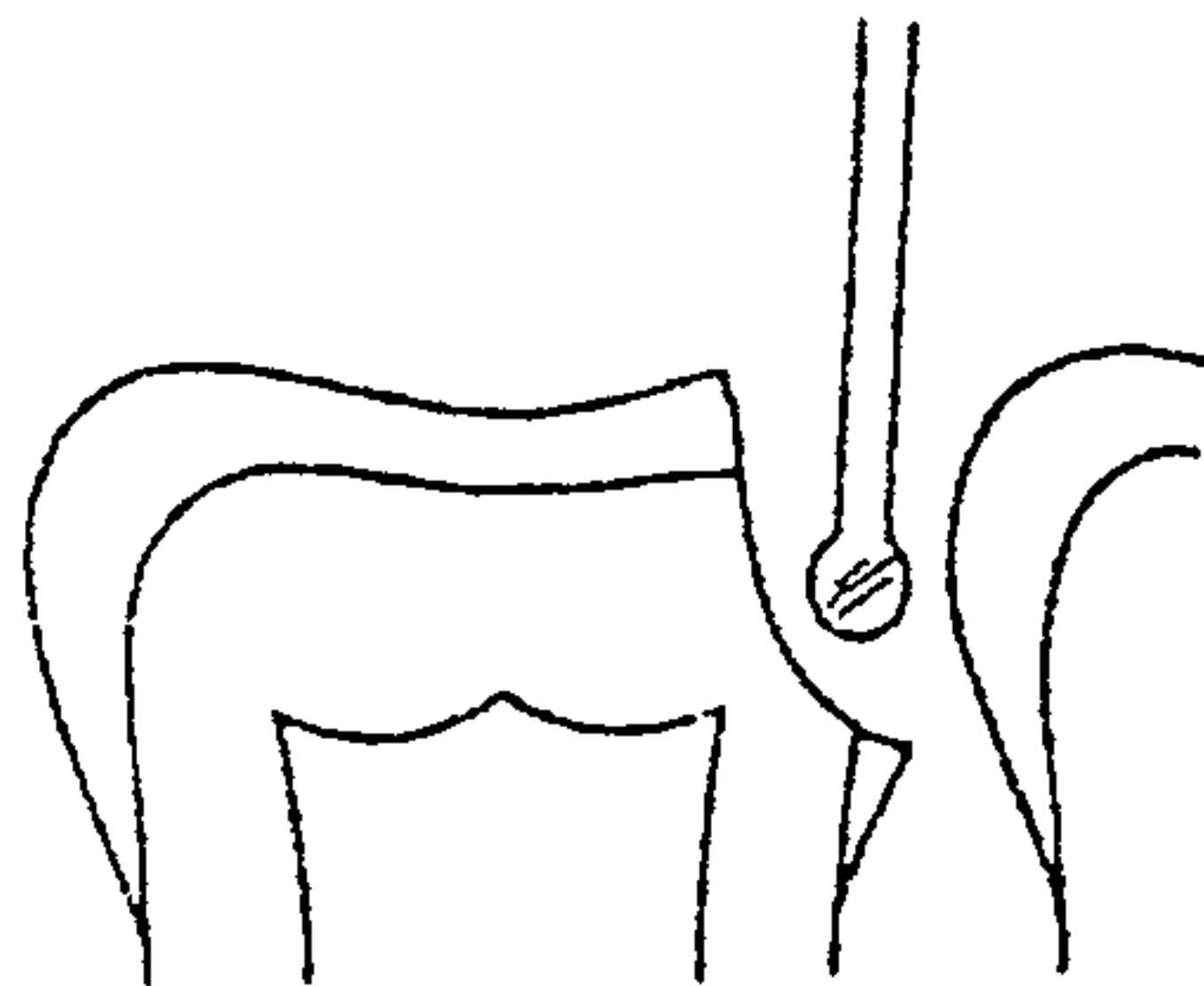


7. Ask the patient not to eat for one hour. Inform the patient and his parents that the wafer will slowly dissolve in the mouth fluids.

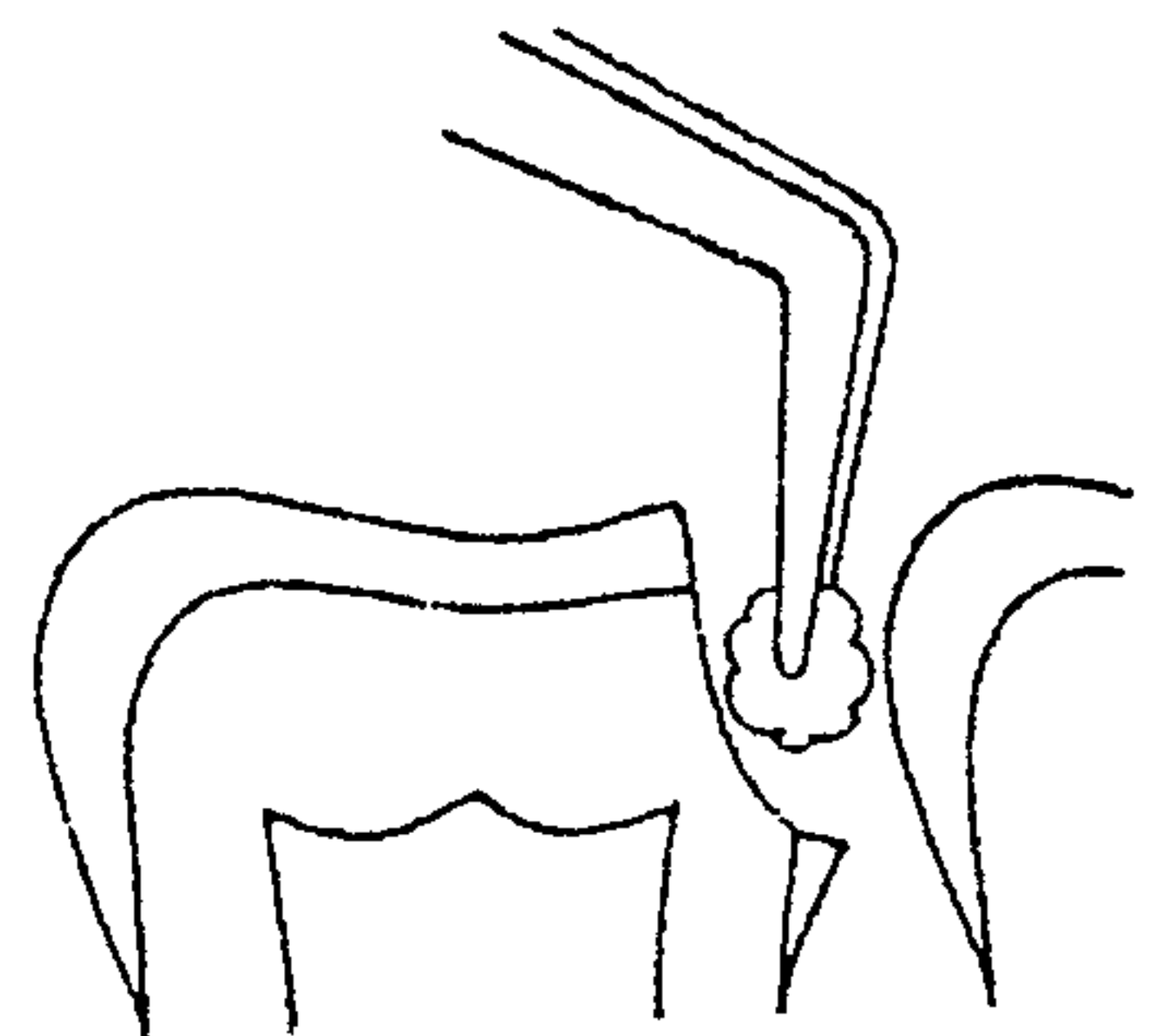
CATEGORY 2

Open lesions with no obvious pulpal involvementA. No history of pain

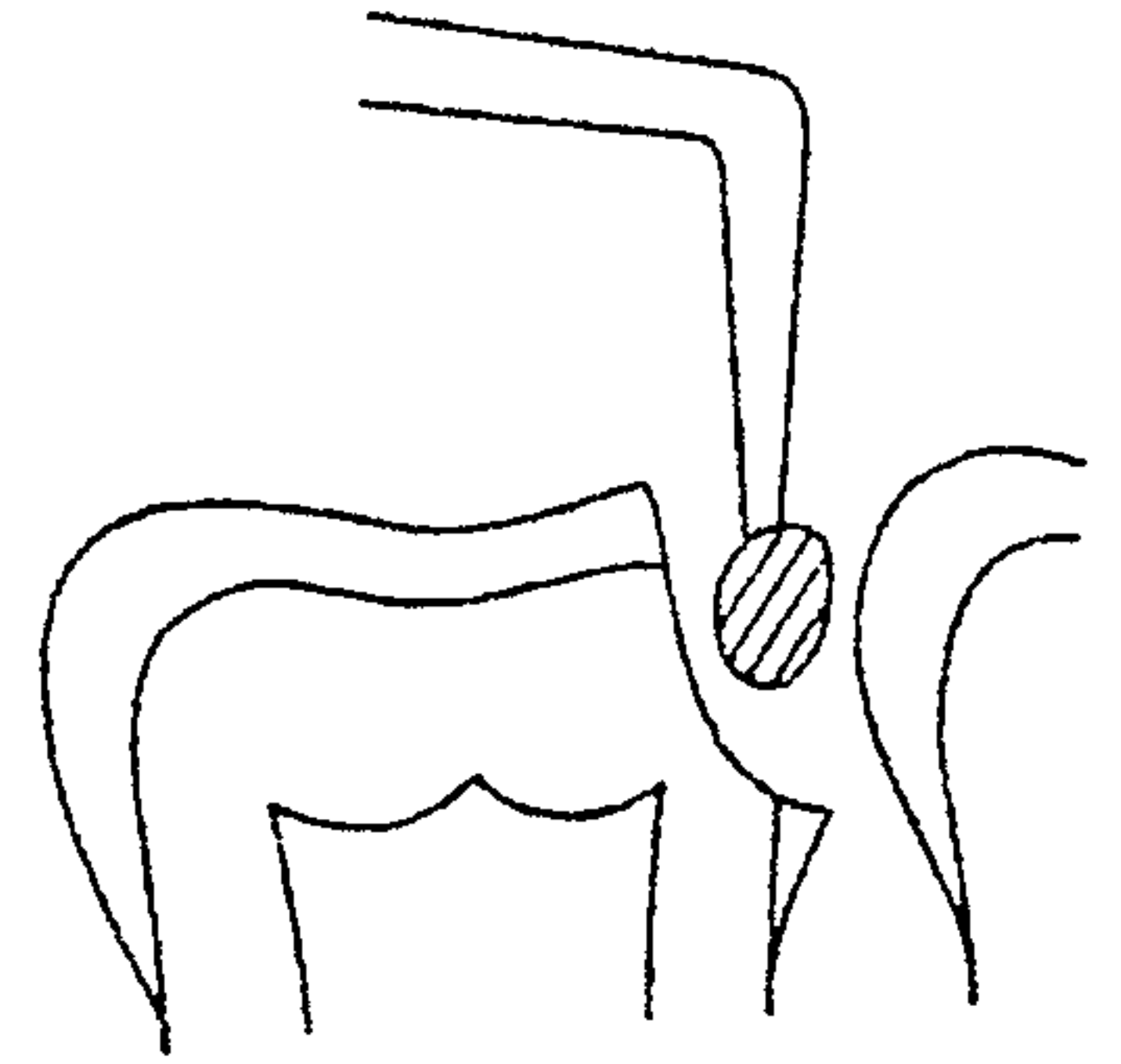
1. Isolate area to be treated with cotton rolls. As in Category 1 treatments, Junior Garmers Clamps are mandatory for lower arch isolation.
2. Remove any obvious pieces of food from the lesion with an excavator trying not to remove any of the caries in the process.



