FLUORIDATION
Errors and Omissions in Experimental Trials

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MELBOURNE UNIVERSITY PRESS
First published in 1959
Second edition, enlarged, 1960
Printed and bound in Australia by
Melbourne University Press, Parkville N.2, Victoria
Registered in Australia for transmission
by post as a book

London and New York: Cambridge University Press
PREFACE TO THE SECOND EDITION

Soon after the publication of the first edition of this monograph, in September 1959; copies were sent, by the Australian Dental Association, to the workers in charge of all the studies considered. As a result, critical reviews were published in the February 1960 issue of the Australian Dental Journal. The New Zealand Dental Journal of January 1960 also contained a critical review. These have not indicated the necessity for any modifications in Parts One and Two which are, therefore, reprinted unchanged. However, in this edition a Part Three has been added in which these criticisms are reprinted, at length, and some comments made. It is again stressed that in this book consideration is limited to some aspects of five crucial experimental trials of artificial fluoridation. Results reported from “naturally fluoridated” areas are not considered.

P.R.N.S.

Dental School,
University of Melbourne
June 1960

PREFACE TO THE FIRST EDITION

Endorsements of the process of the mechanical addition of fluorides to public water supplies, with the aim of reducing the incidence of dental caries, rely mainly on the results published from five trials which were set up to test, primarily, the efficacy of this process.

Important deficiencies in the methods used were revealed during a preliminary investigation of reports of these trials. Therefore this study was undertaken in an attempt to answer the question: Can the claims of considerable dental benefits as a result of artificial fluoridation be regarded as established, or are they based on an unsound foundation.

P.R.N.S.

Dental School,
University of Melbourne
February 1959
ORIGINAL ACKNOWLEDGMENTS

MEMBERS of the Department of Statistics, University of Melbourne, have given most careful consideration to all the statistical matters mentioned in Part One of this monograph; they have checked the computations in Part Two and have also given advice regarding statistical matters mentioned in Part Three. Their assistance is gratefully acknowledged. Part One was published in the Medical Journal of Australia, in February 1958, pages 139-140. I should like to express my thanks to my co-author, and to the Editor of the journal for permission to reprint the paper and also to the Editors of the Australian Dental Journal and the New Zealand Dental Journal for permission to reprint the book reviews shown in Part Three. Extracts from Part Two were presented at the fifteenth Australian Dental Association Congress, Adelaide, a3-7 February 1959. Professor Sir Arthur Armies and Dr Paul Pincus have suggested improvements to the draft of Part Two, and Miss H. N. Rankine, the Librarian of the Dental School of the University of Melbourne, has given valuable assistance. This investigation has been supported by grants from the University of Melbourne Research Fund.

LATER ACKNOWLEDGMENTS

The family of Dr Sutton wish to particularly thank Mr Glen S.R. Walker for the support and great friendship he gave to their father during his lifetime. They wish also to acknowledge the invaluable hours of learned advice and assistance he so willingly gave to them in preparing this [later] book for publication. [from collected works on P.R.N. Sutton]

ABOUT THE AUTHOR xiii
FOREWORD

By Professor Albert Schatz, Ph.D., Philadelphia, U.S.A.

(Professor Schatz discovered the antibiotic Streptomycin which was the first effective means of treating human tuberculosis. For this and other research, he received honorary degrees and medals, and was named an honorary member of scientific, dental and medical societies in Europe, Latin America and the United States. He was a recipient of France’s highest award for services to humanity.)

Here’s freedom to him who would read,
Here’s freedom to him who would write.
There’s none ever feared that the truth should be heard,
But they whom the truth would indict.

Robert Burns (1759-1796).

The importance of this book transcends fluoridation because it is concerned with science, values, ethics, integrity and professionalism. The book is also concerned with democracy; that is, with freedom of speech and a free press. The “fluorocracy”, on the other hand, has too often engaged in censorship; opponents of fluoridation have been denied opportunities to speak at meetings and publish in professional journals. In a democracy, every individual should have the opportunity to publish what he wants, provided that he writes with propriety, pays whatever publication costs may be involved and assumes responsibility for what he has printed.

The fluoridation controversy is symptomatic of a deep-seated pathology in present-day science. The magnitude of that malady; that is, misconduct in research, which the public is well aware of, motivated the U.S. Academy of Sciences to convene a Panel on Scientific Responsibility and the Conduct of Research. (Responsible Science Ensuring the Integrity of the Research Process. Vol. I, Washington D.C.1992). The Panel’s investigation, which cost $888,000, precluded consideration of certain kinds of scientific misconduct which specifically apply to fluoridation. Polluted science has occurred “when new scientific evidence threatens fluoride’s protected pollutant status. The government immediately appoints a commission, typically composed of several veteran fluoride defenders and no opponents. Usually, these
commissions dismiss the new evidence and reaffirm the status quo. When one didn’t in 1983, the government simply altered the findings.” (Griffiths, J., 1992, Covert Action No, 42, page 26.)

The controversy about fluoridation was inevitable because fluoridation was, in a real sense, conceived in sin. Fluoride is a major waste product of industry and one of the most devastating pollutants of the aluminium industry. The government has not only dismissed the danger and left industry free to pollute, but it has promoted the intentional addition of fluoride - most of which is recycled industrial waste - to the nation’s drinking water. Since 1950, when fluoridation was sanctioned, approximately 143,000 tons of fluoride are pumped into two-thirds of the reservoirs of the U.S. each year! (Griffiths, 1992).

One may also be interested in what I call “the pig mentality”. In 1952, a U.S. Congressional Investigation concerned a recommendation by the U. S. Department of Agriculture that farmers not add to the water or feed of pregnant pigs because the fluoride did something to the unborn pigs. When one of the investigating committee asked whether “it might be wise for the U.S. Public Health Service or some group of people to enquire what might happen to pregnant women and the unborn child when they are given fluoride”, the answer was, “There is more money available for matters that have economic value than there is for health.” (Schatz, A. 1976 Cancer News Journal Vol. II, No. 4.)

It is also important to understand how fluoridation was originally “sold” to the public. “The public relations strategist for the water fluoridation campaign was none other than Sigmund Freud’s nephew, Edward L. Bernays ... known as “the father of public relations “ Bernays pioneered the application of his uncle’s theories to advertising and government propaganda. The government’s fluoridation campaign was one of his most stunning and enduring successes.”...”Those who manipulate this unseen mechanism of society constitute the invisible government which is the true ruling power of our country - our minds are moulded, our tastes formed, our ideas suggested, largely by men we have never heard of.” (Griffiths, 1992).

Now let us return to democracy to which this book on fluoridation makes a major contribution. “Knowledge will forever govern ignorance and a people who mean to be their own governors must arm themselves with the power which knowledge gives.” (James Madison). This book gives us that kind of knowledge about fluoridation.
According to Sir Arthur Amies, “The passion to regulate the lives of others is deep-seated in many individuals. When this is based on political expediency, it is bad, and when it is inspired by an idealism which wishes to inflict benefits on others, it can be dangerous.” (Schatz, A. 1976. Increased Death Rates in Chile Associated with Artificial Fluoridation of Drinking Water, with Implications for Other Countries. Anthony University Jour. of Arts. Science and Humanities. 2: 1. Copies of this publication may be obtained from the Library of Congress, Washington, D.C.) U.S. Supreme Court Justice Louis Brandeis expressed a similar concern as follows: “Experience should teach us to be most on our guard to protect liberty when the ... purposes are beneficial.”

Philip R.N. Sutton’s book presents and interprets the proverbial handwriting on the wall for fluoridation.
ABOUT THE AUTHOR


Dr Sutton wrote his first article pointing out errors in fluoridation trials, in the Medical Journal of Australia, thirty-five years ago. He continued to study and write about fluoridation, published numerous articles and two previous books on this subject.

In 1935, on his twenty-first birthday, he graduated with honours from the University of Melbourne, having completed the five-year course of the Bachelor of Dental Science. He immediately undertook post-graduate study and research in Physiology and Biochemistry and established a private practice in Brighton, Victoria which he conducted for twenty-five years.

On the outbreak of war in 1939 he enlisted in the Australian Army, serving in the Dental Corps for a total of five years. In North Borneo he was a member of an Australian Army medical team which saved British and Australian servicemen who had just been released from a small prisoner-of-war camp where they had been dying from starvation at the rate of six a day. Observations he made at that time formed the basis of a thesis submitted to the University of Melbourne which gained him the degree of Doctor of Dental Science - the highest dental research degree.

After the war he persuaded the Professor of Statistics at the University of Melbourne to establish a course, now called Statistics for Research Workers, and, having completed the course, joined the Statistical Society and was later elected chairman of the Biometric Society.

He was invited to become one of the Foundation Fellows to form the Royal Australasian College of Dental Surgeons. Dr Sutton was elected to the Council of the Victorian Branch of the Australian Dental Association (which appointed him as its representative on the Preventive Dentistry Committee which employed a public relations consultant to promote fluoridation, which had just been introduced into Australia. Therefore, because of that association, at that time he could have been said to be a promoter of fluoridation.

In 1956 he was appointed a Senior Research Fellow of the University of Melbourne and took his family for a year to Raratonga island, South Pacific, where he provided free dental treatment and studied tooth abnormalities in Polynesians which resulted from their diet and habits.
On his return to Melbourne in 1957, Professor Sir Arthur Amies, Dean of the Faculty of Dental Science, asked him to check the numerical data published from the original fluoridation trials and the scientific methods used in them. He discovered so many errors that to record them he was forced to write a 72-page monograph Fluoridation: Errors and Omissions in Experimental Trials (Melbourne University Press, 1959). He published a second 142-page edition in 1960 which answered the criticisms of the first edition, showing that they were false. This book remains scientifically unchallenged.

In 1964 Sir Arthur Amies invited him to become the first Senior Lecturer in Dental Science, a position from which he resigned eleven years later to have more time to continue his Pacific islands studies of Polynesians and Micronesians.

During a year’s leave, in 1970-1971, he worked in London at the Maudsley Hospital for psychiatric patients, with the cooperation of Dr Denis Leigh the Secretary General of the World Psychiatric Association and Editor of the Journal of Psychosomatic Research. The aim was to extend his knowledge of the relation between mental stress and acute dental caries (which he had published in Nature in 1962; N.Y. State Dental Journal, 1965; Advances in Oral Biology, Vol. 2, 1966, Academic Press).

He published a second book Fluoridation, 1979: Scientific Criticisms and Fluoride Dangers as a 285-page submission to the Victorian Government-sponsored Committee of Inquiry into the Fluoridation of Victorian Water Supplies. This led to him being flown to Edinburgh to give evidence for several days before the inquiry into fluoridation in the High Court.

Dr Sutton had wide-ranging research interests and publications apart from fluoridation, such as his series of papers on the relation between mental stress and dental decay, the initial article being his first publication in Nature. He became a regular contributor to the “ideas” scientific journal Medical Hypotheses, which has published all the eleven papers he has submitted.

Philip Sutton was internationally respected as a dentist and medical researcher and a great gentleman.

Almost without exception, Philip Sutton is mentioned in the references of world publications on fluoridation.

Philip Sutton was noted as a strong fluoridation critic, but never once
stooped to personalities because of his strong conviction that honest science is where debate should be confined.

Philip, as he was affectionately known around Australia, was always available for discussions and advice on fluoridation and practical help in dentistry.

Philip Sutton published the first warning about fluoridation deceptive claims made about the first experimental fluoridation plants. His book Fluoridation: Errors and Omissions in Experimental Trials, published 1959 is only now acknowledged by the fluoridation hierarchy as correct, even though throughout the years since he published his research, the Health Departments of the U.S.A. and government employed dentists throughout the world aggressively attacked his printed data.

It is now documented in the Australian Government National Health and Medical Research Council 1991 Study into Fluoridation that:

“...The quality of the early intervention trials was generally poor.”

So it took over 30 years before Philip Sutton’s research data was acknowledged as correct by the Australian Government and other international organisations.

It would be difficult to find a more academically qualified and practical dental doctor with qualifications that set him above the so-called “experts” foolish enough to criticise his work.

Philip Sutton gave evidence at fluoridation enquiries around Australia, he also attended public meetings, often speaking on fluoridation. He always answered the questions that usually came fast and furiously.

The world has lost a great scientist, but he left a standard of quality research in his publications (including articles published in most countries of the world), and in his books, suggesting a standard that should continue to form the basis of proper debate on fluoridation.

To the end of his life Dr Sutton was a seeker of truth. Unfortunately he did not live to see this his final work published as he died on 12th March 1995.
CONTENTS

PREAMBLE: Material added after the second edition from a later book
LATER ACKNOWLEDGEMENTS viii
FOREWORD x

xvi
INTRODUCTORY COMMENTS

Before the first edition of that monograph ["Fluoridation: Errors and Omissions in Experimental Trials"] was published in 1959, as a matter of courtesy a copy of the final draft was sent, for his information, to the Federal President of the Australian Dental Association, Dr (later Sir Kenneth) Adamson - who was well known to me.

At Dr Adamson’s request, the monograph was discussed with him for several hours in the presence of a friend of his, a consultant physician with extensive knowledge of academic statistics, who, to Dr Adamson’s obvious surprise, did not make any criticism. This was not unexpected, for during its preparation it had been most carefully checked by Professor Maurice Betz, the head of the Department of Mathematical Statistics in the University of Melbourne.

Of course, the results published in the monograph threw considerable doubt on the pro-fluoridation stance which had been adopted by the executive of the Australian Dental Association. Therefore, having failed to find any errors in the monograph himself, Dr Adamson sought criticisms from others by sending copies of this final draft to a number of fluoridation “experts”, including the authors of the five studies which were discussed in the monograph. Some of their replies to Dr Adamson were later published in the February, 1960, issue of the Australian Dental Journal as “Book Reviews”. However, none of the published Criticisms were written by authors of the Grand Rapids and Newburgh studies. Upon inquiry, the Australian Dental Journal said that replies had been received from authors of those two studies, but that the language of their replies, particularly that of Dr David Ast of the Newburgh trial, was so immoderate that it had been considered unwise to publish their comments.

These criticisms of the monograph were backed up by an editorial in that issue which mentioned Part One of the first edition, which had been reprinted from an article by the present author, with Professor Sir Arthur Amies, in the Medical Journal of Australia, I February, 1958(a). That editorial in the Australian Dental Journal (1960) stated:

“It is important, however, not to be stampeded by this criticism [in the monograph] since to be of value it must have the hall-mark of informed authority.”

That dental editor was so biased towards fluoridation that he was prepared
to brush aside the fact that Part One of the monograph, that article in the Medical Journal of Australia, had as co-author the “informed authority”, Professor Sir Arthur Amies, Dean of the Faculty of Dental Science, University of Melbourne, who had closely studied fluoridation since its inception, and who had carefully considered the material in Part Two before it was published.

The editor of the Australian Dental Journal contended that this article (Sutton and Amies, 1958a) contained a “fundamental error”, in that it stated that proposals to fluoridate domestic water supplies are almost entirely based on the results of the Brantford, Grand Rapids, Newburgh and Evanston projects. He claimed that the scientific basis of fluoridation was established firmly before those trials. However, he could not have read the reports of those four trials in which all the authors stated that their trials were set up to test the fluoridation hypothesis. For instance, the authors of the Grand Rapids study (Dean et al. 1950) stated:

“... in 1945, three studies to determine the caries prophylactic value of artificially fluoridated drinking water were started in the United States and Canada.”

There would have been little point in establishing these long-term trials (planned to last for ten years) if the editor of the Australian Dental Journal had been correct, and the scientific basis of fluoridation had been established firmly prior to these trials.

Sadly, as the evidence against fluoridation has mounted over the years, the executive officers of the Australian Dental Association, instead of reassessing their stance, have become more and more dogmatic in their statements regarding this process. This attitude has jeopardized the status of dentistry as a scientific discipline which maintains an open mind, so that opinions can be modified as new scientific facts emerge which show that the views held are no longer tenable.

It should be known that there is no evidence that the great mass of dentists in private practice have studied fluoridation data. As in the case of most scientific matters, which they have neither the time nor the specialized training to investigate, their opinions are based on those expressed by the executive officers of the Association who, they assume, provide them with a well informed and honest appraisal of scientific subjects. Unfortunately that assumption, in the case of fluoridation, is not justified.
The same situation occurs in other countries. The President of the International Society for Research on Nutrition and Vital Substances, Professor H.A. Schweigart, pointed out in 1967 that the German organization of dentists had requested the fluoridation of drinking-water on behalf of its 35,000 members, but that most of the members were not consulted. He stated:

“The fluoridation of drinking-water releases a fluorine circuit which includes vegetables, fruit and other horticultural products as well as milk, and has an uncontrollable effect on the human organism.”