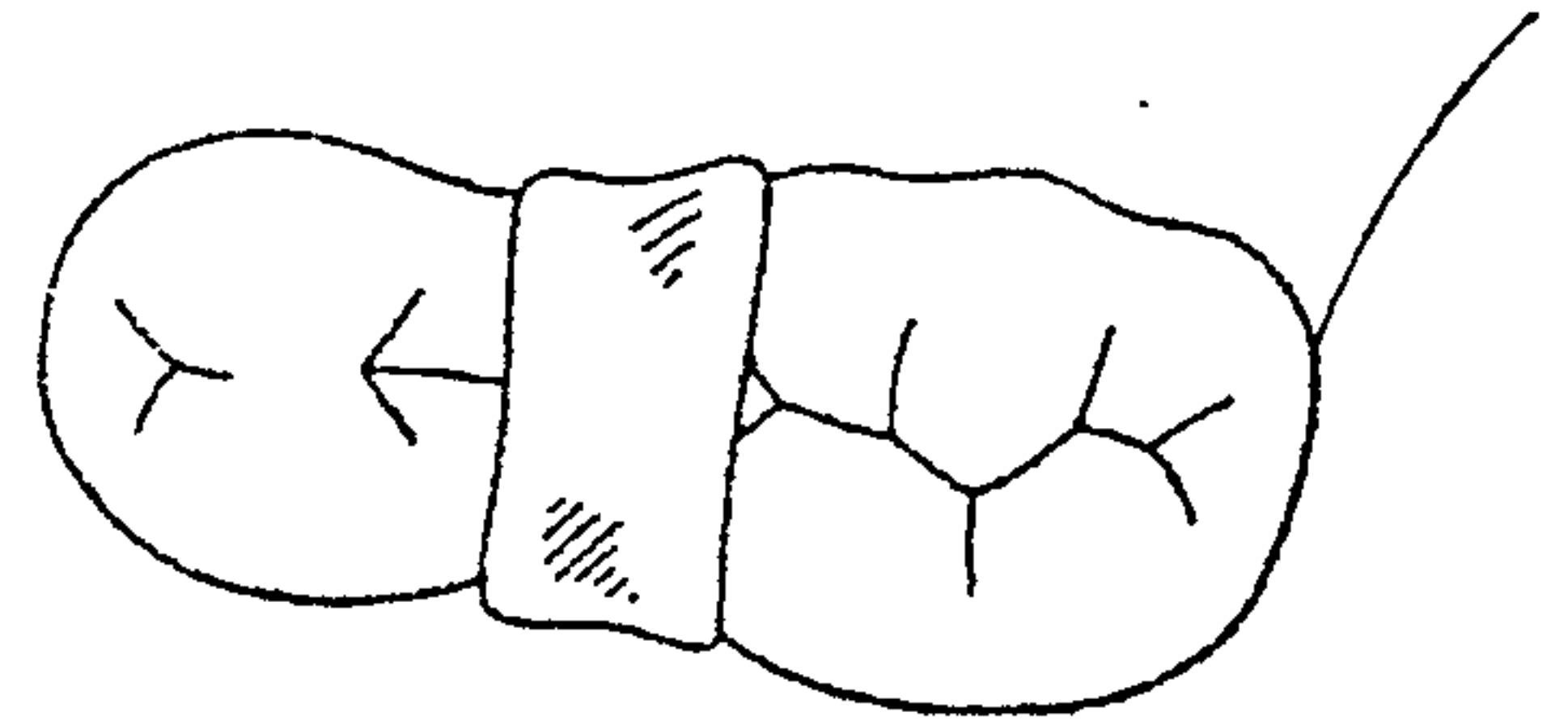
3. Place one drop of silver fluoride on a waxed paper or plastic surface.
4. Apply silver fluoride to lesion on a pellet of cotton wool. Move cotton wool continuously with the point of a probe for the entire 3 minute application period.



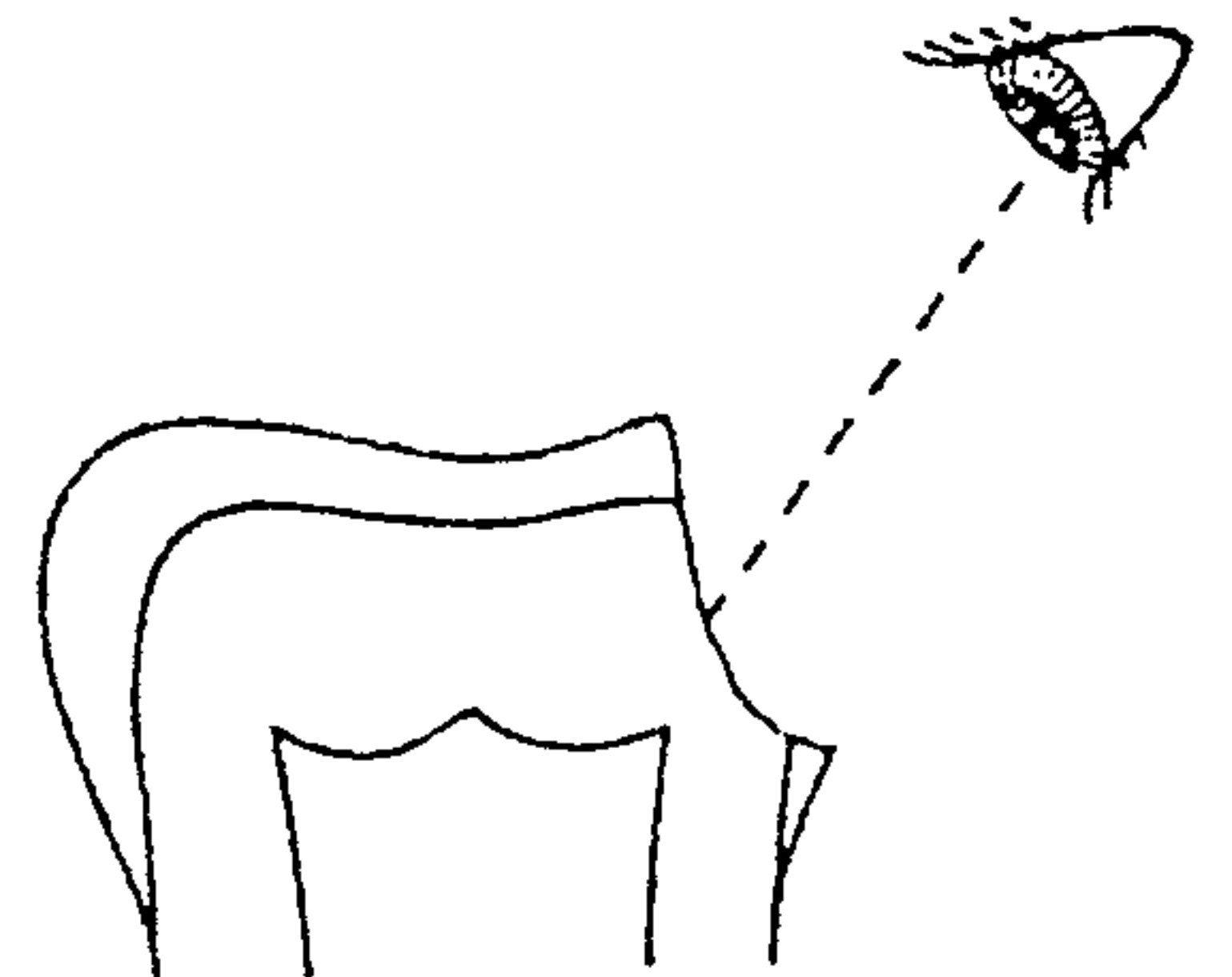
5. Apply a thin layer of Floran Spot Application Paste to lesion. It is important that the cavity is not overfilled with paste as unnecessary pressure may be developed within the cavity during adaptation of the Stomahesive Wafer (Step 6). Pain may result in such circumstances.



6. Cut out a small piece of Stomahesive Wafer (approximately 5 mm wide by 10 mm long) and carefully adapt to enamel around the periphery of the lesion. Try not to force wafer into the lesion itself as this may cause pain in situations where the lesion is particularly deep.



7. One or two weeks later carefully examine the surface of the treated lesion. The entire surface should have a black mat appearance. If it does not, check that the non-pigmented area is not a pulp exposure. If the non-pigmented area is carious dentine repeat Steps 3 to 6. Lesions which have responded correctly are then prepared for a glass ionomer cement restoration.



SPECIAL CIRCUMSTANCES FOR EXPERIENCED OPERATORS

For open carious lesions in occlusal or approximal surfaces when there has been NO HISTORY OF PAIN OF PULPAL ORIGIN and when radiographs show a DISTINCT LAYER OF SOUND DENTINE between the base of the carious lesion and the pulp, the treatment can be modified from that just described.

In such cases, the tooth is prepared for a glass ionomer cement restoration then AgF solution is applied using Steps 1, 3, and 4 (see page 4 of previous procedure). A 3 minute AgF application time is not required in these circumstances. Once AgF is applied it is then dried under a gently stream of air from an air/water syringe. The dried AgF has a golden yellow colour. The cavity is then restored with glass ionomer cement following the procedures outlined.

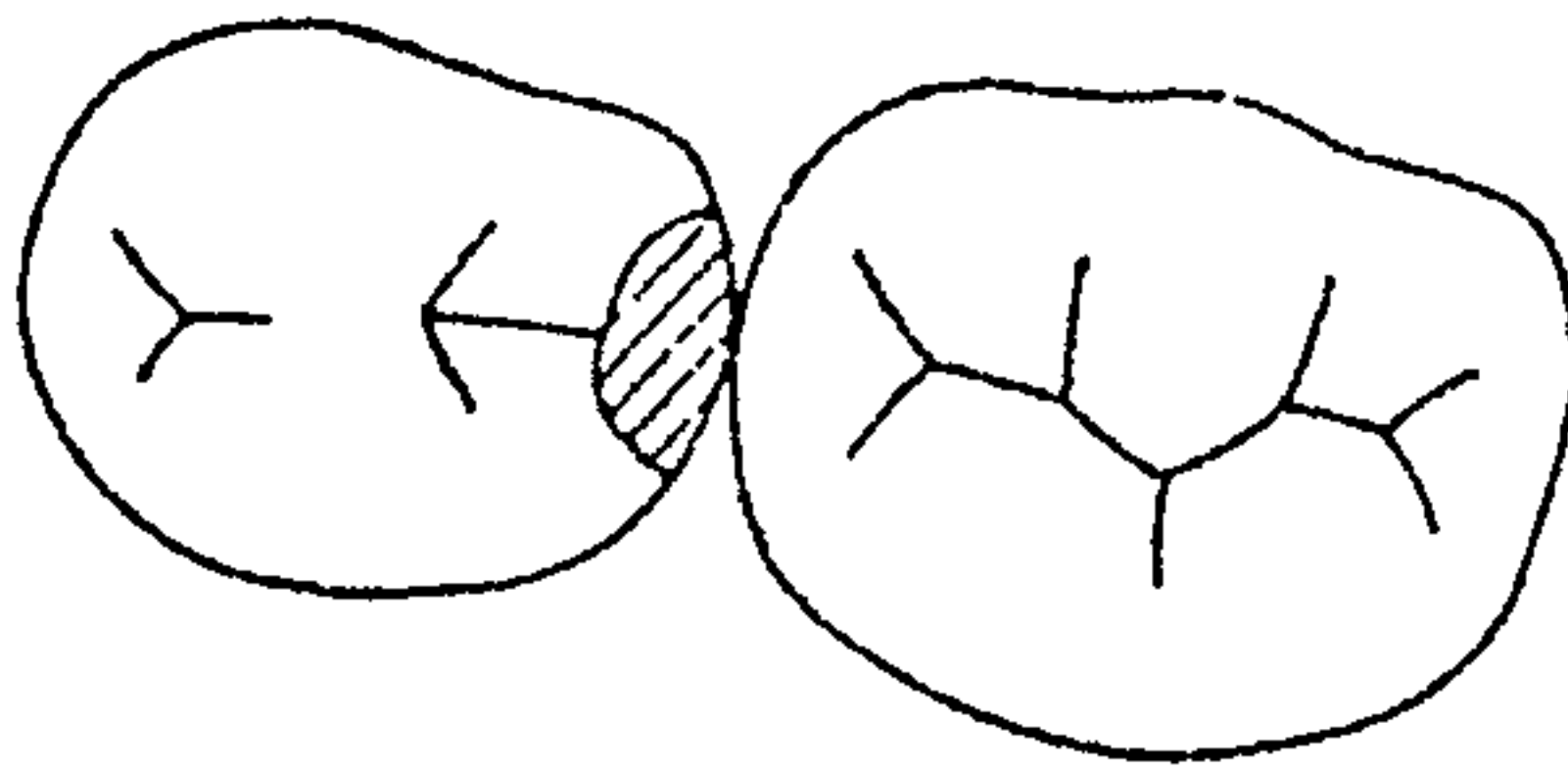
B. History of Pain

Pain from Food Impaction

A very common cause of pain in the deciduous dentition is the impaction of fibrous foods (meat, chicken etc.) between teeth. The main symptom is pain after chewing.

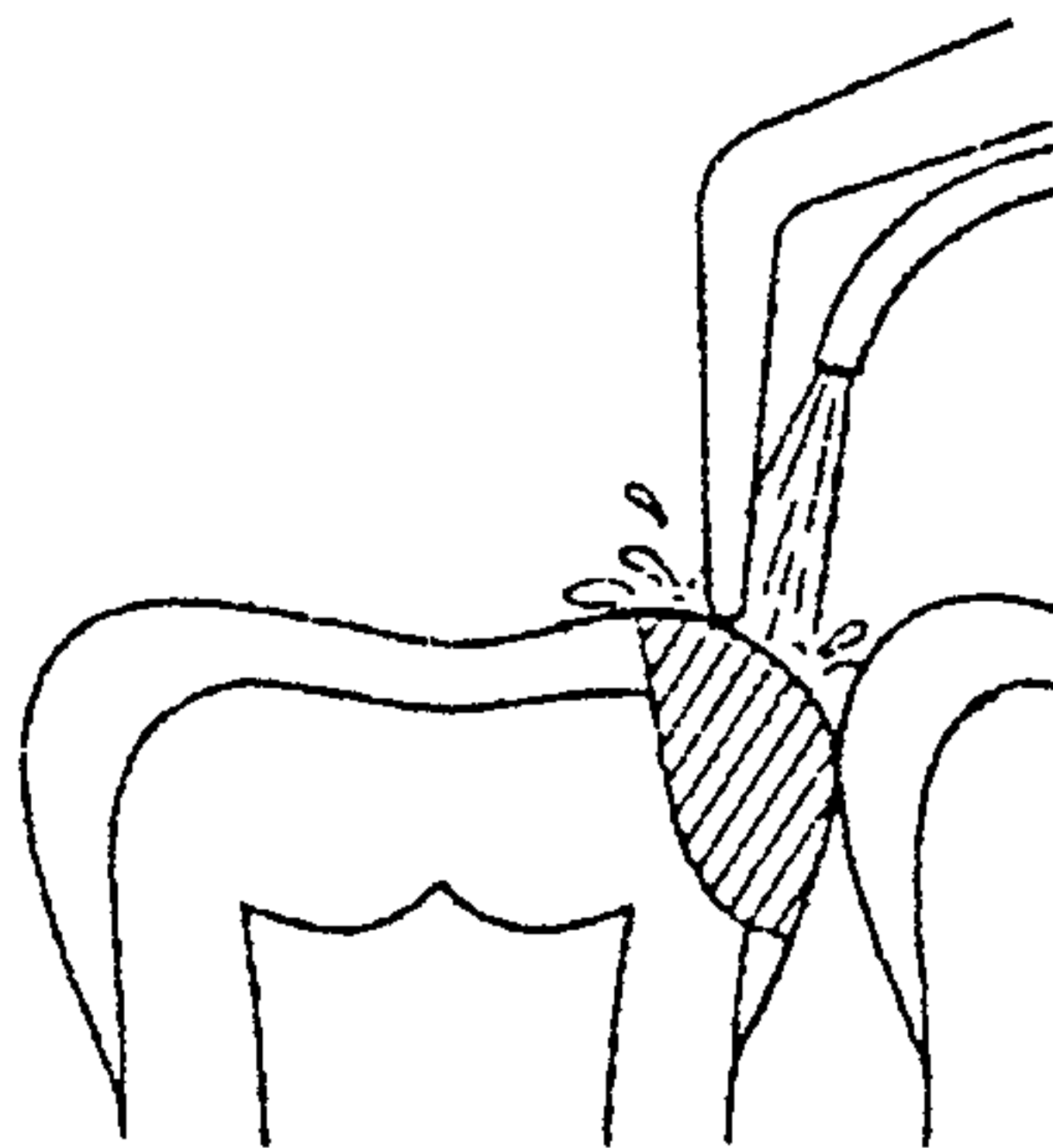
1. Check the bite-wing radiographs to ensure that there is a discernible layer of "sound" dentine between the base of the lesion and the pulp. If such a layer is absent treat tooth as though it had a pulp exposure.
2. Lift layers of impacted food from between the teeth with a probe taking care not to exert pressure on the underlying gingiva during the operation. Pressure will cause discomfort.
3. Place one drop of silver fluoride on a waxed paper or plastic surface.
4. Apply silver fluoride to lesion on a pellet of cotton wool. Move cotton wool continuously with the point of a probe for the entire 3 minute application period.
5. Dry lesion with a pellet of cotton wool.

6. Mix IRM to a medium consistency and place over lesion. Do not carry out any cavity preparation prior to IRM placement.



7. Recall patient. If symptoms have subsided the IRM is replaced after one to two weeks with a glass ionomer cement restoration.

Use a round bur in an ultra-slow speed handpiece, an ultrasonic scaler or a Morse 00 scaler to remove IRM restorations.