At least some executive officers of the A.D.A. have promoted fluoridation for many years, saying that it is efficacious and absolutely safe. It seems they are now so afraid of losing “face” that they are prepared to make false statements and to mislead even their own members about this medication. Such an incident occurred in an anonymous newsletter distributed to all the members of the Australian Dental Association in 1989. This bulletin was entitled “Disaster in Canberra”. No mention was made of the dental effects of this “disaster”—the cessation of fluoridation in Canberra by order of the A.C.T. Legislative Assembly. The “disaster” seems to be the fact that that decision was contrary to the policy of the executive officers, and to the advice they had given during “... a vigorous lobbying campaign to inform members of the Assembly of the Association’s views on fluoridation” and, therefore, was damaging to their prestige and image. The newsletter said that another study which purported to reach the same conclusion as Dr Diesendorf’s [which the newsletter criticized] was by Colquhoun in New Zealand. The newsletter stated:

“When the data was re-examined for previous fluoride exposure by the N.Z. Medical Research Council Colquhoun’s “findings” evaporated.”

This statement in the ADA News Bulletin is false. The Director of the Medical Research Council of New Zealand stated in a letter, dated 8 January, 1990, to Dr John Colquhoun, that:

“... this Council has not at any stage set out to re-analyse your research data, nor has it contracted others to do so.”

In reply to a request by the present author for a copy of their “analysis” cited by the Executive of the A.D.A., the Administrative Officer of the New Zealand Medical Research Council, in a letter dated 7 February, 1990, stated (in part):

xix
“Neither this Council nor any of its research Units or investigators have produced a paper on this work [by Dr Colquhoun] nor am I aware of the possible source of this information.”

These two letters show that the statement by the Executive of the A.D.A. is not true.

A similar statement to that in the ADA News Bulletin was incorporated into a long (10-page) misleading letter to members of the ACT Legislative Assembly and was a factor in tricking some of them into reversing their vote and restoring fluoridation to Canberra, without waiting for the finding of a five-member Parliamentary Committee which the Assembly had set up to investigate this matter. The report of this committee was published in February 1991 (See Appendix II).

The concept of fluoridation arose from the results reported from “naturally fluoridated” areas of the U.S.A., during investigations into the cause of the unsightly condition then called “mottling” of the teeth (“dental fluorosis”). The main investigator was Dr Trendley Dean (1934), who became known as “the Father of Fluoridation”.

In 1983, Dr Rudolph Ziegelbecker, of the Institute of Environmental Research, Graz, Austria, commented on these studies. One of them showed that with a fluoride concentration of 0.5 ppm in Wisconsin the DMF rate per 100 children aged 12-14 years, was 710 - twice that of the DMF rate (342) in children of the same age in Colorado, where the fluoride concentration in the drinking-water was also 0.5 ppm. He found that:

“The calculation shows that in Wisconsin, fluoride in the range from 0.12 to 0.5 ppm was not correlated with caries incidence”, and he stated that “This study by Dean, used by the respondents [in a High Court case in Edinburgh] to support the hypothesis that fluoride reduces the caries incidence, is clearly unsound in its premises and conclusions and gives no one evidence that fluoride reduces caries incidence.”

Ziegelbecker also considered the famous diagram showing the dental caries / fluoride relationship in 21 cities in the U.S.A. This was prepared by Dean, Arnold and Elvove in 1942 and was published in many text-books, having a marked influence in promoting the idea that the prevalence of dental caries was inversely related to the fluoride content of drinking-water.

Ziegelbecker (1983) stated that this chart of the “inverse relationship” between fluoride ingestion and dental caries prevalence was based on:

xx
“... an inexcusable illicit selection of data”
because dental surveys from more than 650 counties and cities were known to Dean, but that he had:

“...selected 21 cities in such a manner that the result supported the thesis of the “inverse relationship” between the natural fluoride content of the common water supply and the caries incidence in children.”

(More than forty years after its publication this false diagram was still used, being tendered in evidence in 1981 by the pro-fluoridation respondents in that High Court case in Edinburgh, who stated that it was a careful and important study.)

Contrary to the contention of the editor of the Australian Dental Journal, there is no doubt that the early results reported from the Grand Rapids trial brought about the endorsement of fluoridation by the U.S. Public Health Service in 1950 (Lohr and Love, 1954), and undoubtedly formed the basis of later proposals to fluoridate drinking-water.

The editor of the Australian Dental Journal did not make further comments on that paper (Sutton and Amies, 1958a) after Associate-Professor Noel Martin, the main advocate of fluoridation in Australia, had failed, in two long letters to the Medical Journal of Australia on 22 February and 14 June 1958 (Martin, 1958a, 1958b) to point out any errors in the paper.

In a reply to Martin’s letters it was noted (Sutton and Amies, 1958b) that:

“Despite the fact that the length of his [Martin’s] criticisms considerably exceeded that of the paper, he did not indicate even one error in the statements made in demonstrating that there are disturbing features in the published reports of fluoridation trials.”

The same Associate-Professor Martin was appointed on 12 November, 1959 by the Dental Advisory Committee of the National Health and Medical Research Council of Australia to be the chairman of a committee of three—the other two members were professors of statistics—to investigate the contents of the monograph Fluoridation Errors and Omissions in Experimental Trials. Apparently, even with their expert assistance, he was not able to criticize the book and hoped that the matter would be forgotten, for more than three years later his report had not been submitted. However, this was noticed, and he was then instructed (25 March, 1963) to present it at the next meeting of that Committee of the NH&MRC. He did so, but his
report was not released.

After the passing of the Freedom of Information Act, under the provisions of that Act, the chairman of the Anti-Fluoridation Association of Victoria sought for two years to see that report by Associate-Professor Martin. When the report was not forthcoming the matter was taken to the Administrative Appeals Tribunal (in effect, a court) in July 1985, the respondent being the Secretary of the Commonwealth Health Department. After a lengthy case in the Tribunal it was announced that although the other records of the NH&MRC were available, Associate-Professor Martin’s report could not be found, and that no further search would be undertaken by the government to locate this official report by Martin and his committee.

(There is no doubt that this report existed at one time for Sir Arthur Amies, who was then a member of the Dental Advisory Committee of the NH&MRC, told the present author that he had read it, but that it was merely fluoridation propaganda and had not provided any valid criticism of the monograph. Presumably it had been removed from the NH&MRC files and destroyed.)

Soon after the first edition of the monograph was published, the stored printer’s type at the Melbourne University Press (which was usually held for at least six months) was melted down without authority by an unknown person, thus almost preventing the publication of a second edition. However, the type was re-set, at considerable expense...

There are accounts of similar attempts being made in other countries to prevent the publication of books which criticize fluoridation. One well-known case was the book The Toxicology of Fluoride, edited by Professor T. Gordonoff. According to Professor Albert Schatz (1965), one publishing house set the type:

“But it was then warned that if it went ahead and published this particular book the dental community would stop patronizing it. In the face of this threatened economic boycott and enticed by an offer of compensation to cover all expenses incurred (approximately 10,000 Swiss francs), the publisher “dropped” the book.”

It was published two years later by Schwab & Co.

As Schatz said in 1965:

“There are powerful forces which now have a vested interest in perpetuating fluoridation because their reputations depend on its
Suppression of discussion regarding fluoridation

The same vested interests are promoting fluoridation today, in the 1990’s using similar techniques to prevent the spread of the knowledge that fluoridation has failed: The repression and abuse of opponents of this process and the suppression of published evidence against it, and making it difficult to publish new material which those interests consider even questions fluoridation.

This discouragement of the discussion on fluoridation is still pursued very actively. The U.S. Public Health Service (U.S.PH.S.) - now the Department of Health and Welfare - distributes enormous funds to its many agencies. It also finances many research grants, both in the U.S.A. and in other countries. This control of grants has a restricting effect on the scientific discussion of fluoridation, for since 1950 it has been a process strongly promoted by the U.S.PH.S. American professors have admitted that they have to think of their grants and, therefore, avoid the subject of fluoridation. This is understandable for, apart from the financial aspects, if they questioned fluoridation there would be a distinct possibility that they would be added to those who are abused and whose personal reputations are attacked.

The refusal to consider any material which questions fluoridation is well illustrated by the experience of Professor Albert Schatz. In 1976 he published reproductions of photostat copies of three envelopes he had used in 1965, each containing the same short article about increased death rates associated with fluoridation in Chile. He had written previously to L.C. Henderson, the editor of the Journal of the American Dental Association about this paper but had not received a reply. The photographs show that the editor had refused to accept each of the three envelopes, in succession, and that they had therefore been returned, unopened, to Professor Schatz (his name was on the outside of each envelope).

In 1961, the American Dental Association’s Bureau of Public Information, in a re-issue of a publication entitled Comments on the Opponents of Fluoridation, grouped several reputable scientists with alleged members of the John Birch Society, the Ku Klux Klan, an escapee from a hospital for mental patients, and others, in an obvious attempt to injure their reputations by “damning by association.” That dossier condemned the 300 members of
the Medical-Dental Committee on the Evaluation of Fluoridation, solely because they were such a small proportion of the 300,000 physicians and dentists in the U.S.A.

Mr Ralph Nader, the consumer advocate, said in 1971:

“... you just don’t expect to be treated well by H.E.W. [a branch of the U.S.P.H.S.] in its massive research granting if you come out against this kind of thing [fluoridation]. It’s a matter of professional intimidation here.”

In 1988 Bette Hileman, an associate editor of Chemical & Engineering News, stated that John S. Small, information specialist at the U.S. National Institute of Dental Research, had admitted that he keeps files on anti-fluoridation organizations and their leaders, and she said that Ralph Nader had branded such activities as an “institutionalized witch-hunt”.

An attempt was made to prevent the distribution of the monograph Fluoridation: Errors and Omissions in Experimental Trials (Sutton, 1959) in the U.S.A. by, amongst others, the Nutrition Foundation Inc., which wrote to the distributors, Cambridge University Press, New York, on 20 January, 1960, declaring that:

“The professional standing of the Cambridge University Press among scientists and educators would seem to preclude publication of such a book by Cambridge University Press.”

In his reply (25 Jan., 1960) the manager of the Cambridge University Press said;

“... if you find inaccuracies in Dr Sutton’s book, we should be most grateful if you will point them out to enable us to make changes in any future printing.”

He did not receive a reply to his letter.

When this attempt to suppress the monograph failed, the Journal of the American Dental Association published an extensive criticism of it. That influential journal, in July 1960, devoted a three-page editorial to attacking the monograph. It stated:

“Last year the Melbourne University Press of Australia published an 83 page booklet by Mr P.R.N. Sutton entitled Fluoridation Errors and Omissions in Experimental Trials. It is now being circulated to a limited extent in the United States.
The following review prepared by J. Ferris Fuller for the New Zealand Dental Journal is herewith republished in full as it skillfully points out many of the errors and omissions in reporting which Mr P.R.N. Sutton has less skilfully employed in compiling his observations on the errors and omissions in fluoridation.”

By twice incorrectly using the term “Mr “, the editor of the J.A.D.A. conveyed to readers, particularly to American ones, that the author of the monograph was a layman, for all dentists and medical practitioners in America are given the title of “Dr” If the editor of J.A.D.A. had read even the title page of the monograph he must have known that the author had received the postgraduate degree of Doctor of Dental Science from the University of Melbourne. Therefore it appears that this “mistake” was made deliberately to deceive his readers.

Then followed the criticism by J. Ferris Fuller shown on pages ***327 to 330.

However, the editor of the Journal of the American Dental Association was so keen to denigrate the monograph that he failed to check the claims made in the “book review” which he re-published. Apparently he did not realize that the criticism by Colonel Fuller, although superficially “skillful”, was based on misquotations—that this critic condemned statements which the author of the monograph had not made, nor did he realize that Colonel Fuller had concocted many false and misleading comments of his own.

The most important publication which enables a reader to locate articles and books on dental subjects is the annual Index to Dental Literature published by the American Dental Association. This lists not only all articles and letters, but also all books and pamphlets published during the year, and has the reputation for being a comprehensive list. The Indexes for the years 1960 and 1961, which should have listed the first and second editions of the monograph did not do so, nor did they mention the favourable reviews.

However, they indexed the unfavourable ones, so that these omissions were obviously made intentionally by staff of the American Dental Association to suppress this criticism of fluoridation trials, which were the foundation for the endorsement of this process by that Association.

The following pages are a reprint of the second edition of that monograph: Fluoridation: Errors and Omissions in Experimental Trials, 1960, Melbourne University Press, which has been out of print for many years.
PART ONE

SOME STATISTICAL OBSERVATIONS ON
FLUORIDATION TRIALS*

The suggestion that domestic water supplies should be-fluoridated, with the aim of partially preventing the development of dental caries, has gained wide support, and moves are being made in Australia for the widespread introduction of this measure. Much confusion of thought clouds the issue of the desirability, the method of action and the safety of this process. This uncertainty is reflected in two recent events. In November 1956, a Reference Committee of the American Medical Association (1957) stated that “there is a definite need for a re-evaluation of the problem of fluoridation”,† and in March 1957, after a public hearing, the proposal to fluoridate the water supply of New York was not put into practice (Nesin, B.C., personal communication, 1957).

Apart from these considerations, an examination reveals that there are aspects that call for a very careful appraisal of the figures presented in the reports of the experimental trials which have been conducted in Brantford, Canada, and in Grand Rapids, Newburgh and Evanston, U.S.A., and upon the results of which proposals to fluoridate domestic water are almost entirely based.

A preliminary survey of the methods used, of the published figures and of the method of their presentation discloses some disturbing facts. Some of these are as follows. (i) In the clinical examinations no attempt was made to devise a randomization procedure, which would have eliminated bias on the part of the examiners. However, the necessity for such a precaution was recognized by Ast, Bushel, Wachs and Chase (1955) in the Newburgh-

† In December 1957, the American Medical Association endorsed the principle of fluoridation, but that decision cannot affect the facts which have been stated in this paper.
Kingston trial, when they instituted a combined clinical and X-ray study eight years after the commencement of the ten-year investigation. (ii) No estimate was made of variability between examiners, although in some studies several operators were employed, some being changed from year to year (Blayney and Tucker, 1948; Arnold, Dean and Knutson, 1953); some of the examinations in Kingston were made by two dental hygienists (Ast, Finn and McCaffrey, 1950). Furthermore, there appears to be no estimate of variability within the examiner—that is, the variability of individual examiners from inspection to inspection. (iii) The importance of random variation in the DMF rate (decayed-missing-filled permanent teeth rate) does not appear to have been recognized, or else it has been ignored. (iv) Bias is suggested by the presentation of some results, so that the casual reader may be misled (Ontario Department of Health, 1956).

The following observations will serve as illustrations.

1. In each of these studies it has been emphasized that the maximum benefits of fluoridated water are seen only in those subjects who have consumed it during the total period of enamel formation. Therefore, it would be expected that only a slight decrease (due to the possible topical effect of the fluorine) would be seen in the DMF rate between successive years during approximately the first six years of the project, until the first permanent teeth which had been completely formed under its influence had erupted, and that the advent of these “resistant” teeth would thereafter produce a greater drop in DMF rate between succeeding years. However, in the first three years of each project there is a marked relative fall in the reported DMF rate, particularly in the younger age groups; while in the six years-old group in Brantford the rate reached after ten years is no lower than it was after only four years of fluoridation, (Ontario Department of Health, 1956; Hutton, Linscott and Williams, 1956). It would appear that the results reported are not those which would be expected if the theory mentioned above is correct.

2. In four of these studies (Hutton et al., 1956; Hill, Blayney and Wolf, 1956; Arnold, Dean and Knutson, 1953; Ontario Department of Health, 1956) the method of expressing changes in caries experience was the same. The final rate was subtracted from the baseline rate, and the difference was expressed as a percentage of the latter rate. It is obvious, therefore, that with
this method, relatively small variations in the baseline values will produce substantial alterations in the percentage reduction obtained. For instance, in the seven-year-old children in Evanston, during the last five years reported, the increase in caries immune deciduous dentitions was 361 per cent, but for the whole of the nine-year period 1946-55 the increase was only 58 per cent “Hill et al., 1956). The authors claim that “difference between 1946 and 1955 rates is statistically significant” However, such a claim is not warranted, owing to the marked variation in the values observed in the intervening years. The effect of variations between years is seen in the six-yearold group in Brantford. By the use of this method of calculation the reduction in the DMF rate for the period 1944-50 was 82 per cent, but the apparent benefit had dropped to 52 per cent, a decrease of 30 per cent, after an additional two years fluoridation (Ontario Department of Health, 1956). An improved method of indicating relative changes in the DMF rate would seem to be desirable - in particular, one which would permit statistical tests to be applied.

3. As an instance of the divergent results which can be reported by different examiners, those from the two independent trials in Brantford may be compared (Ontario Department of Health, 1956). The National Health and Welfare authors reported a reduction in the DMF rate in the six to eight years age group of 69 per cent from the inception of their examinations in 1948 to the 1954 results. However, in the same city, in the same age range and between the same years, the reduction in the DMF rate obtained by the City Health Department examiner was only 25 per cent, less than half of that claimed by the authors of the other study. The Health Department DMF figures for 1954 were given for individual age groups without statement of the number of children involved in each group. The 25 per cent reduction is based on a DMF rate obtained by simple averaging of the six, seven and eight year DMF rates. For 1948 the actual numbers of children are available (Hutton, Linscott and Williams, 1951). The uncertainty in the computed reduction of 25 per cent is most unlikely to account for the gross difference between it and the figure of 69 per cent quoted by the National Health and Welfare authors.