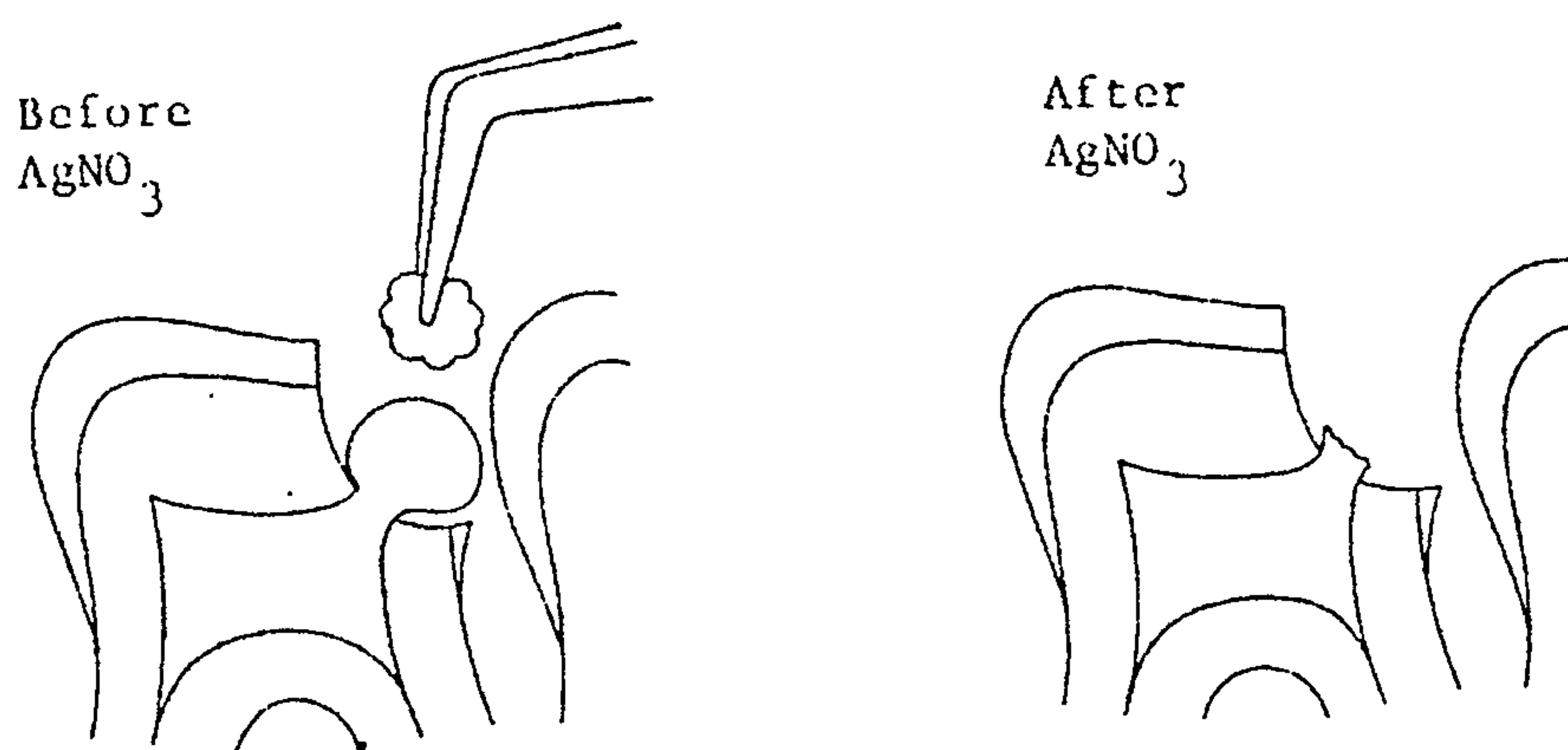
B. No History of Pain

Completely or Partially Necrotic Pulp

Treatment is identical to that described under heading "Chronic Alveolar Abscess".

Chronic Hyperplastic Pulpitis

With chronic hyperplastic pulpitis, tissue grows out from an exposure site into the cavity. Invariably such a condition gives little or no discomfort to the patient. The treatment is identical to that described under the heading "Pulpitis" except that the tissue is first reduced in size through the use of ammoniacal silver nitrate. A pellet of cotton wool soaked in this solution is placed on the tissue and left, under constant observation, for 3 to 5 minutes. At the end of this period the tissue will be found to have shrunk markedly and one can proceed with the next stages. (Ammoniacal silver nitrate consists of 3 g AgNO_3 , 1 ml distilled water and 5 ml strong ammonia).



Root Stumps

Root fragments from deciduous teeth can be excellent space maintainers and should be retained whenever possible. If a piece of tooth is projecting occlusally from a root fragment it may cause discomfort when the patient chews. In such circumstances the offending projection is removed using a bur in a high speed handpiece until it is flush with the gingival margin. Several situations that are encountered clinically are illustrated:

PLACEMENT OF GLASS IONOMER CEMENT RESTORATIONS

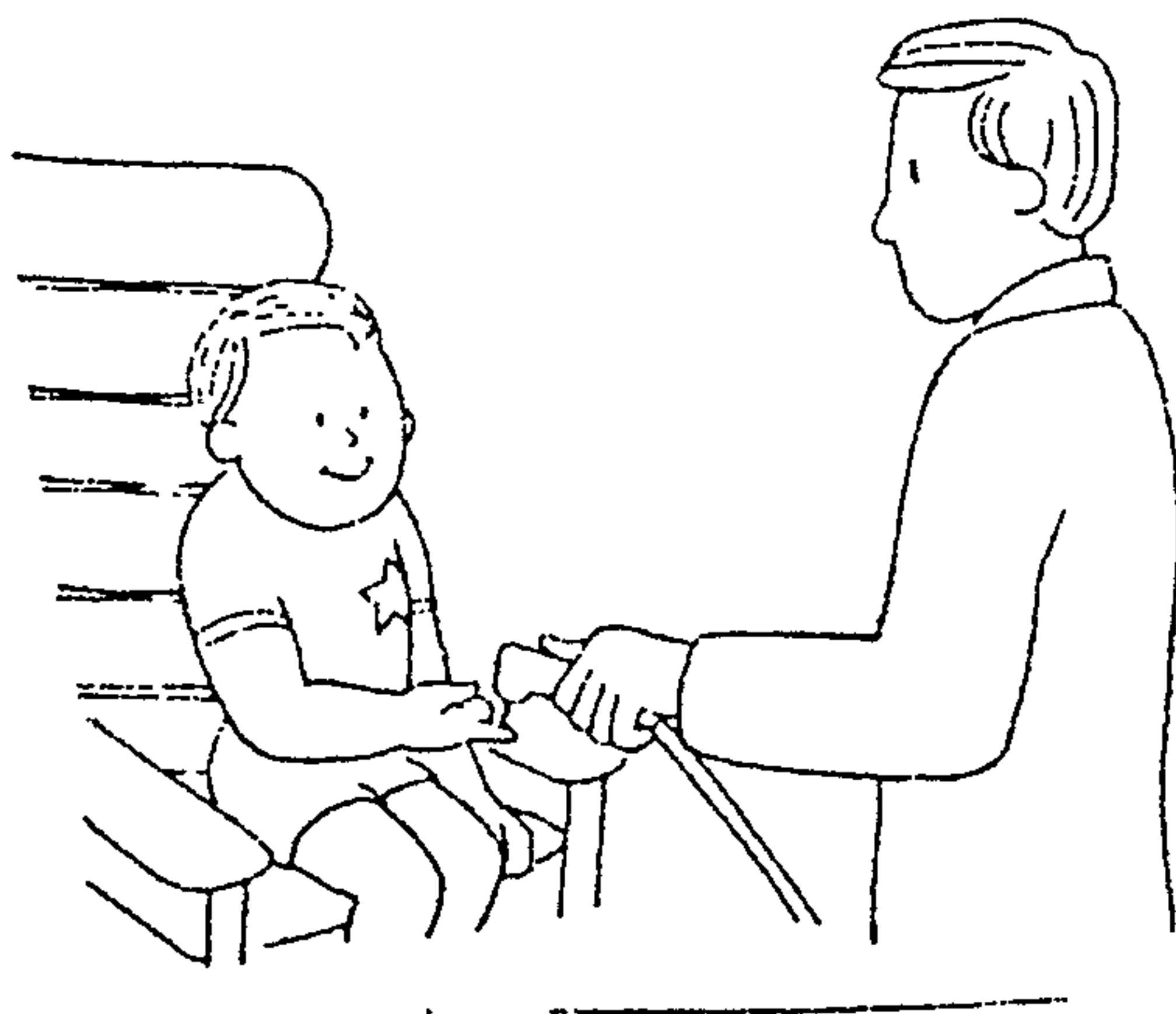
The following procedures pertain only to carious lesions that have been treated with silver fluoride at a previous appointment.

Cavity Preparation

Although glass ionomer cements (GIC) will bond to enamel and dentine, mechanical retention is essential for adequate longevity of GIC restoration in deciduous teeth.

Cavity preparation can be carried out atraumatically by using ROUND burs in an 8 to 1 reduction handpiece (e.g. W&H Series 808). The burs must be new and rotated at no more than 60-100 r.p.m. in a forward direction. Reduction handpieces have excellent torque and will not stall at very low speeds. Firm pressure can be applied without discomfort to the patient.

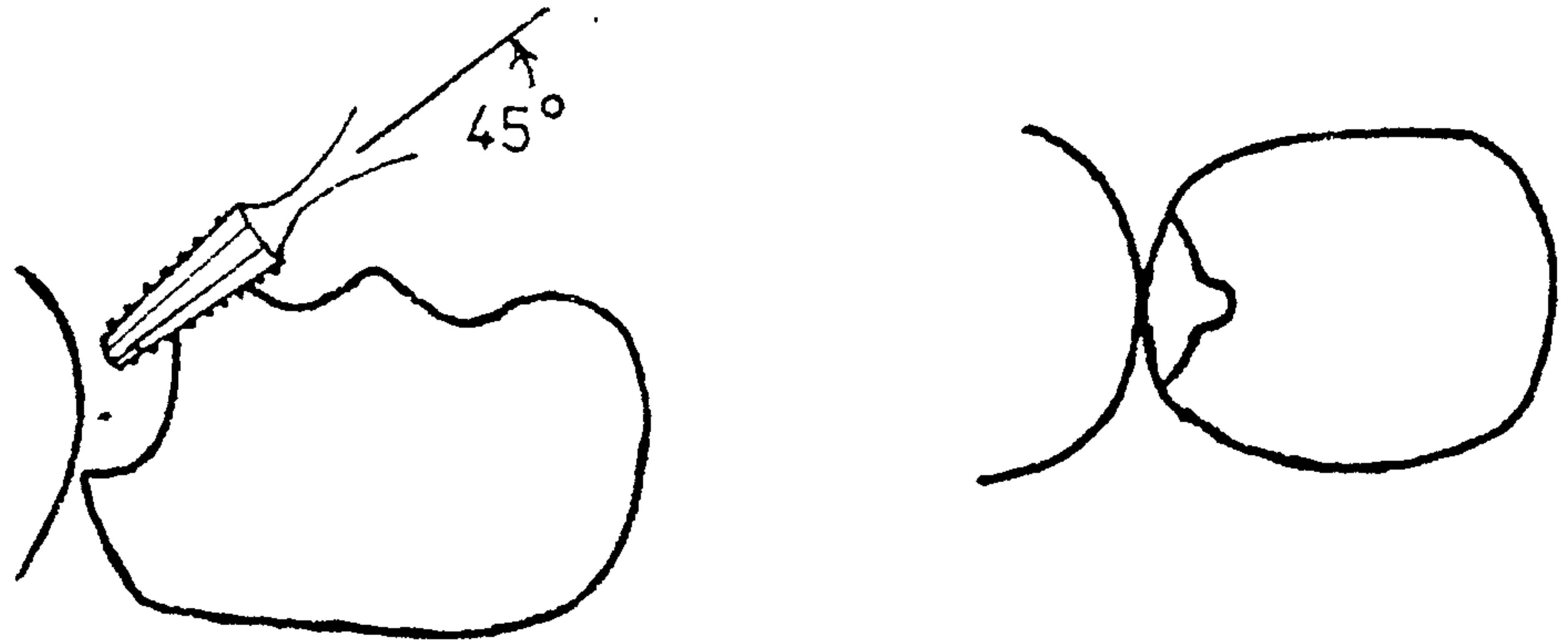
Before cavity preparation run the bur on one's own finger then the patient's finger to acquaint him with the instrument. Whilst this may seem an unnecessary step to some, failure to do so can introduce unwanted apprehension into the visit.



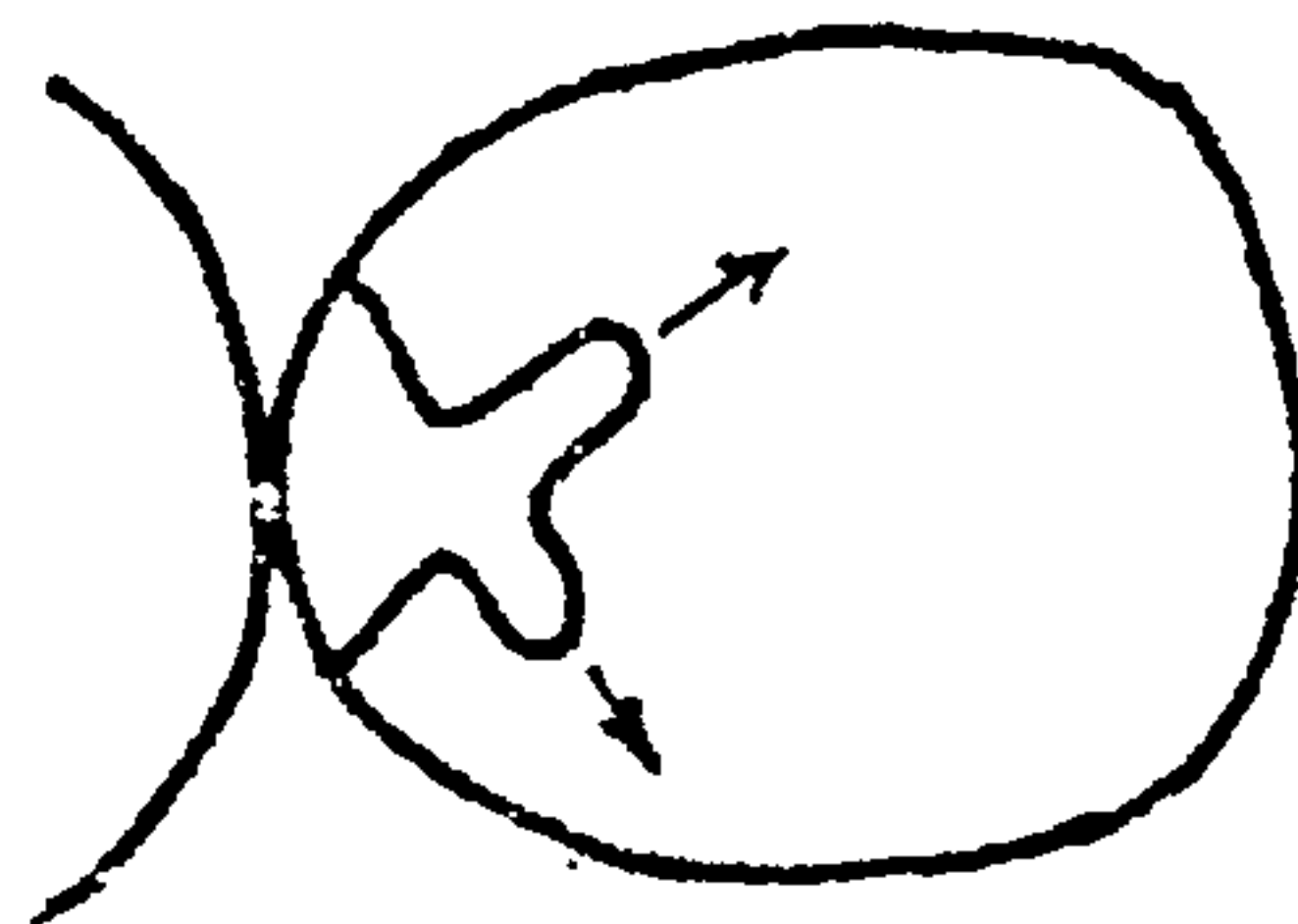
Preparation of Distal Cavities in Upper and Lower Deciduous First Molar Teeth

a. Occlusal Lock

Use a small tapering fissure bur (Ash No. 700) angled at 45° to the occlusal plane to cut a starting point in the centre of the occlusal cavo-surface margin of the cavity.



From this starting point establish an occlusal lock with a No. 2 round bur. The lock should be just into dentine.



b. "Proximal Box"

With a No. 1 round bur establish retentive grooves in the "proximal box" in the buccal and lingual walls just inside the dentino-enamel junction. It is essential that the glass ionomer cement abut onto sound enamel and dentine on all cavity walls.

Preparation of Proximal Cavities in Deciduous Second Molar Teeth*

Usually no occlusal lock is required in the preparation of proximal cavities in deciduous second molar teeth. A No. 1 or No. 2 round bur is used to prepare retention just inside the dentino-enamel junction.

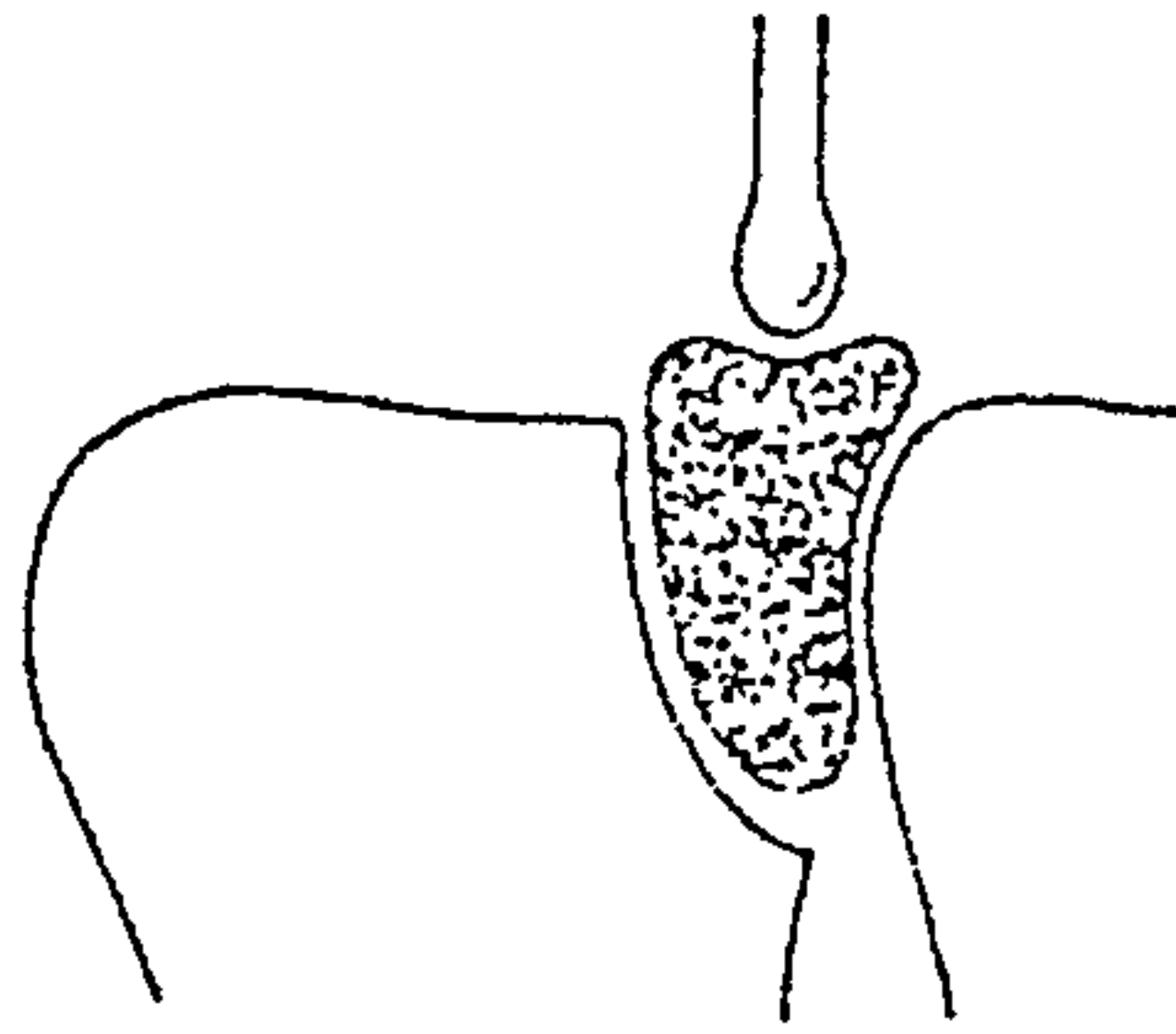
Chemical Treatment of Cavity Preparations

Before the placement of glass ionomer cement apply silver fluoride on a pellet of cotton wool to the floor and walls of the cavity. Mop up any excess with another pellet of cotton wool before drying the cavity with an air syringe. A fine coating of dried silver fluoride (which is yellow in colour) should then be apparent.