4. In Table II of the Report of the Ontario Department of Health (1956) to the Ontario Minister of Health, the mean numbers of decayed or filled
deciduous teeth are shown. In the column headed “% Reduction Since 1948”, there are dashes opposite the control cities of Sarnia and Stratford. These, surely, would lead the reader to suppose that no reductions had taken place in these cities, particularly as the footnote states that “the rates for Stratford, which has had natural fluoridation for 30 years, and Sarnia, which has no fluoride in its water, have remained about the same”. However, in the nine to eleven years age group in Stratford there was a slight decrease of 5 per cent (by the use of the DMF rate reduction method common in these studies), and in Sarnia the same age group showed a decrease of no less than 16 per cent. One would like to know the reason for the omission of these figures, particularly as the latter reduction is almost as high as the 18 per cent claimed for children of the same age in the test city.

Whilst we do not question the integrity of workers in this field, it must be pointed out that the evidence tendered in favour of fluoridation reveals two disturbing features. The first is that what must be essentially a statistical study does not appear to have been planned as such. The second is that even when sufficient information is presented, no comprehensive attempt at statistical evaluation has been considered.

It is possible that a case for fluoridation can be solidly based, but until adequate statistical treatment of all the pertinent factors has been carried out and this would be quite a major undertaking the question should not be regarded as settled. In the meantime, claims concerning the amount of caries reduction are open to doubt.
INTRODUCTION

The fluoridation trials that were conducted in the cities of Grand Rapids, Newburgh and Evanston, in the U.S.A., and the two independent ones in Brantford, Canada, are of more than ordinary importance, because they constitute the main experimental evidence which has led to the introduction of this process as a public health measure. The fluoridation hypothesis is “that a concentration of about 1 part per million of fluoride in the drinking water, mechanically added, inhibits the development of dental caries in the teeth of the users of the water” (Brown, McLaren and Stewart, 1954b). In 1956 Nesin pointed out: “It must be emphasized that the fluoridation hypothesis in its entirety rests on a very narrow base of selected experimental information. It is this very base which is vulnerable to scientific criticism. And, it is upon this very narrow base that the very impressive array of endorsement rests like an inverted pyramid.”

The safety of artificial fluoridation has been questioned by a number of eminent authorities such as Hicks (1956) and Sinclair and Wilson (1955). In 1955 Box stated: “It is my considered opinion that the artificial fluoridation of water supplies, on a wholesale basis, should not be advocated or adopted until fully sufficient findings show that there are no harmful sequelae from a gingival or periodontal standpoint.”

However, these questions need be considered only if the overall dental benefits of fluoridation are demonstrated beyond reasonable doubt, and are also found to be worthwhile from a socio-economic point of view. No suggestion has been made that fluoridation has other than dental benefits.

It has been widely accepted that the existence of marked dental benefits has been established, and the literature abounds with references to reductions of about 60 per cent in dental caries as a result of fluoridation. However, the
published works contain little consideration of the numerical data reported from these trials, as distinct from mere statements of percentage reductions in the caries attack rates.

A preliminary examination revealed that reports of these studies contain errors and show omissions, and statements made in regard to results are not justified by published data; therefore further study has been made of these crucial trials. This study attempts to evaluate their controls, and the discussion is limited to examination of published reports of (i) method of selection of control cities; (ii) their suitability; (iii) the experimental and statistical processes used in gathering and analysing the data (iv) the results stating the dental caries attack rates; (v) some comments made by the authors of these trials (and by others) on these results.

The aim will be to investigate the reliability of the results reported, to assess the adequacy of the controls that were set up and to evaluate the accuracy of the statements made concerning the data obtained.
BASIC CONSIDERATIONS

Before discussing the procedure adopted in each of these studies, several basic matters that are of importance in a fluoridation trial will be considered.

The necessity for controls. Blayney and Tucker (1948) were correct in stating that “A study of this nature must have an adequate control.” The necessity for such a procedure was recognized by the authors of four out of five of these studies. Cities with “fluoride-free” water supplies were selected as controls, and comparisons were made with towns which possessed water supplies with a fluoride content obtained from natural sources, which approximated the concentration which has been called the “optimum” one (Dean, Arnold, Jay and Knutson, 1950; Brown, 1951; Ast and Chase, 1953; Hill, et al., 1951). It is to be noted that in the trial conducted in Brantford by the City Health Department (Hutton et al., 1951) no provision for controls was made.

Requirements of a control. In an experiment such as the fluoridation of the water supply of a city, whereby the whole of its population is subjected to treatment (fluoridation), it is necessary to obtain the control data from subjects who live in a city or cities with “fluoride-free” water supplies. In determining the cities which are to participate in the trial, in order to increase the sensitiveness of the experiment, it is advantageous to employ ones which are alike in as many respects as it is practically convenient to consider. Of course, as Fisher (1951) pointed out, “the uncontrolled causes which may influence the result are always strictly innumerable.”

Because of the nature of these experiments, three main points of similarity must be considered and described. These are (a) the water supply; (b) the climate; and (c) the dental caries attack rates. Other factors, such as socioeconomic status, are of less importance; their influence may be reflected in the caries attack rates.

(a) In its statement of its official policy on this matter, the American Water Works Association (1949) said that the experimental verification of the fluoride-dental caries hypothesis “obviously necessitates the use of a nearby “control” city with a water supply comparable in all respects to that to which fluoride is being added.” The Association referred to “the possible
influence, on the fluoride potency, of other chemical constituents of natural waters, insofar as these and other variables may affect the action of fluoride on the control of caries in a human population.” In 1942 Deatherage reported that “It is these soft waters which cause the most severe mottled enamel.” Therefore, the fact that both the test and the control city in a fluoridation trial obtain their water from the same source does not remove the necessity for a study of the composition of the water. Dean, Jay, Arnold, McClure and Elvove (1939) recognized this, stating, “the possibility that the composition of the water in other respects may also be a factor should not be overlooked. For this reason it seems highly desirable that dental caries studies should be accompanied by complete chemical analyses of the dam waters, including a search for the comparatively rare elements.” However, in none of these trials was the composition of the water stated.

(b) The climate of a city is an important factor in determining the average amount of salts ingested from the water supply, because of its influence on the volume of water consumed by humans. Therefore, cities that are to be compared should not only have water supplies that have a closely comparable composition, but the climates of the cities should also be very similar.

(c) As the main aim of fluoridation is to reduce the dental caries attack rates, it is obviously of importance that the cities to be compared should have closely comparable dental caries rates within yearly age groups, of children. This information can be obtained only by conducting at least one survey in the cities that are suitable for comparison on other grounds, so that the fact that the caries attacks rates are similar is established prior to the fluoridation of the water supply of one of them.

Random sampling. The fundamental importance of random sampling has been acknowledged for many years. In designing an experiment, as Quenouille (1952) said, “it is necessary to allot the treatments to the available material at random if unbiased estimates of both the effect of the treatments and also the reproducibility of the effects are to be obtained.” Therefore, a random device should be employed to determine which of the participating cities is to be the test one.

Variation. Fisher (1950) emphasized this important matter when he said that “from the modern point of view, the study of the causes of variation of any variable phenomenon, from the yield of wheat to the intellect of man, should be begun by the examination and measurement of the variation
which presents itself.” As was pointed out by Hill et al. in 1950: “It is to be expected that the rate of caries in all teeth varies from year to year due to chance.” Therefore, a basic requirement of a fluoridation study is the assessment of the variability of the caries attack rates.

**Examiner variability.** In experiments in which, of necessity, the subjective judgment of examiners is employed, an important consideration is the assessment of “between-examiner” and “within-examiner” variability. The former type of variability is disclosed when different examiners observe the same subjects, and the latter type is seen in the different results reported by the same examiner inspecting the same subjects on different occasions, but which are sufficiently close together to ensure that the dental condition has not undergone appreciable change.

The important effect which examiner variability can have on the results of a study of dental caries attack rates was pointed out by Radusch (1941) and by Dunning (1950). A recent example is seen in the paper of McCauley and Frazier (1957). Their Table I shows that in the examinations made by one examiner in 1955 of Negro boys and girls who were six years old, in both sexes the DMF rate per 100 teeth erupted, and also the DMF rate per child, were found to be about four times as great as those reported for the same age groups in 1952 when they were examined by several examiners. The authors considered that “it is entirely possible that the 1952 findings were influenced by a bias stemming from subjective differences in the appraisal of tooth decay by different dentists”. Between-examiner variability of such a magnitude can, of course, vitiate the results of a study. Unless the examiner variability is determined, and is taken into account, the conclusions drawn from a study of caries attack rates must be treated with reserve.

**Examiner bias.** In designing an experiment of this nature, one aim should be to eliminate examiner bias. This may arise if the examiners know whether the children they are examining belong to the test or to the control city. One method of doing this is to transport to a common examination centre the small number of children, some from the test and some from the control city, that can be examined each day; the examinations being conducted in a random order which is unknown to the examiners. It is not suggested that in the absence of such precautions the examiners exhibited intentional bias; indeed, as Armitage (1954) pointed out, “through fear of being biased” the judgment of an examiner may be influenced.
THE GRAND RAPIDS STUDY

The city selected as the “fluoride-free” city for comparison with Grand Rapids was Muskegon, Michigan, “whose source of drinking water supply and geographical and climatological characteristics were similar to those of Grand Rapids” (Dean et al. 1950). This city was the only control one in which the caries attack rates in each year were published for each yearly age group. Unfortunately, its usefulness was marred by a number of features.

Large differences in sample size. The reliability of a mean rate is greatly influenced by the number of observations on which it is based. Because of the small number of subjects included in some age groups in some years in Muskegon, little reliance can be placed on the values stated. In twelve categories fewer than twenty children were examined. One “group” consisted of only one child, whereas one contained 462 children (Arnold et al., 1953). In the test city the variation in sample size was even greater, from 18,606 to 3 subjects.

Sampling by school class. “selected age groups of children are examined within each of the schools. Selection is made on the basis of school grade or class, using all children present in a class or grade of a school.” (Arnold et al. 1953). These grades or classes were examined in 1945, an additional grade being examined in 1946, 1947 and 1949, and two more in 1950, making a total of eight grades in 1950 and 1951. In the last mentioned year Muskegon ceased to act as a control.

Different methods of sampling. In Grand Rapids the “annual study sample was selected after careful review of census data and consultation with city planning department officials. On the basis of available information, the 31 school districts of Grand Rapids were classified on a socio-economic basis. From the 79 schools in these districts, 25 representative schools were selected, and the examiners were assigned schools on a basis of equal sized samples of comparable population groups” (Arnold, et al., 1953). However, that strange procedure was not followed in Muskegon, the same authors stating that “In Muskegon, the annual examinations have been conducted in almost all schools, excluding only a few small schools on the periphery of the city where many students are from rural areas.”

Changes in examiners. In the report of this study up to and including 1951
(in July 1951 Muskegon ceased to be a control), it was stated: “There have been changes in dental examiners with the exception of one officer who has participated in each series of examinations. Each new examiner has been calibrated against this one officer to standardize diagnostic criteria” (Arnold et al., 1953). The degree of success of this odd calibration procedure was not stated.

*Examiner variability not assessed.* In 1953 Arnold et al said that “Bite-wing X-ray examinations were made of a representative sample of children examined by the different examiners to evaluate, in part, the “examiner error”.” However, such a procedure cannot replace the data that could have been obtained by a correctly designed examination process, which would have enabled the determination of between-examiner and within-examiner variability.

*Late examination of control city.* The authors of this study did not determine the caries attack rates in children in Muskegon prior to accepting this city as a suitable control, for comparison of two of their statements makes it clear that the results of the basic examination in the control city were not known until after the water of the test city was fluoridated. They stated that “Fluoridation of the Grand Rapids water supply was started January 25, 1945” (Dean et al., 1950), and that the “basic examinations in Muskegon were not done until late spring of 1945” (Arnold et al., 1953).

*Water of control fluoridated.* Another fact which limits the usefulness of Muskegon as a control city is that its water supply was fluoridated in July 1951 (Arnold et al., 1953), so that the results obtained after that date had no value as controls for those of Grand Rapids. This event occurred six and a half years after the institution of fluoridation in Grand Rapids, and therefore at a time when, in the latter city, few of the permanent teeth had erupted in the children that had been ingesting fluoridated water since birth.

*Ignorance of commencement of fluoridation in control city.* The fact that Muskegon had ceased to be a control by having its water fluoridated in July 1951 was not always realized. For instance, Black (1955) in a paper “Presented before Section on Public Health Dentistry, ninety-fifth annual session, American Dental Association, Miami, Fla., November 8, 1954” - over three years after the institution of fluoridation in Muskegon (Arnold et al. 1953) - said that “At Muskegon, Mich, the control city where fluoride-free water is used, the incidence of dental caries is unchanged and approximates
the norm.” Black was commenting on the findings made “After eight years of fluoridation at Grand Rapids” (that is eighteen months after the fluoridation of the Muskegon water). No information has been found in the literature with regard to the “national norm”, in fact a feature of these trials has been the divergent pre-fluoridation rates. The differing caries attack rates seen in different localities, even in the same state, were illustrated by Hagan (1947) and by Hadjimarkos and Storvick (1949, 1950).

A similar statement to that of Black (1955) was made by Martin (1956) in a lecture delivered at the fourteenth Congress, Australian Dental Association, Melbourne, March 1956; that is, over four and a half years after the Muskegon water was fluoridated. He said: “The decay rates in the fluoride free control area (Muskegon) have remained unchanged.” The paragraph containing the above-mentioned quotation cites as reference Arnold et al. (1953), who in that paper said: “The water supply at Muskegon remained unchanged until July 1951, when the city started adding fluorides to its water supply.”

**Variations in Muskegon rates.** Tables 3 and 4 of the report of the seventh year of the Grand Rapids study (Arnold et al., 1953) show that both the def (decayed, extraction indicated, or filled deciduous teeth) and the DMF rates reported from Muskegon from year to year differed considerably from those of the first examination. Despite this fact, the statement that the incidence of dental caries in Muskegon was unchanged was made by Black (1955) and Martin (1956), amongst others. These statements are at variance with that of the authors of the study (Arnold et al., 1953), for they mentioned the changes in these words: “A similar comparison of results at Muskegon shows the percentage reduction to range from 1.5 percent in 6-year olds to a high of 15.5 percent in the 11 year olds” in the permanent teeth.

**The magnitude of the changes.** The statement which has been quoted above does not reveal the magnitude of the changes which were observed in the DMF rates in the control city. The percentage reductions given were obtained by the method commonly used in all these studies, that is, the difference between the most recent and the original DMF rate was expressed as a percentage of the original rate, the variations obtained in the intervening years being ignored. The changes which occurred would have been more obvious if, for instance, the results for Muskegon had been computed in 1946 instead of in 1951. In that case the “reduction” would have been 40.7 per cent instead of 1.5 per cent in the six-year old, and 32.7 per cent instead of 15.5 per cent in the eleven-year-old children.
**Sampling error.** Arnold et al. (1953) stated that the percentage reductions obtained in Muskegon “may, in part, represent sampling error.” It was not conceded that such an error could also apply to the results from the test city, nor were suggestions made as to the cause of the remainder of the reductions in the control city. These cannot be attributed to changes in the water supply, for they stated: “The water supply at Muskegon remained unchanged until July 1951, when this city started adding fluorides to its water supply” (Arnold et al., 1953).

**Variation in Muskegon.** The variability from year to year in the mean rates reported from Muskegon, which, it will be recalled, were stated to have remained unchanged (Black, 1955; Martin, 1956) are illustrated in Figs. 1 and 2. The data from which these figures were drawn are from Tables 2 and 3 (with errata corrected) of Arnold, Dean, Jay and Knutson (1956). The point shown in Fig. 2 for the sixteen year-old children in 1946 should be disregarded, as this age “group” consisted of only one child.

Comparison of the series of baseline rates for DMF in Grand Rapids and in Muskegon does not reveal that one series was consistently higher than the other. However, with the exception of the eleven and twelve-year old children, the def rates in Muskegon were higher than they were in the test city.

**Comparison with Aurora.** Arnold et al. (1953) said: “To establish what might be termed an “expectancy curve” for this study, a natural fluoride area, the city of Aurora, Ill., was selected. The Aurora water supply contains 1.2 ppm F and has a reliable “history of constancy back to 1895.” It was not stated whether factors other than the fluoride content of the water supply were considered in selecting this city. The fact that other influences can be of importance was shown in the recent study by Russell (1956) in Montgomery-Prince Georges counties. Prior to the institution of fluoridation, in the total sample of subjects the def rates for children whose mean ages were 5.44, 6.47, 7.45 and 8.49 years were lower than those of children of similar ages in Aurora.

**Limitations of Aurora data.** The caries attack rates reported from Aurora consist of a single series obtained by several examiners in 1945-6. Therefore, there is no information with regard to variations from year to year in the mean value of the rates, and examiner variability was not considered.
Figure 1. The mean number of def deciduous teeth per child in Muskegon, Michigan, the “fluoride-free” control city for Grand Rapids, Michigan, at each year of examination. The 1945 examination was made in the “late spring”, those of the other years, in October and November. Data from Table 2, Arnold et al., 1956. Three months prior to the 1951 examination the water of this city was fluoridated. It has been stated (see p. 145 - 146) that these rates “remained unchanged” during the period shown.
Figure 2. The mean number of DMF permanent teeth per child in Muskegon, Michigan, the “fluoride-free” control city, at each year of examination. Data from Table 3, Arnold, et al., 1956. It has been stated (see p. 145 - 146) that these rates “remained unchanged” during the period shown.
The United Kingdom Mission (1953), after having observed the Evanston study, described it as “one of the most elaborate investigations.” Hill et al. (1950) considered that they had planned the study so “as to measure every variable that might exert an influence and obscure the findings.” It is the only trial in which bite-wing examinations were made for all subjects examined.

The importance of X-ray examinations. Blayney and Greco (1952) reported that in this trial “the X-ray disclosed 53.84 per cent of the total number of carious lesions observed by both clinical and X-ray methods”. That said: “We believe it extremely important to employ both clinical and X-ray techniques in any study program which is directed toward the determination of the prevalence or the control and reduction in the rate of caries attack.” This result must throw considerable doubt on the accuracy of the caries attack rates which were reported from the test and control areas in the other studies considered; for in these, X-ray examinations were incomplete or absent.

The ideal control community. The authors of the study stated that “It seemed logical to think of Oak Park, Illinois, as the ideal control community because of its close similarity to the study area” (Blayney and Tucker, 1948). The manner in which that city resembled Evanston was not stated. The United Kingdom Mission (1953) made the important observation that in Evanston the economic level was high, and “dental care was outstandingly good.”

Lower caries rates in control community. It soon became apparent that Oak Park could not be called “the ideal control community”, for Hill et al. (1951) stated that “Comparison of the caries rates of all children in the study area (Evanston, Ill.) and the control area (Oak Park, Ill.) prior to the addition of sodium fluoride to the communal water supply of the study area indicated a lower caries rate for school children of the control area.”

Different rates in student groups. The authors continued:

In an effort to find the source of these differences in caries prevalence, it was found to be due largely to differences in the make-up of the student groups examined in the two areas. While in the study area 22.2 per cent of the children examined were attending parochial schools, no such children
were included in the control area: and while 5.6 per cent of the children in the study area were Negro children, only 0.1 per cent of the children in the control area were Negro. Statistically significant differences were found to exist between the caries rates of Negro and parochial school children on one hand and public white school children on the other hand. Generally the caries rates of parochial school children were found to be higher and those of Negro children lower than those of white children in public schools.

Exclusion of data. Hill et al. (1951) continued:

Therefore, comparisons of caries rates for the study group and the control group are based on the caries experience of public white school children only, while such comparisons involving children in only the study area are based on the caries experience of all children in total. The caries rates for the Evanston white school children in the 1946 survey and the Oak Park white school children in the 1947 survey were very similar.

Six lines later, it was stated: “In further comparing the rates for Oak Park (control) and Evanston (study area) it is apparent that the baseline figures are very similar.”

The only comparisons that can be made from the paper which has just been mentioned are the figures for the children aged twelve, thirteen and fourteen years. Negro and parochial school children constituted 27.8 per cent of the Evanston children. By excluding this part of the data the rates in that city were then considerably lower than those in the control city, the rates (Table IV) being 707.51, 946.17 and 1133.33 in every 100 of the Evanston public school white children for the ages twelve, thirteen and fourteen years; those in Oak Park being 774.29, 970.00 and 1194.64 for the same three ages.

An altered explanation. A different, but, at first sight, a reasonable explanation for the exclusion of the data of Negro and parochial school children, when making comparisons with data from Oak Park, was given in the XV Report (Hill et al., 1957a): “As the control area (Oak Park) examinations included only public school white children it was necessary to evaluate the Evanston data on the basis of school groups, public white, parochial, and Foster (Negro) to make comparisons of like groups.” It can be seen that in that paper the exclusion of data was attributed, not to the
fact that this process was undertaken because there was “a lower caries rate for school children of the control area” (Hill et al., 1951), but to the different racial composition of, and type of school attended by the children in the two cities. Hill et al. (1950) mentioned that one of their seven “other objectives” was “to compare the dental caries experience of white with that of Negro school children.” No reference was made to the possibility of a difference being found between the rates of white public and parochial school children. However, the original statement (Hill et al., 1951) makes it clear that the different school groups were taken into account only after the unsatisfactory results of the first Oak Park examination became apparent.:
“In an effort to find the source of these differences in caries prevalence.”

In assessing the accuracy of the second (1957a) explanation, it should be realized that in the younger age group “comparisons of like groups”, or even the dissection of the data into the three school groups, were not published in the reports dealing with that age group, namely the 1950, 1952, 1954, 1956 and 1957b papers, or even in the XV Report (Hill et al., 1957a) which dealt with both age ranges, but showed this dissection for the children of the older age group only. Furthermore, when, after a delay of more than ten years, the 1947 Oak Park rates for the younger children were published for the first time by Hill et al. In 1958, no “comparisons of like groups” were made by them. The reader is prevented from making this comparison by the fact that, even now, the dissection of this age range into the three school groups has not been published, despite the statement by Hill et al in 1951 that the rates for “school children” were significantly different in each type of school.

“Correction” of data. When making comparisons with the control city, the authors excluded from the three groups of data obtained in the test city the two which diverged most from the rates of the children in the control city (Hill et al., 1951). This process should be considered in connection with the following statement (Hill et al., 1950):

In order to be able to generalize from our findings, we must be certain that any such variables as effect caries experience are represented in our study to the same extent as in the population. Before drawing any ultimate conclusions, we will, therefore, correct our data in such a manner as to include only those groups of children which are representative of
the population, with respect to dental caries experience. We feel that this precaution is necessary to allow the ultimate findings to be considered valid and reliable.

However, the process which they described - the arbitrary selection of a section of the data, which is then termed “representative” - instead of making “the ultimate findings to be considered valid and reliable”, would render a report based on this selected data unfit for serious consideration.

“Population” sampled. It is not clear what the authors meant by the term “the population.” If the population referred to was that of Evanston, the sample of children examined in this study - if properly drawn - provided an unbiased estimate of the dental condition of the population of that city; if only some of the data are included, the results will be biased. If this term “population” was intended to refer to the general population of the U.S.A., it should be realized that the results from Evanston can represent only a stratum of the country as a whole, varying as to climate and racial composition, to mention only two variables.

It will be recalled that the caries rates were said to be significantly different, even between children attending the different types of school in Evanston; and also that the rates in that city were considerably different from those in Oak Park, which was at first stated to be “the ideal control community” for Evanston (Blayney and Tucker, 1948). These differences emphasize the fact that caution should be exercised when applying results obtained in a test city to a wider population, of which the test city may not be representative.

Altered methods in latest report. In the latest report (Hill et al., 1958) which shows the findings for the permanent teeth of children in the control city of Oak Park, the authors have published in the same tables as the results of the control groups, the DMF rates, not of the public school white children, but of the total sample of Evanston children. This is strange in view of their statement that “comparisons of caries rates for the study group and the control group are based on the caries experience of public white school children only” (Hill et al., 1951). It would appear that they no longer held the opinion which they stated the previous year (Hill et al., 1957a) that it is necessary “to make comparisons of like groups.”

As a result of this change in procedure the differences between initial caries rates in Evanston and Oak Park are diminished. In children aged twelve to fourteen years, the pre-fluoridation rates reported for the 1,226 public
school white children in Evanston were far closer to the values found in the Oak Park children than were either the rates of the 96 Negro, or of the 379 parochial school children (Hill et al., 1957, 1958). However, the rates of the Negro children were lower, and the rates of the parochial school students were considerably higher than those of the public school white children. By adopting the authors' latest (1958) method, which is to add the results of the three groups, it is found that the pre-fluoridation rates of the twelve and fourteen-year-old children are considerably less divergent from those of the initial examinations in Oak Park - and those of the thirteen white children of those ages. Whether this situation arises with regard to the six-, seven- and eight-year old children cannot be determined, for no dissection into the rates prevalent in the three school groups has been published.

Late examination in the control city. The United Kingdom Mission (1953) stated: “Before fluoridation started a dental survey was made of 4,375 children in the selected groups in Evanston and of 2,493 children in Oak Park. Further examinations have been carried out each year since 1947 and will continue until 1962.” However, the examinations in Oak Park were not commenced until after the fluoridation of the Evanston water supply on 11 February 1947, for Blayney and Tucker (1948) stated: “The study in Oak Park was instituted on Feb. 26, 1947”. Also, at the time of the United Kingdom Mission Report (1953), no further examinations had been conducted in Oak Park; even in Evanston only one age group was examined during each year, as can be seen by inspecting the “schema for study” published by Blayney and Tucker in 1948, and reproduced in several subsequent reports.

Only two examinations in the control city. This “schema” indicates that the design of the trial provided for only two examinations - eleven years apart - to be made in the control city. It would appear that the authors did not anticipate changes in the caries rates of the control, such as were reported in Muskegon (Arnold et al., 1953), and, as will be seen later, in Sarnia (Brown et al., 1954b), and in Kingston (Ast, Finn and Chase, 1951). The first examination was made in 1947, and the second, although not scheduled until 1958, was commenced in 1956 when it became apparent that the water supply of Oak Park would be fluoridated (Hill et al., 1956). This examination was completed on 14 November 1956, soon after the fluoridation of the Oak Park water on 1 August (Hill et al., 1958).

A ten-year delay in the publication of data. Caries attack rates for the six-,
seven- and eight-year-old children which were obtained in Oak Park in 1947 (Blayney and Tucker, 1948) have only recently been published by Hill et al. (1958). This great delay is inexplicable and is particularly unfortunate, because it is in regard to these younger children that the major claims are made for reduction of dental caries as a result of fluoridation. No explanation was offered for this delay, and the members of the United Kingdom Mission (1953) did not comment on this strange omission, merely saying that “The incidence of caries among the children aged 6-8 years is compared with the baseline data of Evanston itself while caries experience of children aged 12-14 years is compared with that of Oak Park.”

*Gross differences in initial caries rates.* The latest report (Hill et al., 1958) reveals that in the younger children there were gross differences between the initial caries attack rates in Evanston and Oak Park. The rates were: 46.85, 26.89 for age six years; 153.49, 102.63 for age seven years; and 249.93, 222.44 for age eight years in Evanston and Oak Park respectively.

In regard to the great difference between the pre-fluoridation rate for the six-year-old children in Evanston and the initial one for children of that age in Oak Park, 46.85 and 26.89 respectively, a footnote to Table I (Hill et al., 1958), referring to the former rate, stated: “This figure results from the very high DMF rate of 87.91 found in one school in 1946.” However, as the children were drawn “from 24 schools in the study area” (Blayney and Greco, 1952), it is probable that the rates for six-year-old children in most schools approached the figure of 46.85, unless the school with the high DMF rate also happened to provide a disproportionately large number of six-year-old children.

It should be noted that no comment on the magnitude of this rate of 46.85 was made in any of the four reports in which it had been shown previously (Hill et al., 1950, 1952, 1956, 1957a); all of which were published before the rate of 26.89 for Oak Park was released, and therefore before a comparison with it could be made. The rate of 46.85 was used in all those papers - and even in their latest report (1958) - in calculating the “% reduction”, and in computing the “Probability of difference due to chance.”

*Much unpublished data.* The members of the Evanston Dental Caries Study devoted most of the years 1947 and 1956 to the collection of data
from children in Oak Park (Blayney and Tucker, 1948; Hill et al., 1958). Despite this fact, the major part of each of the two tables shown in the XVIII Report (Hill et al., 1958) was devoted to a re-presentation of data obtained in Evanston, although this report was said to have as its purpose the comparison of the permanent teeth dental caries experience rates in children examined in Oak Park in 1947 and 1956. The Oak Park data were restricted to four lines of figures showing the DMF rates in permanent teeth. No report was made of other findings such as those which had been shown in reports on Evanston children. For instance, in the XV Report (Hill et al., 1957a), no fewer than eight tables relating to the twelve-, thirteen and fourteen year-old children only were devoted to these other findings. This very incomplete presentation of the data obtained in Oak Park is unaccountable.
Disagreements between results. In their XVIII Report, Hill et al. (1958) stated: “The DMF rates and percentage reduction from year to year for the Evanston children of all age groups shown in Tables I and 2 have been published in previous reports. However, four of the figures for the year 1955, shown in Table I of the 1958 Report, are different from “the rates and percentage reduction” given, for the same year, in Table I and the text of the XVI Report (Hill et al., 1956). The DMF rates at age seven years were only slightly different (40.95 and 40.92, in the XVI and the XVIII Reports respectively), but at age eight years the two rates were 114.04 and 120.32. It is very improbable that these different rates are due to typographical errors, for they were confirmed by the “per cent reduction from 1946”, which was given in the summary and in Table I of the respective reports as 73.32 and 73.34 for children aged seven years, and as 54.37 and 51.85 for those that were eight years of age. This “reduction” was shown in the XVIII Report as 85.96 for the six-year-old children, but in the XVI Report it was given as “80 per cent” in the findings and as “85.96 per cent” in the summary.

Disagreement between tables. The DMF rate in terms of tooth surfaces was given only twice in this study (Hill et al., 1955, Table X and 1957a, Table XII). In both papers the “DMF rate per 100 surfaces” for children aged fourteen years was 14.82 in 1949 and 13.94 in 1952. However, in the former report this rate was given as 15.09 in 1946, but in the latter one, for children of the same age in the same year, the figure shown was 15.92. As a result of this change, the “% differences from 1946” were altered from 1.78 to 6.85 (1949) and from 7.62 to 12.44 (1952). By using these new rates it can be said that “all 3 methods, namely; per hundred children, per hundred teeth, and per hundred surfaces all express approximately the same proportion of percentage differences in rates” (Hill et al., 1957a). This result is a good illustration of the comment made on the method most commonly used in these studies to express changes in caries experience, that “relatively small variations in the baseline values will produce substantial alterations in the percentage reduction obtained” (Part One, p. 137).

It may be mentioned that the “total tooth surfaces considered” for thirteen-year-old children in 1954 (Table X11, Hill et al., 1957a) should be 58,325 not 58,352; and that for fourteen-year-old children in 1949, in the column of that table giving the “% differences from 1946”, the figures shown should be 6.91 not 6.85. In their XI Report (Table IX) and their XV Report (Table
XI), Hill et al. (1955, 1957a) showed different figures for children aged twelve years examined in 1952. Although both tables show the same total number of teeth considered, in the former table children were shown as examined, with a “DMF rate per 100 teeth” of 25.76, and a difference from 1946 of 19.50 per cent. In the latter table, the figures were 516, 25.60 and 20.00 per cent respectively. In 1953 Hill et al. published the figure of 19.50 per cent.

*No data for deciduous teeth.* The authors have not published any data regarding the deciduous teeth of children in the control city, either for the first (1947) or the second (1956) examination. The most important omission, the def rates, could have been shown by adding only two lines to Table I in Hill et al. (1958). This omission is particularly unfortunate in view of the fact that in the deciduous teeth in Evanston during the first four years of fluoridation the def rate of the six to eight years group was considerably higher than the initial one (Hill et al., 1952). It was not until nine years after the commencement of the study that a significant reduction in this rate was reported.

In 1950, Hill et al. stated that the caries rate for deciduous teeth in these children “does not indicate any trend”, despite the fact that in Table I of that report the initial rise in this rate during the first two years of fluoridation was shown by them to be statistically significant (P = 0.005). Two years later these authors altered their opinion of the significance of this rise. In 1952 they re-published the same data for children aged six, seven and eight years in 1946 and 1948, but computed different rates for the combined age group six to eight years. The rise in the def rate was then said to be not statistically significant.

*Variations in caries rates in control.* The meagre data regarding caries attack rates in Oak Park which have been published are included in Tables I and 2 of Hill et al. (1958). Of the six age groups shown, between the years 1947 and 1956 the authors reported a significant increase in the DMF rate of children aged seven years, and non-significant upward trends in the rates of those aged eight and thirteen years, and downward ones in the caries attack rates in children aged six, twelve and fourteen years. (The question of “significant” changes in the rates in control cities will be considered later.) The authors said: “The children 12, 13 and 14 years of age, Table 2, have only minute differences between the 1947 and 1956 rates. These are not
considered to be significant.” The footnote to that table is more definite, in each comparison stating: “Difference is not statistically significant.” Although these differences of 61.20, 34.96 and 58.87 DMF teeth, for children aged twelve, thirteen and fourteen years respectively, were termed “minute differences”, those seen in the rates of the twelve and fourteen-year-old children are approximately a third the size of the absolute drop in the rates recorded for the same age groups in Evanston since the inception of fluoridation. It cannot be assumed that the fluctuations in the rates during the intervening period of nine years, when no examinations were made, did not exceed the differences between the initial and final rates. It will be recalled that considerable variations occurred in Muskegon (see Figs 1 and 2).