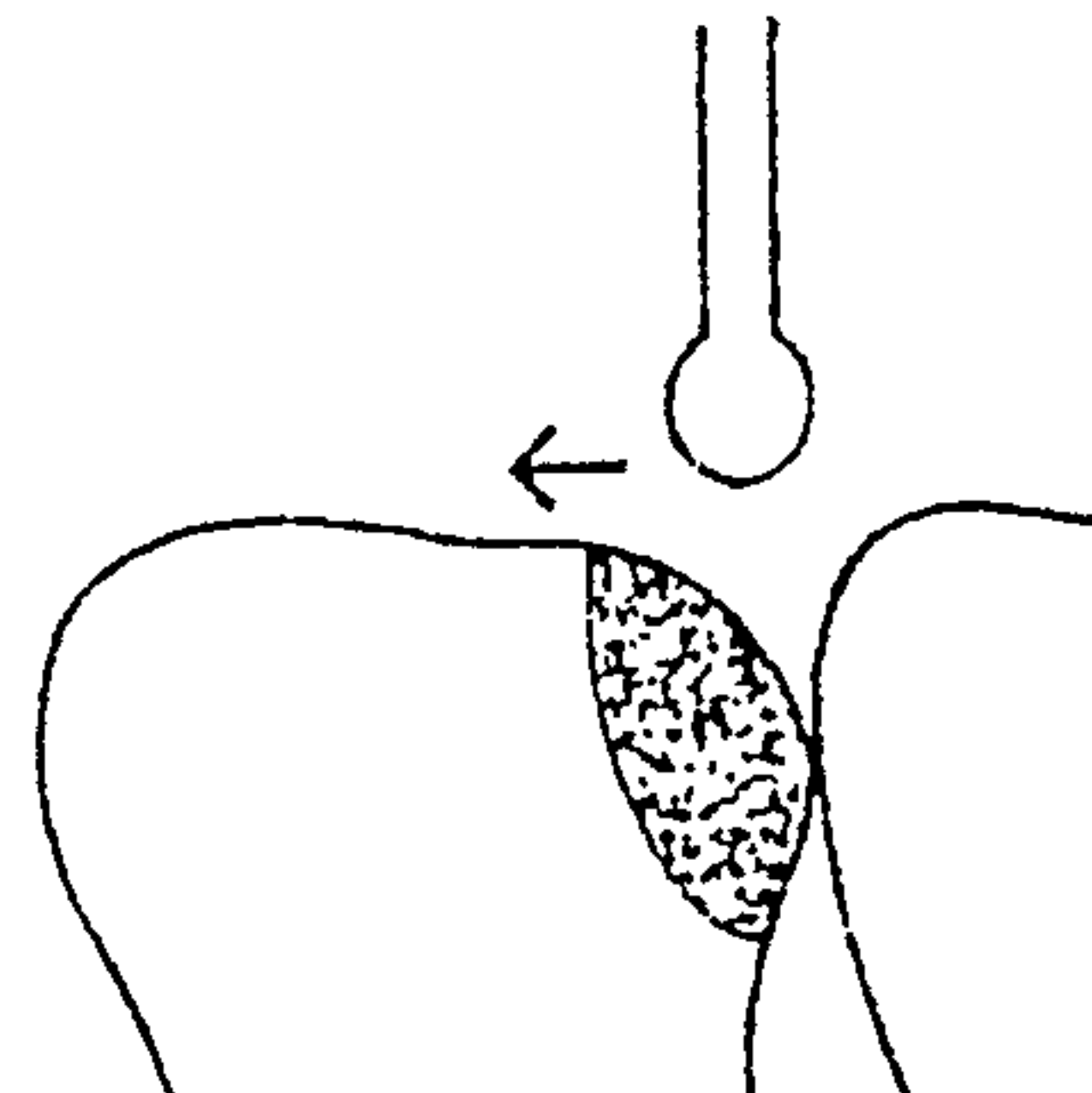
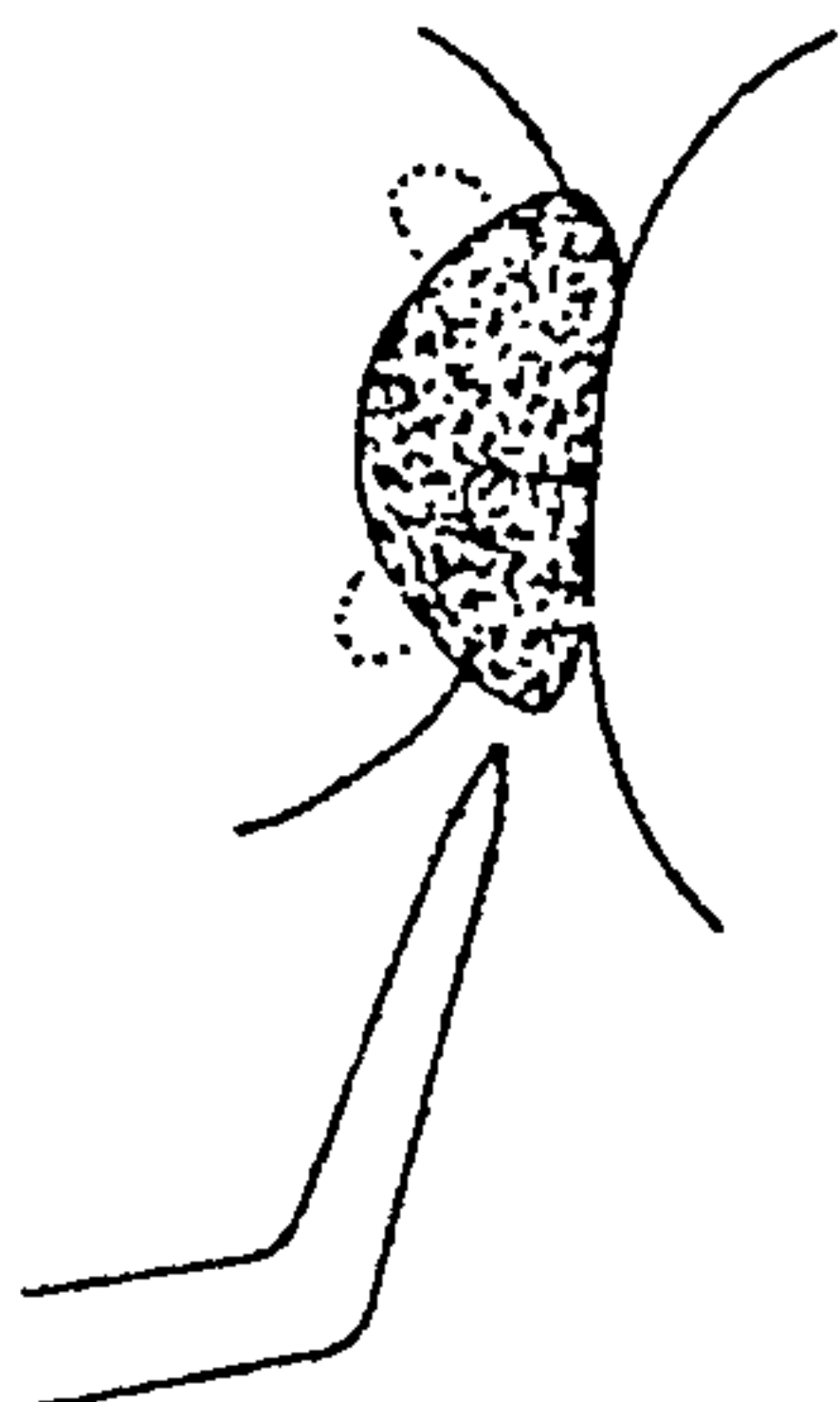
The application of silver fluoride will also assist in arresting gingival bleeding and facilitate the attainment of adequate dryness of the cavity.

Restoration Placement

1. Isolate the tooth to be restored with cotton rolls and dry cavity carefully. (Glass ionomer cement restorations are very sensitive to moisture for the first 10-20 minutes after placement). Mix glass ionomer cement to a uniform thick consistency and pack into prepared cavity with a round ended instrument

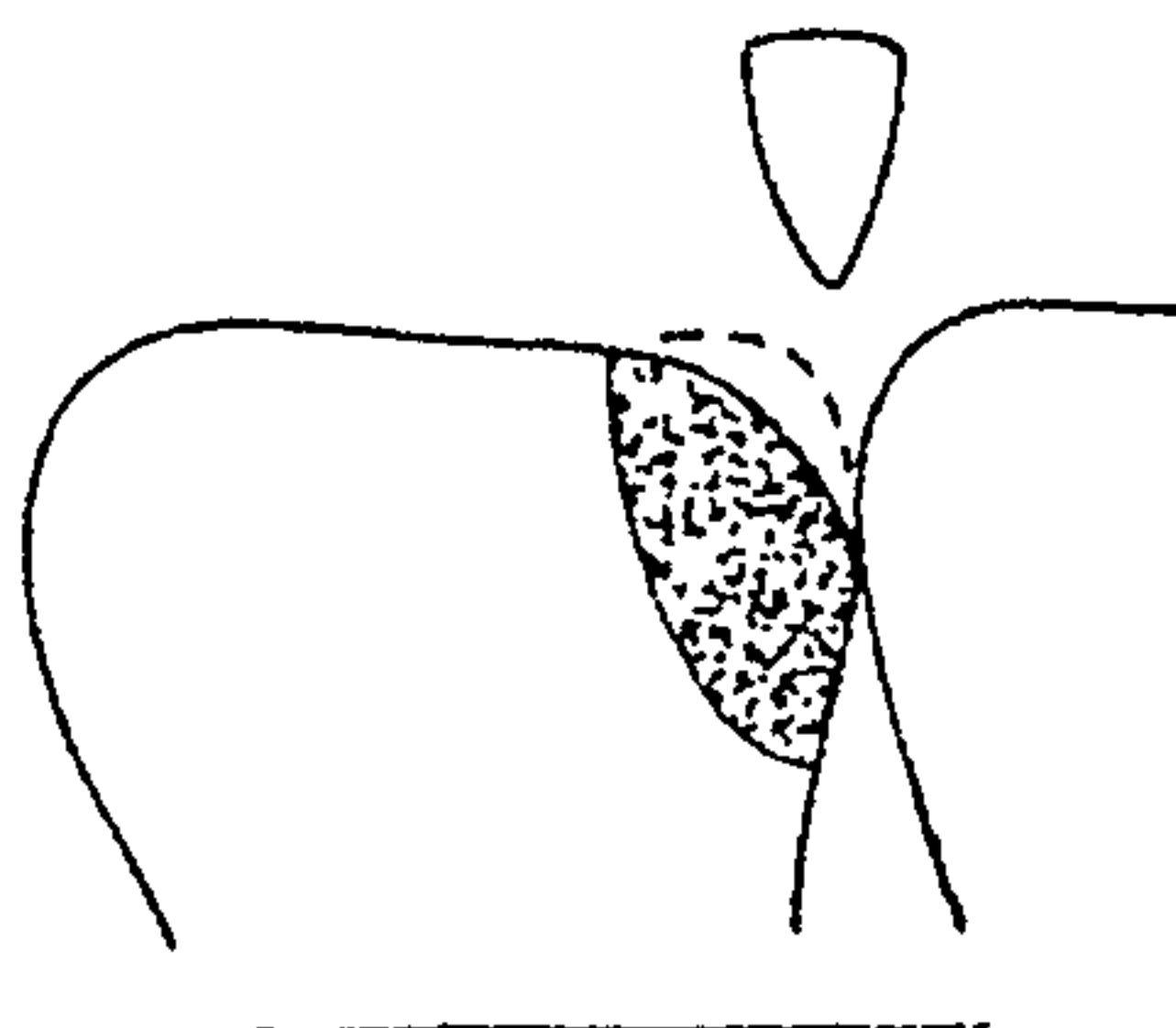


2. Trim material whilst it is still plastic. Use a round ended instrument to help shape the occlusal surface. A $\frac{1}{2}$ Hollenbach Carver is used to shape the approximal surface and form the final contour of the occlusal surface. All instruments should be used with a "stroking" motion working from the restoration towards the cavity walls.