Inadequacy of the control. Blayney and Tucker (1948) realized that “A study of this nature must have an adequate control.” Therefore, it is strange that in the “schema” which they published there was provision for only two examinations, eleven years apart, to be made in the control area. It should have been obvious that the usefulness of data gathered in such a manner would be, at most, very limited. The explanation given by the authors for their failure to examine the children in the control city “every year” (instead of only twice) was the strange one that “It was not necessary to do so in as much as Evanston and Oak Park are subjected to the same advertising campaigns, have a similar economic level, participate in comparable educational programmes, and so forth” (Hill et al., 1958). It is extraordinary that the authors advanced this explanation and that they adhered to such a plan, despite the marked disparity in caries rates disclosed in the first examinations in Evanston and Oak Park (Hill et al., 1958), which makes it obvious that the latter city was a poor choice in seeking an “adequate control” for the former one.

Differences between school groups. Hill et al. (1951) stated that “statistically significant differences were found to exist [in 1946] between the caries rates of Negro and parochial school children on one hand, and public white school children on the other hand.” However, they made a further statement that “the caries rates of parochial school children were found to be higher and those of Negro children lower than those of white children in public schools” (Hill et al., 1951). These two statements are inconsistent. The first appears to mean that the comparisons between Negro children and white children in public schools, and that the comparison between white children
attending parochial schools and those attending public schools, were both statistically significant in 1946.

“Nearly comparable” or significantly different? The XV Report Hill et al., 1957a) stated that “In 1946 and 1954 the public school white children and the Foster School (Negro) children maintained nearly comparable DMF rates”. The actual rates” (per 100 children) in 1946 for twelve, thirteen and fourteen-year-old white children attending public schools were 707.51, 946.17, and 1133.33; for the Negro children of the same ages they were 658.82, 861.76 and 1035.71. (The rates of each school group of younger children were not published.)

It is not understood how the same authors could on one occasion (Hill et al., 1951) state that there were “statistically significant differences” between the two series of rates, and later (Hill et al., 1957a) describe them as “nearly comparable DMF rates” It may be thought that the word “maintained” referred to a comparison between the DMF rates of the white children in public schools, and of the children in the Negro school, between 1946 and 1954. However, this cannot be the case, for the authors claimed for these twelve, thirteen and fourteen-year-old children “a reduction of approximately 21.96 per cent in dental caries-experience rates of the permanent teeth” (Hill et al., 1957a). (In this study, percentages were frequently shown “approximately” to two decimal places.) Table IV of that paper shows that both the Negro and the public school children participated in the reductions reported.

Decline in eruption rate. An observation of considerable interest is obtainable from Tables V and VI of the X Report (Hill, et al., 1952). The former table shows the rates per 100 six, seven and eight-year-old children that had occlusal surface pit and fissure caries or fillings in their first permanent molars; the latter one, the number of these teeth which were free from those defects. The mean number of erupted first permanent molars per 100 children may be obtained, in each age group, by adding these two rates to that showing the extracted and congenitally missing permanent molars. It is probable that the number of congenitally missing teeth was negligible and that the number of permanent molars which had been extracted in these young children was small, particularly in the six years age group (five and a half to six and a half years). Therefore, it would be expected that, in each age group, the mean number of erupted molars per 100 children would be similar at the time of each examination. This was the case in children aged eight years; the figures for the examinations made in 1946 (pre-fluolidation),
1948, 1950 and 1951 being (to the nearest whole number) 387, 387, 384 and 386 respectively. At age seven years the numbers erupted were 330, 336, 320 and 315; but in the six-year-old children, the number of erupted molars showed a marked and progressive decline 189, 156, 140 and 132 during the period covered by those four examinations.

The question naturally arises whether the eruption rate of these teeth had decreased; a possibility of extreme importance in interpreting the results of a fluoridation trial. However, further consideration of this matter is prevented by the authors’ failure to publish this type of data when they reported the results of the two later examinations (conducted in 1953 and 1955) which were made of children of these ages; and the “schema for study” indicates that children aged six to eight years will not be examined again until 1960.

This failure to publish this type of data for the 1953 and 1955 examinations

Figure 4. Suggestion of a progressive decline in the number of erupted first permanent molar teeth in six-year-old children in Evanston. The results obtained in the examinations conducted in 1953 and 1955 were omitted from the published reports.
is extraordinary, especially in view of the fact that the authors continued to show similar data for the permanent molars of the older age group (Hill et al., 1955, 1957a); the latter report, the only one showing results for both age groups, gave the prevalence of occlusal pit and fissure caries and fillings in the molars of the older, but not of the younger age group.

In considering the eruption of teeth, the odd method of assessment used in this study must be taken into account. Hill et al. (1955) said: “Only teeth which were 50 per cent or more erupted were considered. A carious or filled tooth was, of course, considered regardless of its stage of eruption.”

_Strange superiority of artificial fluoridation._ The authors of this study compared the Evanston DMF rates per child with those of children in Aurora, Illinois (Dean et al., 1950) in the expectation that after sufficient time had elapsed for all the erupted teeth to have been formed since fluoridation commenced “the Evanston rate will closely approach the Aurora rate” (Hill et al., 1957a). It is surprising that this parity between the rates of Aurora and Evanston was expected, because in the Aurora survey only clinical methods of examination were used, but in the Evanston examinations X-ray surveys were used routinely. Hill et al. (1951) stated: “We find our baseline figures for caries experience in Evanston and Oak Park approximately 32 per cent higher than those of Dean and his co-workers for Evanston and Oak Park in 1941. We assume this may be explained partially by differences in the techniques of examination, particularly in the use of X-ray in the current investigation.” The United Kingdom Mission (1953) stated that in this study “the minutest radiolucency was taken as indicating caries.”

In view of these findings, it is even more strange that Hill et al. (1957a) were able to report: “The Evanston 6 and 7-year-olds of 1953 have a lower dental caries experience rate after 71 to 82 months of fluoridation than the Aurora 6 and 7-year-olds of 1945-1946 with lifetime exposure to water naturally fluoridated to 1.2 ppm.” That this difference was not only slightly below the 1945-1946 Aurora rate for children of the same age” (Hill et al., 1957a) can be seen by comparing the actual rates reported. In Evanston and Aurora respectively, the rates were 14.73, 28.0 at age six years and 53.35, 70.5 at age seven years (Hill et al., 1957a; Arnold et al., 1953). It should be noted that in Evanston two years previously (1951), after a shorter period of fluoridation, the rate for the six-year-old children was even lower, 12.36 (Hill et al., 1952) and was less than half the Aurora rate; in 1955 (Hill et al.,
1956) it had become 6.58, less than a quarter of the Aurora rate. Blayney and Greco (1952) found that in children in the Evanston study, with regard to proximal caries “the 6-year-olds have the highest percentage (83.90) disclosed by X-ray findings only. In the 7-year-old group 79.04 per cent of proximal lesions were demonstrated by X-ray findings only”. Therefore, if clinical methods of examination only had been used in Evanston, as was the case in Aurora, what may be thought to be a strange superiority of artificially over naturally fluoridated water as a means of reducing dental caries attack rates would have appeared to have been even more marked.

“Weighting” of results. The method of combining the results of the six, seven and eight-year-old children into one category introduces an important source of error when comparisons are made between the results obtained in the control city and in the test one, or between those found on different occasions in Evanston. Owing to the great differences in caries attack rates which are observed between children of these ages (the baseline DMF rates for these three ages in Evanston were 46.85, 153.49, and 249.93, according to Hill et al., 1950), the results may inadvertently be “weighted” by including a preponderance of young or of old children in the age group six to eight years. If this occurs, the average value will be lower or higher than it would have been if the three ages had been equally represented in the sample. In comparing the results of the control and the test cities, “weighting” of this nature could make it appear that large differences were present, when, in fact, they were either slight or absent, or the presence of actual differences could be hidden.

An example of “weighting”. The results of the pre-fluoridation, and of the first post-fluoridation survey at Evanston (Hill et al., 1950), clearly demonstrate the process of “weighting” and show that its occurrence is not merely a theoretical possibility. On these two occasions, the number of children in each of the age groups six, seven and eight years that were examined in 1946 was 461, 759 and 771 respectively; the corresponding numbers seen in 1948 were 756, 838 and 440. On both occasions the results of the three ages were combined, and a caries rate was computed for the age range six to eight years.

Significant tests and ‘weighting’. Despite the rather obvious “weighting” in the examples which have just been cited, tests were applied to determine the significance of the difference between the caries attack rates found during
the two examinations in the combined age range six to eight years. In regard to the permanent teeth, it was stated that “The probability of this difference being due to chance is 0.0000” (Hill et al., 1950). Curiously, in those teeth a decrease in the caries rate was reported, contrasting with the statement of a significant rise in the rate of the deciduous ones.

**Random variation ignored.** Hill et al. (1950) stated: “It is to be expected that the rate of caries in all teeth varies from year to year due to chance. A significant reduction of caries prevalence can therefore be assumed to exist only when the statistical analysis of the data provides almost absolute certainty that the observed differences are not due to chance.” However, in a subsequent paper (Hill et al., 1956) these authors ignored the variations in the intervening years, even when these were as marked as those in Table 5 of that report, and stated: “Difference between 1946 and 1955 rates is statistically significant.”

**Original results altered.** In the X Report (Hill et al., 1952), and in all the later ones, alterations were made to the rates shown for the years 1946 and 1948 in children of the combined age group six to eight years, which were published by Hill et al. in 1950 (Tables I to VI). The original rates were replaced by values which are the means of the mean rates for the children of each of the three ages six, seven and eight years (Hill et al., 1952, Tables 11 to IX).

**System of computation changed.** The change in the system of computation was explained by Hill et al. (1952) in these terms: “The group averages, shown in previous reports, represents weighted averages of the individual mean caries rates. Inasmuch as the composition of the groups of children with respect to the number of 6, 7 and 8-year-olds varies from year to year, it was felt that unweighted group averages form a more sound basis for comparison of group caries rates between years.”

**The new method of computation.** In 1952 Hill et al. stated that “The new averages were obtained by taking a simple arithmetical mean of the individual caries rates of the 6, 7 and 8-year-old children.” This description of the new method is apt to cause some confusion, for it is considered to describe accurately the old method. It was used by these authors in 1950, and then abandoned by them in favour of the new one. The results for 1950 and 1951 in Table IV of Hill et al. (1952), and those for 1953 in Table I of
Hill et al. (1957a), and for 1955 in Table I of Hill et al. (1956) make it clear that in this new method of calculation, the rate per 100 children aged six to eight for each examination was obtained by taking a simple arithmetical mean of the rate for each of the three ages six, seven and eight years.

Errors in amended rates. The amended rates published by the authors (Hill et al., 1952) for the age group six to eight years need further amendment, and the difference between them is even less than that stated. The mean of the three values shown for 1948 in their Table IV, 23.54, 103.58 and 194.09, is found to be 107.07, not 92.07 as stated; also, the mean of the three values for 1946 - 46.85, 153.49 and 249.93 is 150.09 not 149.76. These errors were repeated in the XV and the XVI Reports (Hill et al., 1957a, 1956).

The figure 149.76 was shown also in the XIV Report (Hill et al., 1954). In that report the rate for age six to eight years was said to be “65.82 in 1953.” However, in Table I of the XVI Report (Hill et al., 1956) the rate for 1953 for age six to eight years was given as 63.52. The latter figure is the mean of the three mean rates shown for the six, the seven and the eight year-old children.

The XIV Report (Hill et al., 1954) stated: “The combined 6 to 8-year-old children had a permanent tooth DMF rate of 149.76 per 100 children in 1946 and 65.82 in 1953. This is a difference of 60.38 per cent.” In fact, by using their standard method of calculation, the “difference” is 56.05 per cent.

A confusing calculation. The situation is made even more confusing by the figures shown in Table 6 of the XVI Report (Hill et al., 1956). If the method commonly used in these trials is employed, when the difference between the DMF rates for 1946 and 1955, which is 95.90 (the rates being 149.76 and 53.86), is expressed as a percentage of 149.76, the “per cent difference” is 64.04, not 64.11 as shown. However, if the correct figure of 150.09 (which does not appear to have been mentioned in these reports) is substituted for 149.76, the “per cent difference” becomes 64.11 as shown in their Table 6.

Was sampling used? The six, seven, eight and twelve, thirteen, fourteen year age groups were chosen for study (Blayney and Tucker, 1948), but it was not stated whether all children of these ages (the ages were taken to the nearest birthday) were examined, or whether a sampling method was used. The VII Report of Hill et al. (1951) said that “0. 1 per cent of the children in
the control area were Negro.” However, in the XV Report (Hill et al., 1957a) it was stated that “the control area (Oak Park) examinations included only public school white children”. It is not clear whether the Negro children in that city were excluded from the examination by design, or by the chance of a sampling method. The former alternative is suggested by the statement of Hill et al. (1955) that “In the control village of Oak Park, only public school children were studied”.

Were children “continuous residents”? It is not clear whether all the children included in the early reports (Blayney and Tucker, 1948; Hill et al., 1950, 1951, 1952, 1953, 1954) were “continuous residents”. Although the questionnaires recorded the residence record of each child, it was not until the X1 Report (Hill et al., 1955) that the statement was made that “The data given in this report are limited to those children whose entire lives have been on Lake Michigan water.” The United Kingdom Mission (1953) stated that “The study includes only white children attending public schools in the city who have lived in the area continuously from birth.” However, as the first part of that statement presents an incomplete description of the authors’ method, doubt is raised as to the accuracy of the statement made in regard to continuous residence.

Disturbing disagreements. In the following paragraphs are cited some disturbing disagreements between the statements made regarding the number of children examined. No suggestion has been found that more than one series of examinations was conducted in Evanston in each year from 1946 onwards, and in Oak Park in 1947 and 1956. Therefore, although the situation is uncertain regarding sampling and continuous residence, it would be expected that all the reports would agree with regard to the number of subjects of each age that were examined in each individual year. The exception is the XVII Report (Hill et al., 1957b), which compares the caries rates of white with those of Negro children; for it was stated that “in this report no attempt has been made to limit the examinations to continuous resident children.” Therefore, it would be expected that the sample sizes shown in this report may be larger than those published in other reports.

Gross discrepancies between sample sizes. The numbers of children of each of the ages twelve, thirteen and fourteen years that were examined in 1946, 1949, 1952 and 1954 were given in the second column of Tables XI
and XII of the XV Report (Hill et al., 1957a), the same figures appearing in both tables. It is to be noted that in eleven out of the twelve cases, the sample sizes given there are different from those shown in Tables 111, V, VI, VII, VIII, IX and X of the same report. In six cases the samples were larger in Tables XI and XII than in the other tables mentioned, and in five cases they were smaller. The largest discrepancy was between the number of children aged twelve years that were examined in 1949. Tables XI and XII showed this figure as 627, and the other tables gave 522 as the sample size. Similar discrepancies (for 1946, 1949 and 1952) are present between the sample sizes shown in Tables IX and X of the 1955 paper of these authors, and Tables 1, 111, IV, V, VI, VII and VIII of that report. The authors (Hill et al., 1957a) stated: “The number of teeth and surfaces associated with the DMF rates from 1946 through 1954 are shown in Tables XI and XII.” In other tables mentioned in that report the “Rate per hundred children” was employed, but there appears to be no reason why the number of children examined should not be the same for both of these comparisons. No explanation for the different sample sizes was advanced by the authors.

Disparities in Negro sample sizes. Marked disparities are seen between the sample sizes shown for Negro children, for, judging from Table 10 of the XVII Report (Hill et al., 1957b), data from only about half of the Negro children aged twelve to fourteen years who were examined in 1946, and of less than a third of those examined in 1954, were included in the XV Report (Hill et al., 1957a). The number studied is given in Table IV of the latter paper as 96 in 1946, and as 79 in 1954. However, the XVII Report (Hill et al., 1957b, Table 10), shows that 188 Negro children of those ages were examined in 1946, and 250 in 1954.

The XI Report (Hill et al., 1955) also shows that 96 Negro children were examined in 1946. The VII and XVIII Reports (Hill et al., 1951, 1958), although they do not state the number of Negro children, indicate the same sample size, 1,701 children, as the XI and XV Reports (Hill et al., 1955, 1957a). In the last mentioned report, referring to the 1954 results, the authors said: “It is admitted that the Foster (Negro) school sample (79) was limited.” Why, then, were so few of the 250 Negro children aged twelve to fourteen years that were examined in that year included in the report? Were less than a third of these children continuous residents?

The situation with regard to children aged six to eight years cannot be
investigated, because the XVII Report is the only one in which the data of the younger age group of Negro children are shown separately from those of the white children.

Further unexplained differences. The position revealed in the last paragraph is further confused by the presence of large variations between the number of white children, aged twelve to fourteen years, whose data were shown in earlier reports, and the number given in Report XVII. In the former reports (Hill et al., 1955, Table 11; 1957a, Table IV) the number of these children examined in 1946 (public plus parochial schools) is stated to be 1,605, but, according to the XVII Report (Hill et al., 1957b, Table 10) the number seen in that year was 1,368. In 1954 the examinations of white children totalled 1,247 (Hill et al., 1957a, Table IV), but the figure of 1,905 is shown in the XVII Report (Hill et al., 1957b).

In the younger children, as no dissection of the data into school groups has been published, only the total number inspected can be considered. The XVII Report (Table 10) states that 1,754 children were examined in 1946 and 2,952 in 1955; but Table I of the XVI Report (Hill et al., 1956) shows 1,991 and 1,376 examinations respectively. The two statements of sample sizes (XVII Report figures minus the XVI Report ones) therefore differ by -237 and +1,576 children.

It is possible that the larger sample sizes shown in the XVII Report for the examinations in 1954 and 1955 were due, despite the sizes of the increases (171 Negro and 658 white children aged twelve to fourteen years, and 1,576 children aged six to eight years), to the inclusion of all subjects, and not only those who were “continuous resident children”. If, at the time of commencement of the study in 1946, children who had not lived in Evanston “continuously” since birth were excluded from the main study, an explanation can be found for the larger number of Negro children included for that year in the XVII Report. However, it is strange that that report, which included children who were not “continuous residents” (Hill et al., 1957b), in 1946 should be based on 237 fewer white children aged twelve to fourteen years and on 237 fewer white plus Negro children aged six to eight years than were included for that year in the other reports mentioned.

Incompatible statements. The authors made incompatible statements regarding the total number of children examined during the initial
examinations in Evanston and Oak Park. In Report II (Blayney and Tucker, 1948) it was stated that the “baseline observations were made on 4,375 North Shore” (study area) “children and 2,493 Oak Park children.” These figures were repeated in 1950 by Hill et al. However, Tables I to VI of the latter paper show that 1,991 children aged six to eight years were examined in Evanston in 1946; Tables 1, 11 and III of Hill et al. (1951) indicate that 1,701 children aged twelve to fourteen years were examined in that year, that is, a total of 3,692 children. One or both of these figures (1,991 and 1,701) were repeated by the authors (or may be obtained by adding figures for individual yearly age groups) in 1952, 1955, 1956, 1957a and 1958.

The third total sample size for Evanston in 1946 is shown in the XVII Report (Hill et al., 1957b). By totalling the figures in Table 10, it appears that 1,754 children aged six to eight years, and 1,556 aged twelve to fourteen years, were examined, a total of 3,310 subjects. From Tables I and 2 of Hill et al. (1958) it is deduced that a total of >2,051 children were examined in Oak Park in 1947 (see figure 5, p. 167).

Therefore, three very different sample sizes were given for the 1946 examination in Evanston: 4,375, 3,692 and 3,310; and two total sample sizes of 2,493 and 2,051 subjects examined in Oak Park in 1947. The smallest sample size for Evanston (3,310) was given in the XVII Report, despite the statement of the authors (Hill et al., 1957b) that “in this report no attempt has been made to limit the examinations to continuous resident children.”

Remarkable changes in assessment of statistical significance. In the footnote to Table II in Hill et al. (1952) it was stated: “It should be noted that the caries rates per 100 children for the 6-8 year olds as a group shown in this report, vary slightly from those shown in previous reports.” Although these were said to be slight variations, the remarkable fact emerges that, although based on the same data, the difference between the 1946 and the 1948 caries attack rates for the deciduous teeth of children of that age range, which was said to be statistically significant (the probability being given as 0.005) in the 1950 Report, was stated by the same authors, in 1952, to be “not statistically significant.”

On reading the X Report (Hill et al., 1952), it appears that even more extraordinary changes of opinion with regard to the significance of results based on the same data occur in five comparisons between the rates of permanent teeth; significant differences (probability “0.0000”) being altered
to “not statistically significant.” However, a correction (J. dent. Res., 31, 597) stated that the footnotes to Tables IV, V, VI, VII and VIII were incorrect, and that the statements: “Differences are not statistically significant” should have read “Differences are statistically significant”. It is considered likely that the correction is incomplete, and that in the footnote to Table IX of that paper, the word “not” should be deleted. If this alteration is not made, that footnote indicates that the difference between the rates for 1946 and 1948 is “not statistically significant”, although two years earlier, the difference computed from the same data was stated in the footnote to Table VI of Hill et al. (1950) to be significant (probability “0.0000”).

At first sight, the employment of statistical terminology in the presentation of this study engenders confidence in the results reported, but the few examples which have been cited clearly indicate their unreliability.
THE BRANTFORD STUDIES

In the city of Brantford; Canada, two independent trials were conducted.

The City Health Department Study

In this study no control procedure was attempted. However, it will be considered briefly because two pre-fluoridation surveys were made by the school dental officer and his assistant (Hutton et al., 1954). This is the only one of these five trials in which more than one pre-fluoridation survey was made in the test city; and, with the exception of Muskegon, none of the control cities provided data obtained in successive years from individual yearly age groups.

Were results combined or averaged? Hutton et al. (1951) stated that “The results of these two [pre-fluoridation] surveys have been combined and are shown in Table I.” In Tables I and II of the Ontario Department of Health Report (1956) the rates for those two surveys were shown separately. With the exception of those of the nine-year-old children, for both the deciduous and the permanent teeth, the mean of the two rates for each age is identical (to one decimal place) with the mean rate computed from the figures of the combined survey which were supplied by the authors (Hutton et al., 1951, Table I). This result could have arisen only if (with the exception of the children who were nine years old) the number of children of the same age examined on both occasions was equal, or almost exactly so - a most unlikely event; or if the results were not combined, as stated by the authors, but the rates obtained in 1944 and 1945 were averaged. The United Kingdom Mission (1953) stated that “the average figures of these two years” were used. If the rates for the two years were averaged, there were errors in computing the rates of the nine-year-old children, or errata in one or more of those three tables. The figures shown in Tables I and II of the Ontario Department of Health Report (1956) should be treated with caution, because in both of these the year of fluoridation is stated incorrectly, and in the former table the “% Reduction Since 1944-45” for age seven years should be 66, not 51; whereas in the latter one, the “% Reduction Since 1948” for age nine to eleven years in Stratford and Sarnia should not have been indicated by dashes, but by five and sixteen respectively.
The National Health and Welfare Study

The other study in Brantford was conducted by the Canadian Department of National Health and Welfare, and was described by the New Zealand Commission of Inquiry (1957) as “the most complete of the 10-year North American studies”.

Late commencement. Unfortunately, this trial was not begun until January 1948, over two and a half years after the commencement of fluoridation of the Brantford water supply (Brown, 1951). Such delay must affect the value of this study, unless it is assumed that the structural theory of reduction of dental caries as a result of the ingestion of fluorides is correct, and that this is the only way in which fluorides may affect the incidence of caries. This theory was advanced by Cox and Levin in 1942, and was widely accepted at the time these trials were initiated (Dean et al., 1950; Ast et al., 1950). If this theory is correct (as was noted in Part One), little change can be expected in the DMF rates until about six years after the commencement of the study. It is evident that this theory must still be held to be correct in some quarters, for the recent report of a dental caries survey conducted by McCauley and Frazier (1957) stated: “Although fluoridation of the Baltimore City water supply was begun Nov. 26, 1952, (27 months before the survey), there was no reason to anticipate substantial change in the caries experience of these children in this relatively short period of time.” However, even before the commencement of the Department of National Health and Welfare study in Brantford, the City Health Department examiner’s figures for 1947 showed great reductions in the DMF rates since the introduction of fluoridation. This result was not published by Hutton et al. until 1951, but must have been available to the investigators who “came to the scientific rescue of the project early in 1948” (Hutton et al., 1956). Indeed, in his first report, Brown (1951) acknowledged the help and advice of two of the three authors of the City Health Department Report (Hutton et al., 1951).

The control cities. The city of Sarnia was chosen as the “fluoride-free” control, and Stratford as the control city with a water supply which “contains 1.3 ppm. of fluorine from a natural source” (Brown, 1951). The reasons for the selection of these cities were not given, except that it was said: “sarnia and Stratford, two cities in Western Ontario known to be comparable to Brantford, except for the fluoride content of their water supplies, agreed to serve as controls” (Brown et al., 1954b). Also, Brown, Josie and Stewart
(1953) said that Sarnia was “a city” which has fluoride-free water and is sufficiently similar in size, location, and other attributes for purposes of the comparison”. The United Kingdom Mission (1953) stated: “Before this study was undertaken the socio-economic status of the three communities was examined and found to be reasonably comparable.”

Superior dental care in Brantford. The United Kingdom Mission (1953) said: “Brantford, however, over a period of 15 years, has provided more free dental services for children than most Canadian cities, and this has resulted in the ratio of corrected to total defects being higher than in either Sarnia or Stratford.” It considered that in Brantford “dental care was outstandingly good.” Also, Brown, in 1952, said:

“the recordings so far obtained indicate both a higher treatment and an apparently better oral hygiene status of the Brantford children when compared with the controls, and it is therefore suggested that caution should be exercised in the interpretation of the rates shown. The lack of a pre-fluoridation survey on a comparable basis is a further limiting factor in interpreting the results.”

No pre-fluoridation survey. The authors of this Brantford study (Brown et al., 1953) said:

“As the study does not include a pre-fluoridation survey, the full amount of benefit which the Brantford teeth have received since fluoridation cannot be illustrated directly from the data for Brantford. Some idea of the extent of the benefit can be obtained by comparison with the data for Sarnia.... By 1948 the Brantford data were not greatly different from those for Sarnia.”

This remark suggests that the data for the two cities prior to fluoridation in Brantford were similar, and that this process had had little effect on the caries rates up to the time of the 1948 examination in Brantford.

Doubtful comparability of rates. Owing to the delay in setting up this study, it cannot be established how closely the dental caries attack rates in Brantford resembled those in Sarnia, at the time fluoridation was instituted in the former city. There is evidence that the dental condition of the children in those two cities was not closely comparable, for Brown et al. (1953) stated that “even by the time of the first survey, mean tooth mortality in Brantford was much lower than in Sarnia, for all age groups.” This comment implies that, even by the time of the first survey, as a result of fluoridation
the tooth mortality in Brantford had decreased considerably. This concept is not consistent with the one mentioned in the last paragraph. At the time of the first examinations, the tooth mortality in the six to eight years age group was more than four times as great in Sarnia as it was in Brantford, and in each of the other two age groups it was almost twice as great (Brown et al., 1953, Table 3.)

Figure 6. The gross differences observed in the tooth mortality (teeth which are missing or which must be extracted) in Brantford and its control city of Sarnia, during the initial examinations. Canadian Department of Health and Welfare study.

The influence of treatment. The fact that such large differences were reported in tooth mortality rates in the two cities even in the older age groups suggests that dental treatment in them was different, and the authors stated that “Both preventive and treatment measures may have a decided effect on tooth mortality rates” (Brown et al., 1953). It may be recalled that the United Kingdom Mission (1953) noted that Brantford was unusually well provided with free dental services “and this has resulted in the ratio of
corrected to total defects being higher than in either Sarnia or Stratford.”

**Tooth mortality.** Brown et al. (1953) said that “there has been a decrease in tooth mortality in Brantford between successive surveys”; but, in fact, the 1953 rate (Table 3) in the children aged six to eight years was the highest up to that time. This statement was corrected in the next report (Brown et al., 1954b) by prefixing “in almost all cases” to the previous statement. In this connection, the authors remarked in 1953 that, “as well as the fluoridation of the Brantford-water supply, other factors such as differences in preventive or treatment measures are probably affecting the Brantford position.” There appears to be no reason why those of the control cities should not have been similarly affected.

**Differences in oral hygiene.** Additional evidence which suggests that a difference existed between the dental condition of the children in Brantford and that of children in the control cities is provided by the data with regard to oral hygiene. Brown et al. (1954b) stated:

> “Classification and recording of oral hygiene was undertaken because it was considered that marked differences in oral hygiene as between the test and control groups might conceivably affect the findings—or at least might be taken into consideration as a modifying factor, although not a strictly measurable one. However, the figures here suggest that, since 1948, differences in oral hygiene status could not have been a major factor in either the caries level changes within Brantford or the caries level differences between the control cities.”

As no comparisons were made between the control cities, the last phrase of the quotation is thought to refer to the caries level differences between Brantford and each of the control cities. The authors’ Table 11 indicates that, in the first examinations, in the test city the percentage of subjects who had a good oral hygiene status was almost twice as great as that present in children in both the control ones; these were, Brantford 34.3 per cent, Sarnia 19.7 per cent and Stratford 17.8 per cent. Considerable differences between the oral hygiene status of the children in the test and the control cities were also recorded during the later examinations. These were clearly “marked differences” though the authors did not consider them important.
The concentration of fluorides. Brown et al. (1954b) stated:

“The Brantford Fluoridation Caries Study was undertaken with a view to finding out whether or not the raising of the fluoride content of a previously fluoride-free water supply to 1 part per million, by the mechanical addition of sodium fluoride, would reduce the incidence of dental caries to that which obtains where water supplies derive about 1 part per million of fluoride from deposits in the earth.”

A fundamental requirement of a test of this nature is that the water supply of the control city should contain the same concentration of fluorides as that of the test one, but Brown, McLaren, Josie and Stewart (1956) reported: “The Stratford water supply contains a concentration of fluoride which is 60% higher than that used in Brantford.”

Differences of opinion. Several different statements were made regarding the concentrations of fluorides which were present in the water supplies of Brantford and Stratford.

1. Brantford. The New Zealand Commission of Inquiry (1957) said that the water supply of Brantford was “raised to 1.2 ppm. in 1945”. The authors of the City Health Department study (Hutton et al., 1951) stated that in February 1949, “the dosage was raised to produce 1.20 ppm.”; but in 1954 they stated that “The fluoride content of the finished water is maintained at 1 ppm.” In reporting the National Health and Welfare Study, Brown (1952) stated that “a fluorine concentration of between 1.0 and 1.2 ppm. has been maintained in the water supply continuously” since June 1945; and in 1956 Brown et al. said: “Brantford has had more than 10 years of experience with 1 part per million fluoride in its water supply.” These statements that the fluoride content was “maintained” at “1 ppm” and “between 1.0 and 1.2 ppm” should be considered in conjunction with that of the United Kingdom Mission (1953): “For example, in 1951 the average for the year was 1.2 ppm with a variation between 0.75 and 1.45 ppm., however, the figures below 1.1 ppm and above 1.3 ppm were few in number.”

2. Stratford. The supply of Stratford was stated to have 1.2 ppm of natural fluoride in its domestic water (Ontario Department of Health, 1956; New Zealand Commission of Inquiry, 1957). However, in reporting the National Health and Welfare study in 1951 and 1952, Brown stated that it “contains 1.3 ppm. of fluorine”; and, with his co-workers, the following year said
that "in Stratford where the water supply, obtained from deep wells, has contained 1.3 ppm." (fluoride) "from natural deposits continuously since 1917" (Brown et al., 1953). In their next report dealing with dental caries (Brown et al., 1954b), the figure stated was "1.3 to 1.6 parts per million of fluoride"; but in the following report these authors (Brown et al., 1956) said that this water supply "contained 1.6 parts per million of fluoride since 1917", and also that 1.6 ppm fluoride content "has been in continuous use for thirty eight years."

These different statements, although strange, may be considered to be unimportant from the practical point of view, but very small changes in the fluoride content of the water may have considerable effects, as reports by Deatherage (1942) and Galagan and Lamson (1953) indicated. The latter authors found that “In water supplies of the Arizona communities studied, concentrations of fluoride above 0.8 ppm resulted in objectionable dental fluorosis; concentrations of 0.6 to 0.8 ppm resulted in an occasional diagnosis of fluorosis; concentrations below 0.6 ppm did not cause objectionable fluorosis.” The mean temperatures of these communities were between 67 and 72 degrees Fahrenheit.

Three misleading statements. In the summary of the 1954b report of the National Health and Welfare study, Brown et al. stated that during the period 1948-54 “dental caries experience of children in the two control cities, on the other hand, either has remained at about the 1948 levels, or has increased slightly, at all ages studied.” A similar statement was made by them in the 1955 Report (Brown et al., 1956): “During that time [more than ten years] a very important, statistically significant reduction in tooth decay has occurred in all the age groups studied, while in the two control cities of Sarnia and Stratford it has either remained at about the same level or increased somewhat.” The last sentence contains three misleading statements about the control cities:

(a) As this study did not commence until 1948 with “examinations in Sarnia beginning in March of that year, and in Stratford in October” (Brown, 1952), and as fluoridation in Brantford commenced in June 1945 (Hutton et al., 1951), no information is available with regard to the prevalence of “tooth decay” in Sarnia during the first two and three-quarter years, or in Stratford for the first three and a quarter years of the ten-year period of fluoridation which these authors were discussing in their 1955 Report. Therefore, it cannot be known whether this condition
“remained at about the same level” in the control cities during the early years of fluoridation in Brantford.