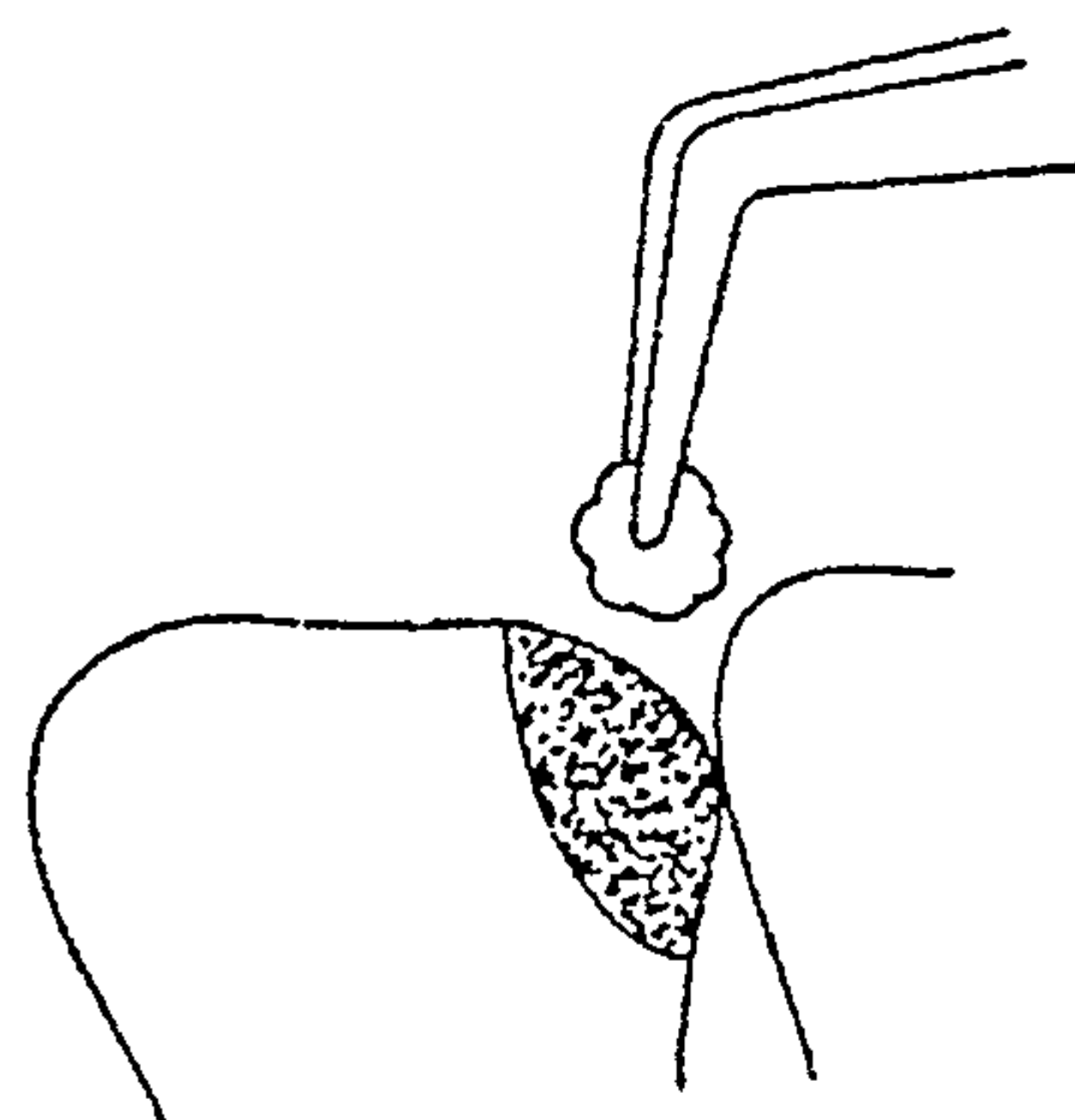


*Plus mesial surfaces of deciduous first molar teeth

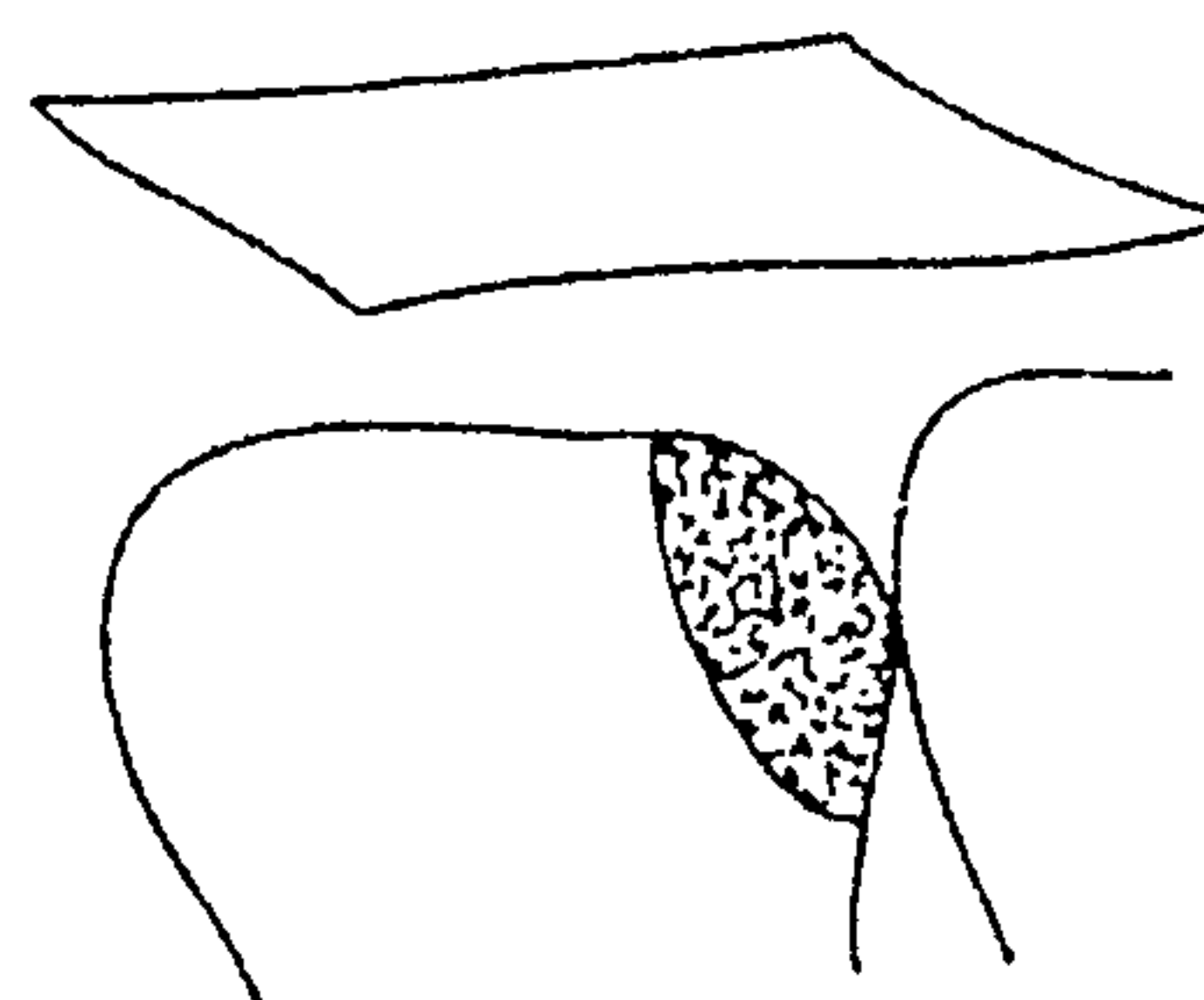
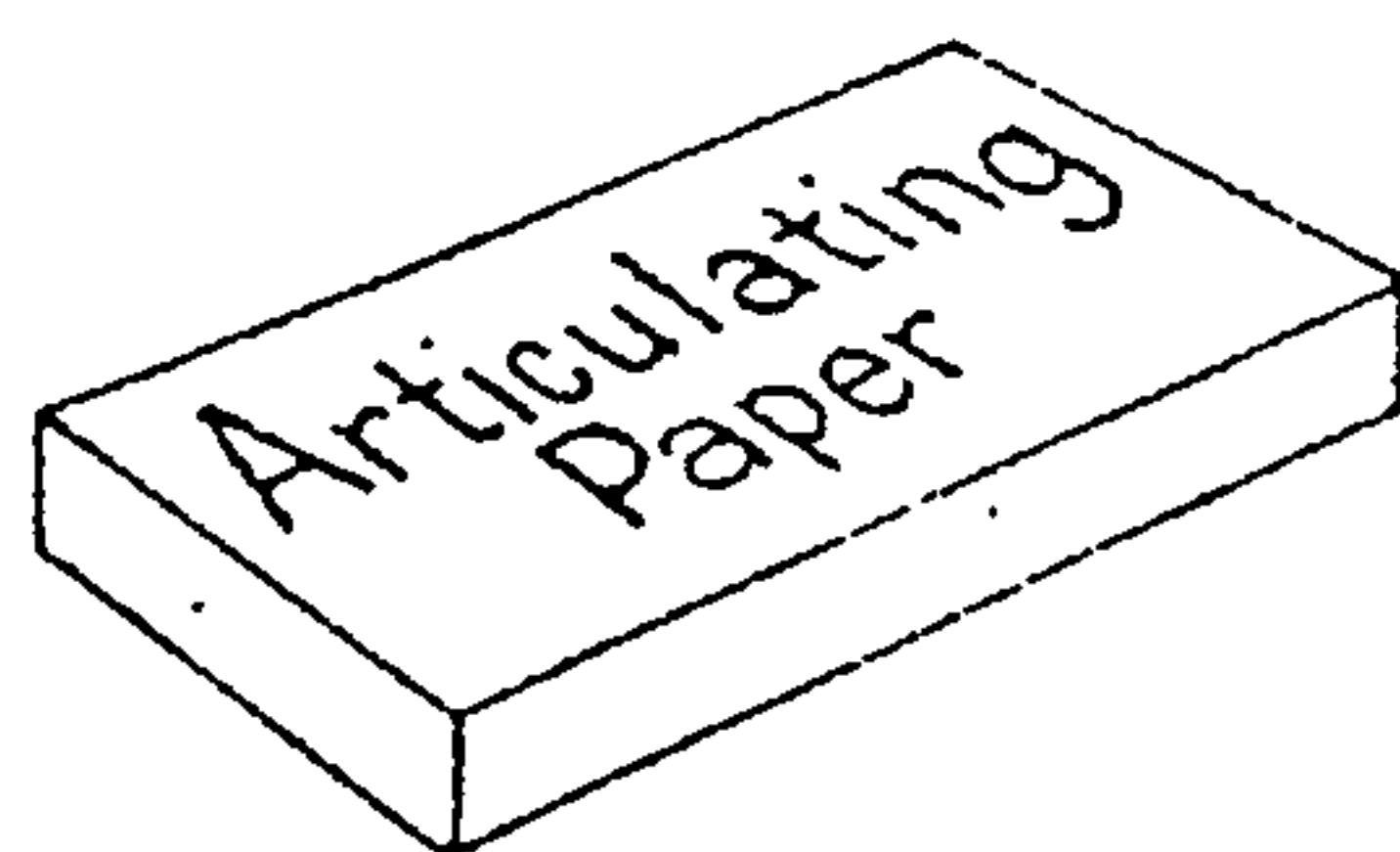
3. Because glass ionomer cement restorations have poor edge strength it has been found necessary to round the marginal ridge area. A 00 Morse Scaler is particularly useful for establishing the correct contour.