(b) No remarks were made in the context of this statement in this (or the previous) paper which suggested that reference was being made to the permanent teeth only, but these statements are not correct for the deciduous teeth. (However, in the former paper, a similar statement to that made in the summary was also made under the heading “Mean DMF Permanent Teeth”.) Decreases in the df (decayed, filled deciduous teeth) rate were seen in the nine to eleven years group in both Sarnia and Stratford, that in the former city being shown by these authors to be statistically significant (Brown et al., 1954b, Table 10).

(c) In both cities the mean rates of DMF and df teeth per child showed changes which were said to be statistically significant (Brown et al., 1954b).

Omission of decreases. The decreases in the caries attack rates of the deciduous teeth, which have been mentioned above, were also omitted in Table II of the Report to the Minister of Health, Province of Ontario, Canada, by the Division of Medical Statistics, Ontario Department of Health, which was made in 1955 (Ontario Department of Health, 1956). Under the heading “% Reduction Since 1948”, these decreases were not shown, but instead, in the appropriate positions dashes were printed, despite the fact that in Sarnia the percentage reduction (determined by the method commonly used in these studies) was 16 per cent, almost as great as that of 18 per cent shown for the same age group in the test city; furthermore, this reduction in Sarnia was stated by the authors (Brown et al., 1954b) to be statistically significant.

Different rates reported. It should be noted that the deficiency in the data of the National Health and Welfare study, owing to its late commencement, could not be decreased by comparing the rates obtained by its examiners with those reported by the City Health Department examiner, because of the considerably lower rates recorded by the last-mentioned examiner when inspecting similar groups of children. For instance, in their examination in 1948, for children aged six to eight, nine to eleven and twelve to fourteen years, Brown et al. (1953) obtained rates of 1.41, 4.07 and 7.68 respectively for the permanent teeth, compared with rates of 0.84, 3.37 and 6.11, for the same age groups of children, in the same city and in the same year, obtained by the City Health Department examiner (calculated from Table IV, Hutton et al., 1951).
Significant fluctuations in controls. In the two control cities “where it is presumed that there has been no appreciable change in either preventive or treatment services” (United Kingdom Mission Report; 1953), it can be seen in Tables 4, 6, 8 and 10 published by Brown et al. (1954b) that some considerable fluctuations in the caries attack rates were recorded; more than half of the inter-year differences in each of the control cities being shown to be statistically significant. However, in the text it was stated that the “dental caries experience of children in the two control cities . . . either has remained at about the 1948 levels, or has increased slightly, at all ages studied.”

(1) Sarnia. In this city the changes between examinations of the rates for the deciduous teeth were not very marked, but there was a significant one between 1948 and 1954 in the nine to eleven years age group. However, in the DMF permanent teeth, there were four definitely significant (three standard error level) and one significant change in the nine comparisons made. In regard to the first permanent molars, there were six significant (including three definitely significant) alterations in the rates, in the nine comparisons made (Brown et al., 1954b).

(2) Stratford. In this city, the rate of df teeth per child showed a significant difference in one case out of the four comparisons made between successive examinations (Brown et al., 1954b). In the DMF permanent teeth per child, the results of the four examinations were: 0.41, 0.75, 0.47 and 0.67 for the six to eight years group; 1.13, 1.76, 1.46 and 1.89 for the nine to eleven years age group; 2.55, 3.12, 3.02 and 3.77 for the twelve to fourteen years age group (Brown et al., 1954b, 1956). These variations between examinations were so large that five out of the six comparisons made (in the 1954b report) between successive examinations were said to be statistically significant, four of them being at the three standard error level. In the last report published (1956), Brown et al. abandoned the method which they had used in the two previous ones, that of showing the standard error of the mean values of the DMF rates, and of making “Inter-City” and “Inter-Year” comparisons (Brown et al., 1953, 1954b). Therefore it was not stated whether the differences between the 1954 and the 1955 DMF rates in Stratford were significant, but it can be seen that they were marked; the difference of 0.75 in the twelve to fourteen years group being considerably larger than any of those stated in the 1954b report to be significant differences between various examinations in that city. When the DMF rates for the first permanent
molars are considered, similar marked changes are seen, and in five of the six comparisons the differences were significant (four definitely so).

**Larger “percentage” changes in a control.** If one resorts to the method commonly used in these trials - that of expressing the alteration in the DMF rate as a percentage of the original rate - these unexplained increases in the control city of Stratford between 1948 and 1955, although they were described as “no change” (Ontario Department of Health, 1956), and as “a slightly higher prevalence of dental caries in 1955, over the 1948 levels” (Brown et al., 1956), are found to be 63 per cent, 67 per cent and 48 per cent, for the six to eight, nine to eleven and twelve to fourteen years age groups respectively. In each case these percentage changes are considerably larger than those of 51 per cent, 44 per cent and 37 per cent which can be computed from the data reported for Brantford. The last-mentioned changes were attributed to fluoridation, and each was stated to indicate “a very important, statistically significant reduction in tooth decay” (Brown et al., 1956).

This is just one instance of the strange results which are obtained when this method of calculation is used. It should be realized that it was the one most commonly employed in fluoridation trials, and was used in formulating the often-expressed claim that (as stated by Arnold et al., 1956): “In children born since fluoridation was put into effect, the caries rate for the permanent teeth was reduced on the average by about 60 per cent.” The recent World Health Organization Press Release (WHO/45, 4 September 1957) stated - with no mention of age - “The prevalence of dental caries in the permanent teeth of children decreased some 60 percent”.

**A smaller “percentage decrease” after long fluoridation.** The “percentage decreases” which have just been mentioned (51 per cent, 44 per cent and 37 per cent, calculated by the method described in the last paragraph) were not stated in the 1956 report of Brown et al., but the figure of 51 per cent for the six to eight years age group is considerably less impressive than the figure of “approximately 69%” published in the 1954b report from this study. Although the final report (1956) gave the rates for 1948 and 1955 only, and therefore did not show the fluctuations between examinations, from the 1954b and 1956 reports of Brown et al. it is seen that the marked change in the “percentage” decrease which has just been mentioned was due to the DMF rate in Brantford in 1955, for this age group, being the highest seen since 1951. Ignoring the fact that in “children born subsequent
to fluoridation” the “decrease” in the DMF rate had dropped to only 51 per cent, the authors stated in the final sentence of their final report (1956): “For every three decayed teeth they would have had, they have only one.”

More misleading comments. Turning from the reports made by the authors of this study about the control cities to some of the comments made by others, it is seen that these are even more misleading. Only two will be mentioned. Martin (1956) stated that during “the 1948-54 period” the “DMF figures for the two control areas have remained at 1948 levels.” The authors of the Ontario Department of Health Report (1956) went so far as to state to their Minister of Health that “it had been established that there has been no change in the already low dental caries attack rates in Stratford ... or in the relatively high rates for Sarnia”.

These two statements are contrary to the results published by the authors of the study (Brown et al., 1954b), which showed that in both the control cities there were statistically significant differences between the caries attack rates at successive examinations. Out of the fifteen comparisons made, only five differences in the rates were not significant, two changes were significant and eight changes were definitely significant.

Unexplained significant changes in controls. All the changes in the caries attack rates in the control cities which were reported to be significant are unlikely to be chance variations; therefore, to what factor or factors must they be attributed. It is possible that they were due, in whole or in part, to alterations in the “weighting”, such as were found in the Evanston study as a result of combining the caries attack rates of children of different ages (Hill et al., 1952). However, as the age composition of the groups was not stated in this study, it cannot be determined to what degree the data was distorted by “weighting”, a condition which is almost inevitably present when data drawn from several different yearly age groups are combined.

Apart from deficiencies which are found in other studies also, in this trial there is an absence of any information regarding the caries attack rates in Brantford and Sarnia, prior to the fluoridation of the water supply of the former city. There is also the fact that no explanation was given by the authors for the significant variations in the caries rates in the control areas. Therefore, a marked decrease in dental caries in the test city as a result of fluoridation cannot be said to have been established.
THE NEWBURGH STUDY

The fluoridation trial conducted in Newburgh differs from the other studies in two important ways:

1. In almost all the comparisons made, the data obtained were compared with those from Kingston, the “fluoride-free” control city, instead of the method used in the other trials, by which most comparisons were made between the initial and the latest observations in the test city.

2. The caries attack rates were stated per 100 erupted teeth, instead of per 100 children or per child. The Evanston study was the only other one in which the caries rate per 100 erupted teeth was published; Hill et al. in 1955 and 1957a showed this rate, but only for children aged twelve to fourteen years.

The control city. Kingston was used as the control area. “Both cities are situated on the Hudson River about 30 miles apart. Each has a population of approximately 30,000. The climate of both cities is also similar, and their water supplies at the outset of this study were comparable and have remained so, except for the addition of sodium fluoride to Newburgh’s supply” (Ast et al., 1950). Ast and Chase (1953) added the information that the two cities had a “comparable age, sex, and color distribution”; and Schlesinger, Overton and Chase (1950) mentioned that they “bore a close resemblance to each other in respect to size and socio-economic conditions”.

Late examination of control city. In Kingston, as in the other “fluoride-free” control cities that have been considered, the basic examinations were not made until after the fluoridation of the water supply of the test city. Fluoridation was started in Newburgh on 2 May 1945 (Ast et al., 1950), but the examinations in Kingston were not conducted until “Sept., 1945 - Feb., 1946” (Ast et al, 1950).

Considerably different composition of waters. In 1950 Ast et al., stated that the water supplies of Newburgh and Kingston “at the outset of this study were comparable and have remained so, except for the addition of sodium fluoride to Newburgh’s supply.” However, both the source and the composition of the water supplies of these two cities are different. The United Kingdom Mission (1953) stated that the source of Newburgh’s water is from “surface water. Algae growths in spring and summer checked by copper sulphate blown on the surface of the water as a powder.” The source of Kingston’s supply was described as “Mountain spring impounded.
Auxiliary supply, small spring reservoir” (Lohr and Love, 1954).

In regard to the composition and other characteristics of these waters, according to analyses of the finished waters made in February 1952 by the U.S. Geological Survey (Lohr and Love, 1954), in each of the ten items - magnesium, sodium, potassium, bicarbonate, sulphate, chloride, dissolved solids, specific conductance, hardness and alkalinity - the values for the Newburgh water were at least four times as great as those obtained from analysis of the Kingston supply. In the very important matter of the calcium content, the Newburgh value of 35 ppm (Ca) was more than five times as large as that of the Kingston one of 6.6 ppm (Ca). Changes in the supplies during the period of the trial, owing to natural or to treatment-chemical variations, are unlikely to have affected these gross differences more than slightly.

![Figure 7](image.png)

Figure 7. The considerably different calcium and magnesium content and hardness of the water supplies of Newburgh and its control city of Kingston, February 1952. Eight other characteristics of the Newburgh water were at least four times as large as they were in Kingston. The authors of this study stated that these waters “at the outset of this study were comparable and have remained so” (Ast et al., 1950).

An unsatisfactory control. In proposing this study, Ast (1943) said: “Much care must be exercised in the selection of study areas which should be comparable in as many essential factors as possible.” The first of these factors which he mentioned was the “chemical composition of past and present water supply”. Therefore it is surprising that Kingston was selected as the control city for Newburgh, for it is clear that in this very important matter the two cities showed considerably different values. The importance of the close comparability of the water supplies was emphasized by the statement
of the American Water Works Association (1949) that the experimental verification of the fluoride-dental caries hypothesis “obviously necessitates the use of a nearby “control” city with a water supply comparable in all respects to that to which fluoride is being added.”

Variations in methods used. An outstanding characteristic of this study is the variation in the methods used, both in gathering the data and in the presentation of the results. There were changes in the examiners; on some occasions clinical examinations only were made and on others X-rays were also used. The statisticians changed, as did their presentation of the data in age groups. The sampling method varied in regard to residence qualifications, and changes occurred in the age range of the children who were examined. In one report data was obtained from selected schools only. In some examinations the sampling method was different in the control city from that used in the test one, All these matters will now be considered more fully.

The dental findings. These were published in five papers. Ast, Smith, Wachs and Cantwell, in 1956, said: “Progress reports were published after three, four, six and eight years of fluoride experience in Newburgh” (Ast et al., 1950, 1951; Ast and Chase, 1953; Ast et al., 1955). The last-mentioned report (Ast et al., 1955) “after eight years of fluoride experience” gave the results obtained during the examinations of 1953-4. The final report, giving the results for 1954-5, apparently one year later than those in the fourth dental report, was said to show the “dental findings after ten years of fluoride experience” (Ast et al., 1956). However, as fluoridation in Newburgh commenced on “May 2, 1945”, and as the examinations given in the final report were made “between October 1954 and June 1955” (Ast et al., 1956), it would appear that, at the most, only a small part of the data of the final examination was obtained “after ten years of fluoride experience.”

Different examiners used. The initial examinations in both cities were made by Finn. “The subsequent examinations in Kingston using the same technic were made by two dental hygienists” (Ast et al., 1950). The examinations in 1951-2 were conducted by two examiners, but “Due to loss of one of the examiners during the examination year, it was deemed advisable to use only those examinations made by the remaining examiner in both cities” (Ast and Chase, 1953). The clinical examinations in 1953-4, and the final ones,
were made by Wachs (Ast et al., 1955, 1956). These changes were made
despite the fact that in 1943 Ast said that “the examinations throughout the
study should be made by the same dentist because of the marked variation in
diagnosis of small carious lesions, pits, and fissures by different dentists.”

The clinical examinations were supplemented by the use of X-rays in the
years 1949-50, 1953-4 and 1954-5 (Ast et al., 1956). In the first of these,
which was confined to children aged seven, nine and eleven years, the
X-rays were taken by a staff dentist and were read by Ast and Finn (Ast et
al., 1951). The next series was taken by Wachs and was read by Bushel (Ast
et al., 1955); the final X-rays were taken by Wachs and a staff hygienist, and
they were read by Wachs and Smith (Ast et al., 1956).

Non-comparability of data. In the last two reports (Ast et al., 1955, 1956),
the carious cavities that were detected by the X-ray were added to those
found in the clinical examinations. Ast et al. in 1955 said that “the data
in this report cannot be compared directly to those earlier data based on
clinical examinations alone.” However, in Table 3 of the 1956 report, the
results of the clinical examination are shown separately, but a satisfactory
comparison with those obtained in the earlier years is prevented by the fact
that in this report the data were not published for yearly age groups, but for
the age ranges six to nine and ten to twelve years. Data for the other two age
groups which were shown in the final report, thirteen to fourteen and sixteen
years, were not published in the previous ones.

The rates for the deciduous teeth were given in only one report (Ast et al.,
1951).

Examiner variability. The between and within-examiner variability was
not investigated, although, early in the study, the importance of this matter
was recognized by Ast et al. (1950) when they stated: “We cannot entirely
rule out the possibility of variation in the interpretations of the examiners.
The fact that more than one examiner was used might alter the differences
between Newburgh and Kingston to some extent.” In the following year
(Ast et al., 1951) it was stated: “In the present report an attempt is made
to demonstrate that through an objective roentgenographic examination of
the teeth of selected age groups, the question of examiner bias in this study
is not likely to account for the differences noted.” However, the only data
published were those of the first permanent molars; and the finding that
“the DMF roentgenographic findings of the first permanent molars only”
in selected age groups shows “consistent differences at each age in favor
of Newburgh” does not provide an estimate of examiner variability such as could have been obtained readily by normal statistical methods.

In addition to the changes in the examiners and in the examination methods, there were changes in the statisticians. The report after three years of fluoridation was made in collaboration with one statistician; those after four, six and eight years with a different one; and a third statistician was employed in the preparation of the final report.

**Different adjustment procedures.** In most of the tables in this study a “Crude rate” and an “Adjusted rate” are shown. The incongruity of making these small adjustments to rates that were obtained by combining data from children of considerably different ages does not appear to have been realized. In some cases even data from children aged between six and twelve years were added (Ast et al., 1950, 1951; Ast and Chase, 1953), the great increase in the caries attack rate between those ages being ignored. The adjustments were made (depending on the type of data) to the tooth population, the first permanent molar population, or the distribution of children. In the first three reports of dental findings (Ast et al., 1950, 1951; Ast and Chase, 1953), they were all made to the appropriate situation in Kingston during the 1955-6 examinations, but the adjustment system was then changed, the crude rates after eight years of fluoridation being adjusted to the situation in Kingston in 1953-4 (Ast et al., 1955), and those shown in the final report to that present in the control city in 1954-5 (Ast et al., 1956).