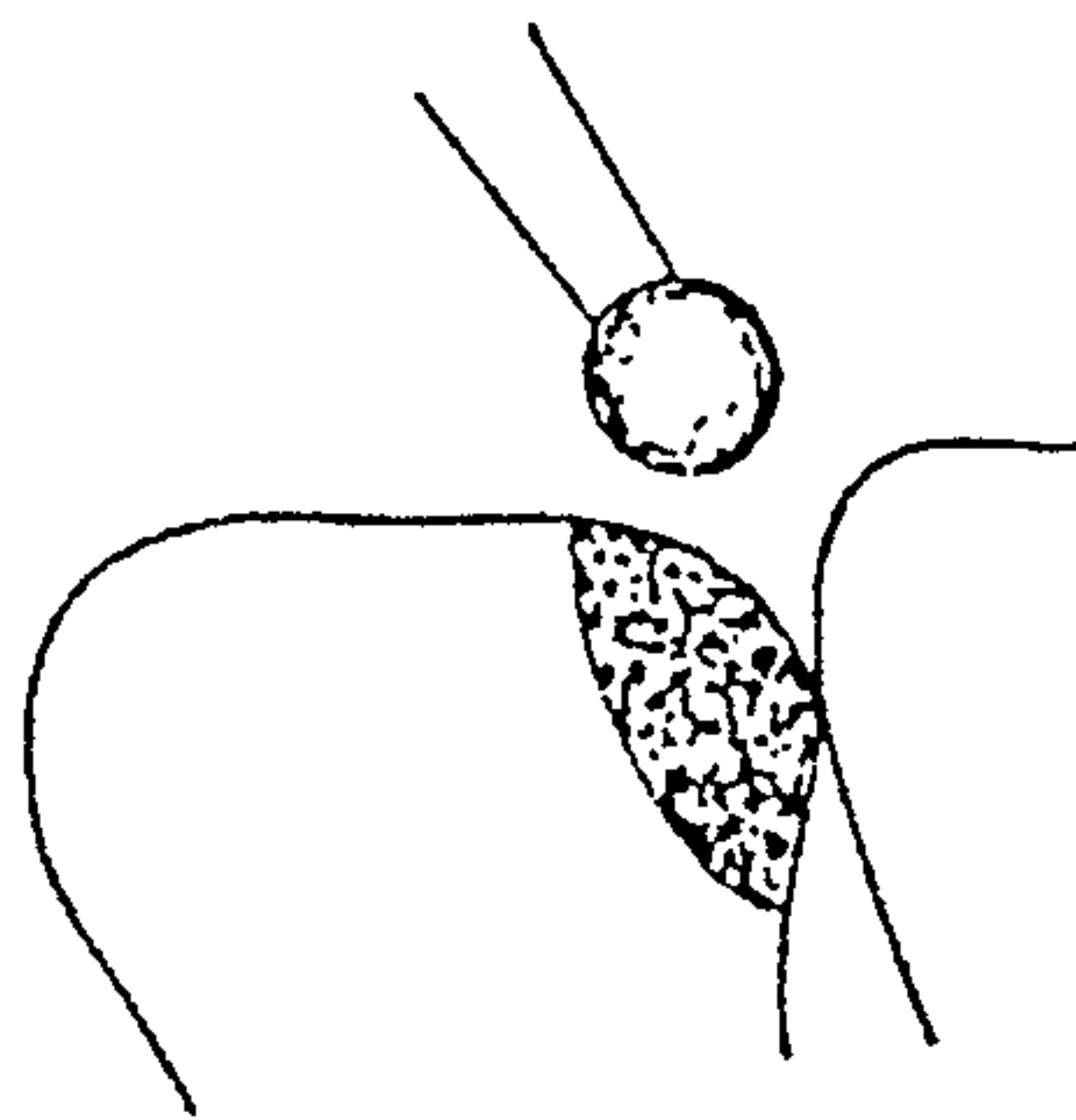
4. Before removing the cotton rolls cover the restoration with Fuji Varnish.



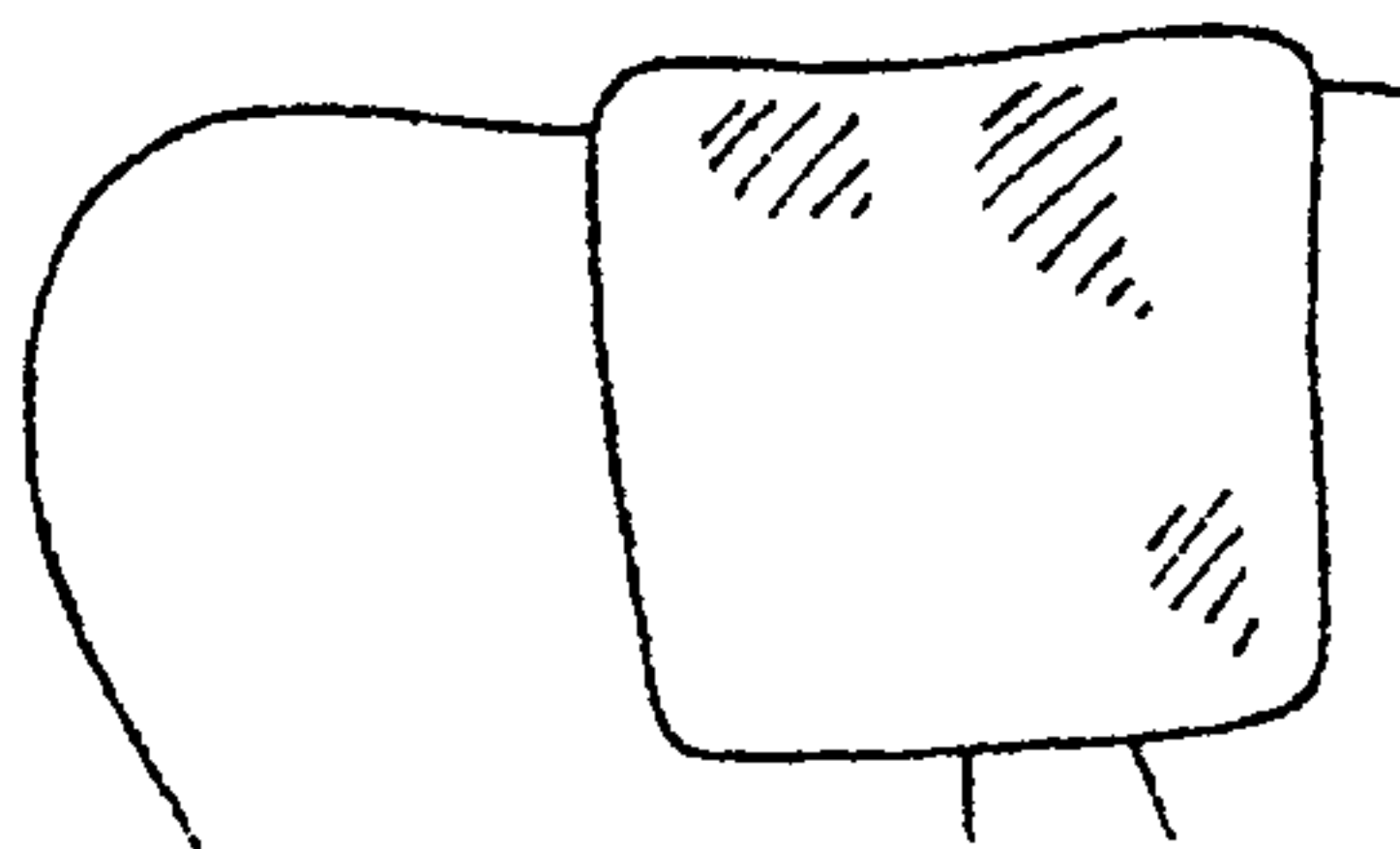
5. Check occlusion with articulating paper.



6. Remove any high spots with a round bur or stone then re-apply varnish.



7. Cover restoration with a small piece of Stomahesive Wafer.



COST OF MATERIALS (MAY 1986)

PRODUCT AND MANUFACTURER	OBTAINABLE FROM:	
GLASS IONOMER CEMENT	MARTIN HALAS	\$16.30 KIT
KRI 1 PASTE	MARTIN HALAS	\$10.85 EACH
LEDERMIX PASTE	DENTSPLY	\$30.83 EACH
CAVITEC CAVITY LINER	RUDOLF GUNZ	\$21.15 EACH
IRM = INTERMEDIATE RESTORATIVE MATERIAL	DENTSPLY AUST. Govt Contract	\$7.40 EACH
STOMAHESIVE OR VARIHESIVE WAFERS	CLIFFORD HALLAM	\$13.00 BOX
FLORAN SPOT APPLICATION PASTE (10% SnF ₂)	McGLOIN Govt Contract	\$3.50
AGF BRAND SILVER FLUORIDE (3 ml bottles)	GOVT COST	\$37.62
FLORAN STABLE STANNOUS FLUORIDE SOLUTION (20% SnF ₂)	McGLOIN Govt Contract	\$5.30
JUNIOR GARMERS CLAMPS	MARTIN HALAS Govt Contract	\$12.49
ROOT FILLER SPIRALS	MARTIN HALAS Govt Contract	\$3.10 PKT