**Variations in age groups.** In discussing the Evanston study, it has already been pointed out that the method of combining the results of different age groups may result in “weighting” the data, so that comparisons between the test and the control cities may be affected. In the examples given from other fluoridation trials in which this method was used, the age groups were consistent from examination to examination; but in the Newburgh-Kingston study the groups varied between examinations, between comparisons made from data obtained during the same examinations, and even the age range of the subjects inspected varied from time to time. In regard to the DMF rate per 100 erupted permanent teeth, the groups were as follows: 6-7, 8-9, 10-12 (Ast et al. 1950); 6, 7, 8, 9, 10, 11 and 12 (Ast et al., 1951; Ast and Chase, 1953); 6, 7, 8, 9 and 10 (Ast et al., 1955); and 6-9, 10-12, 13-14 and 16 years (Ast et al., 1956).
Changes in the age groups were also made in reporting the other data presented in this study, but in many cases the groups were different from those which have just been mentioned.

*Grouping of data hinders comparisons.* In the final report, Ast et al. (1956) said: “The data are combined for six to nine year old children because these children in Newburgh had used fluoridated water throughout their lives”; and the age groups ten to twelve years and thirteen to fourteen years were associated with the tooth calcification pattern. No explanation has been found for the grouping used by Ast et al. in 1950, but this matter will be considered later.

Whatever may have been the reason for adding the data of children of different ages, it has the unfortunate result of making it very difficult to compare the rates which were present in the test (and in the control) city at different stages of the trial, especially as, in the 1955 report of Ast et al., the rates obtained from the clinical examinations were not shown separately from those computed from the combined clinical and X-ray results.

*“Weighting”.* Even if the explanation advanced by the authors of this study is considered to be a reasonable one, there remains the danger of “weighting” the data by combining into one category such divergent material as is provided by children of different yearly ages. One of the tables in which obvious “weighting” is seen is Table I of the first report (Ast et al., 1950), “weighting” being present in several different forms. In the control city, the total DMF rate per 100 teeth (ages six to twelve years) is “weighted”; for the total number of teeth examined is made up (in 1945-6) of only 11 per cent from the six to seven years age group, with its comparatively low DMF rate, and of 67 per cent from the ten to twelve years group with its comparatively high rate (22 per cent was from age eight to nine years). In the latest examination shown in that table (1947-8), the two percentages were 17 and 59 respectively, so that the comparison between the results of the two examinations is also “weighted”. Similar instances of “weighting” are also seen in the data from the test city; but as these are of a different degree, the comparison between Newburgh and Kingston is another instance of “weighting” (Table 1, Ast et al., 1951). It can be seen that some “weighting” occurred within the age groups used in the baseline examinations, principally in the eight to nine years group in both cities.

*Fewer erupted teeth than expected.* In the final report (Ast et al., 1956), from Table I it can be calculated that the number of erupted permanent
teeth in the six to nine years group in Newburgh was less than the number expected, on the assumption that the mean age of eruption of each type of tooth was the same as in the children in Kingston. Also, in the ten to twelve years group (by assuming that in these children at least the eight incisors and the four first molars would have erupted) the number of erupted permanent canines, bicuspids and second molars was fewer in Newburgh than would be expected. Statistically speaking, both these differences are highly significant.

_Delayed eruption or “weighting”?_ These results could have arisen by there being a delay in the eruption of these teeth in the Newburgh children, for it is unlikely that the eruption rate altered in the Kingston subjects. However, Ast et al., (1951) said that “there does not seem to be any change in the eruption pattern among the children in Newburgh, the study city, as compared with those in Kingston, the control city.” No definition of an “erupted tooth” was given, but it is presumed that the authors of this study did not adopt the odd method used in Evanston, where “Only teeth which were 50 per cent or more erupted were considered. A carious or filled tooth was, of course, considered regardless of its stage of eruption” (Hill et al., 1955).

The conclusion of Ast et al. that has just been mentioned was reached only four years after the commencement of fluoridation, and as the teeth considered were partially formed prior to the commencement of that process, they would not show effects which the ingestion of fluoridated water may produce on the early stages of tooth development.

If delay in eruption did not occur in Newburgh, the lower number of permanent teeth present at those ages in that city compared with that present in Kingston was due to a “weighting” effect; there having been, in proportion, more young children in each of these age groups in Newburgh than there were in Kingston. If this is the case, as it is reasonable to assume that the mean DMF rates of the younger children were lower than those of the older ones, it would appear that in these age groups the contrast between the DMF rates in Newburgh and those in the control city was exaggerated in the final report.

_“Smoothing” of initial rates._ In 1951 Ast et al. reported that the “initial clinical examinations made in Newburgh and Kingston in 1944-1946 were made by one examiner, at which time the DMF rates were the same.” However, reference to Table 2 in that paper shows that the DMF rates per
100 erupted permanent teeth were, in Newburgh and Kingston respectively, at age six years, 8.5, 7.2; age seven years 11.7, 12.0; age eight years 17.1, 17.3; age nine years 21.2, 18.9; age ten years 21.9, 21.3; age eleven years 21.8, 21.8, and age twelve years 25.3, 25.4. Also, Table 5. which shows the DF rates per 100 deciduous teeth present, gives the rates in Newburgh and Kingston respectively as 27.2, 21.5 at age five years; 34.2, 32.1 at age six years; 42.3, 43.3 at age seven years, and 48.0, 47.2 for the eight-year-old children. Data for the DF rates of the deciduous teeth of older children were not provided.

In the first report of this study (Ast et al., 1950) no results were given for the deciduous teeth, and the results for the permanent ones were presented in three age groups, six to seven, eight to nine and ten to twelve years. It can be seen that by adding the data from children aged six years, in whom the DMF rate in Newburgh was higher than that in Kingston, to those of the seven-year-old children, in whom the reverse situation was present, the divergence between the rates prevalent in the two cities was reduced. In Table I (Ast et al., 1950) the combined rate was shown as 10.7 in Newburgh and 10.8 in Kingston. In a similar manner, the addition of the data for eight and nine-year old children and those of children who were ten, eleven and twelve years of age produced a levelling effect between the rates of the two cities in these two combined age groups. This process of combining data from children of different ages, when reporting the DMF rate per 100 erupted permanent teeth, although it was employed in only the first and the last dental reports, may have been used in order to simplify the presentation of the data; but it had the unfortunate effect of disguising differences between the DMF rates in the two cities at the time of the basic examinations. In the next report the situation was stated more accurately, Ast and Chase (1953) saying that “the DMF rates in both cities were approximately the same at the start of the study”.

Fluctuations in the control city. In this, as in other studies, it is found that the comments made in the text tend to underrate the changes that took place in the dental caries attack rates in the control city. In the summary of the paper by Ast et al. (1951) it was stated that “the DMF rates in the control city of Kingston show no changes.” In that paper, Table 2 shows the DMF rates per 100 erupted permanent teeth; in Kingston the “per cent change” in the rates of the four age categories six, seven, eight and nine years were 30.5,
7.5, 0.6 and 9.5 respectively. Small changes were shown for ages ten, eleven and twelve years. It should be noted that the six, seven and eight-year-old children all showed decreased rates between 1945-6 and 1949. No attempt was made to explain these decreases, and the water of Kingston “remained fluoride deficient throughout the study period” (Ast et al., 1956).

Fluctuations disguised. The method used by Ast et al. in 1951 was to compute the mean DMF rate per 100 teeth in all the children aged six to twelve years; the Kingston rate for this combined age group declining slightly from 20.2 to 19.9 between 1945-6 and 1949. However, by adjusting to the “permanent tooth population in Kingston 1945-6 examinations”, the authors showed that the rate of 19.9 became 20.2. On this basis it could be claimed that the “rate” in Kingston had not changed, but the incorrect statement was made that the DMF “rates” in the control city of Kingston showed no “changes”. These rates of 19.9 and 20.2 were produced by combining the data of young children - that had few erupted permanent teeth and relatively low DMF rates per 100 teeth with data of older children that had most of their teeth erupted, and considerably higher DMF rates per 100 teeth. The rate obtained in 1949 was then adjusted. This procedure, no doubt unintentionally, disguised the fluctuations in the rates in the control city.

In Table 2 (Ast et al., 1951) the “per cent change” in the Kingston children aged nine years was shown as 9.5, but if the figures 18.9 and 19.1 are the correct ones for the years 1945-46 and 1949, the “per cent change” should have been stated as 1.1, not 9.5.

Variability of caries rates. Unfortunately, the variability of even the mean caries rates cannot be studied, for the rates of yearly age groups were not published in the first and the last reports (Ast et al., 1950, 1956), and the only results shown in the 1955 report of Ast et al. were based on a combined clinical and X-ray examination.

The meagre data supplied for deciduous teeth. Data regarding the caries rates of the permanent teeth were shown in each report of this study; however, only very meagre data were published for the deciduous ones. None were made available in the first report (Ast et al., 1950). In the following year (Ast et al., 1951) the DF rates per 100 deciduous teeth were given, but only
for children aged five, six, seven and eight years, and in each age group the rates had decreased both in the test and in the control cities.

*Unexplained marked decreases in the control.* No explanation was given by Ast et al. (1951) for the decreases in the DF rates in Kingston, where the greatest relative decrease, from 32.1 per cent DF to 24.8 per cent DF, was seen in the teeth of the six year-old children. It would have been of great interest to see whether this trend was maintained in later years, but DF rates were not stated in the tables contained in any of the later reports. However, in the following one (Ast and Chase, 1953) the situation in regard to the deciduous teeth of children five, six, seven and eight years old (now termed “def teeth per 100 deciduous teeth present”) was depicted diagrammatically by means of a histogram, these unexplained decreases in the def rates in the control city being clearly seen, a small one at age five years, and considerable ones at the ages of six, seven and eight years.

*The increase in caries-free teeth in the control.* The only other information published regarding the deciduous teeth was expressed in terms of “Children with caries free deciduous cuspids, first and second molars”. This type of table appeared first in the 1951 report of Ast et al., and the results were given for only those children who were five or six years of age. In both age groups in Kingston the figures suggest an increase in these caries free teeth, the six-year-old children changing, between 1945-6 and 1949, from 17.2 per cent to 25.5 per cent free from caries. These changes were mentioned, but no attempt was made to explain them. In the next report (Ast and Chase, 1953) data for children aged seven years were also included. This report showed that, between 1945-6 and 1951-2, the percentage of children in Kingston who had these deciduous teeth free from caries showed a slight decrease at age five years (28.2 per cent to 26.4 per cent); but in the six-year old children the percentage increased from 17.2 to 26.3; and in those who were seven years of age, it practically doubled (8.3 to 16.5). On this occasion, these changes in the control city were not even mentioned.

*“Analysis” of findings.* In the 1955 report of Ast et al. it was stated that “As an indication of the benefits of water fluoridation to deciduous teeth, a previous report [in 1953] analyzed the findings among the 6 to 7 year old children in each city after six to seven years following the initiation of water fluoridation.” Actually the report showed findings for the children aged five,
six and seven years who had caries-free deciduous cuspids, first and second deciduous molars. At the ages of six and seven years, in both the test and the control cities, there were increases in the percentages of these teeth that were free from caries; therefore, although these increases were greater in Newburgh, they should not have been attributed solely to water fluoridation. In any case, the publication of one table showing, in this selected group of deciduous teeth, the percentage changes that have just been mentioned, a histogram depicting the def rates, and twenty lines of comment in the text on the results displayed, can hardly be said to indicate the benefits of fluoridation to deciduous teeth, or even to constitute an adequate analysis of the findings in regard to the deciduous teeth present in children aged six and seven years.

Changes in caries-free teeth in the control. In the 1955 report of Ast et al. the age range was changed by not publishing the results for the five-year old children, but showing, for the first time, the results for caries-free deciduous cuspids first and second deciduous molars, for eight and nine-year old children. However, these results cannot be compared with those of the previous years, as they were based on a combined clinical and X-ray examination. Nevertheless, a comparison can be made with the rates shown in the final report (Ast et al., 1956). In the year between the 1953-4 and 1954-5 examinations, the rates in Kingston for the ages six, seven, eight and nine years changed from 10.6, 7.0, 7.9 and 0.0 to 11.1, 4.7, 1.8 and 1.6 for the respective ages. Such changes are not unexpected, for marked variations were seen in Evanston, where, also, the examinations were made by a clinical plus X-ray procedure. For instance, the percentages of children aged seven years who were drinking fluoridated water and who had caries free deciduous teeth were, in successive examinations, 11.33 (pre-fluoridation), 8.71, 3.87, 10.66, 13.01 and 17.86 (Hill et al., 1956). It would seem that assessments made on the basis of caries-free groups of deciduous teeth are not very reliable.

Changes in the sampling method. Consideration of these five dental reports shows that the sampling method changed from time to time, and that the method used in the control city was sometimes the same and sometimes different from that used in the test one. In the first report (Ast et al., 1950) it was stated:

“we are considering only those children age 6-12 who were in the original base study and who have had each successive examination
until they reach age 12. Also included are new school children who entered the study at age 6 subsequent to the first examination and were present at each of the successive examinations. Thus, this study group will have only those children who we are assuming have had continuous residence in their respective cities.”

*Continuous residence only assumed.* It can be seen that the “continuous residence” of each subject was based on assumption only, and not on statements made in a questionnaire, such as was used in Evanston (Blayney and Tucker, 1948). Therefore, it is possible that children could have been absent from the city for considerable periods between the times of successive examinations. Also, there is no assurance that the six-year-old children entering the study in any of the post-fluoridation examinations had not come to live in the area since the commencement of the study. Therefore, it is doubtful whether the objective of having “reasonable assurance that the children studied had had continuous residence in their respective cities” (Ast et al., 1951) can be said to have been attained.

*Population changes in Newburgh.* “Early in 1950 questionnaires were given to more than 3,200 children in the Newburgh schools for completion by their parents” (Ast et al., 1951). The questions asked were not stated, nor was the number of replies received, but it was said that:

> “An analysis of the answers to those questionnaires shows that the Newburgh population is a relatively stable one and that the inclusion of the small migrant groups does not alter the caries picture to any significant degree. Consequently, in this report there are included all 5 to 12 year old children present in the schools in Newburgh and Kingston on the days the examinations were made” (Ast et al., 1951).

Since information in regard to the caries attack rates in these migrant groups could not have been obtained directly from the questionnaires, it is presumed that the dental record cards of those children were grouped and that the cards of the children who were judged from the answers not to be migrants were also grouped, and the data contained in the two groups in regard to the caries attack rates were compared. If that process was carried out, it was not mentioned, nor were data published which would enable the reader to assess the situation. If no differences were found between the two groups, it must be considered to be strange because by that time it was said that “The DMF rates among permanent teeth of 6 to 12 year old children
in Newburgh show a consistent downward trend” (Ast et al., 1951). The United Kingdom Mission (1953) reported that the authors of this study had “found that the proportion of immigrants in Newburgh and Kingston was too small to affect the comparison.” However, although the Newburgh population was said to be “relatively stable”, in the 1954-5 examinations in that city 24 per cent of the children were excluded because they failed to fulfil the residence qualifications (Ast et al., 1956).

The workers who conducted the paediatric study in these cities, Schlesinger et al., in 1950 said that in each city “An effort was made to select... children from families which might reasonably be expected to remain for the duration of the study.” In spite of that precaution, they found that 29.9 per cent of their subjects in Newburgh moved from the city during the period of the study (Schlesinger, Overton, Chase and Cantwell, 1956).

Population movement in Kingston. No mention was made of the issue of a questionnaire to children in the control city; apparently it was assumed that migrants to that city would have come from areas with “fluoride-free” water supplies. Schlesinger et al. (1956) found that 22.2 per cent of the children included in the paediatric examinations moved from Kingston during the period of the study; presumably a similar number of new residents settled in the city.

It may be considered that in moving from one locality to another, interruptions could occur to regular conservative and prophylactic treatment of the children, so that their dental health may not have been as good as that of children who lived for many years in the same city. It is possible also that regular dental examinations, by stimulating interest in the teeth, may improve eating habits and oral hygiene measures.

Considerable alterations in populations. In Table I of Ast et al. (1950) the number of permanent teeth erupted is shown. The numbers given for Newburgh in the examination of 1944-5 for the three age groups six to seven, eight to nine and ten to twelve years are respectively 3,579, 7,937 and 24,586. However, by adding in Table I of Ast and Chase (1953), the number of erupted teeth - for the same age groups, and in the same examination - are 5,379, 10,033 and 27,186. It was stated in the former report that “we are considering only those children age 6-12 who were in the original base study and who have had each successive examination until they reach age 12.” It therefore appears that to meet those requirements, it was necessary
to exclude, for the three age groups, 33 per cent, 21 per cent and 10 per cent of the number of erupted teeth, and, presumably, similar percentages of children. A like situation was seen in regard to the Kingston data, the percentages of teeth excluded being 24, 26 and 12. After only four years, it was apparently necessary to omit these large proportions of the data in order to consider only those children who were “continuous residents”, no other explanation being evident for the different numbers of erupted teeth that were stated in the two papers. Although the population of Newburgh may have been “relatively stable” when compared with some unnamed population, it is obvious that the number of migrants was so great that they should have been excluded from the study.

Data of migrants excluded only in Newburgh. The necessity for excluding the data of migrants was later realized, and the method of including in the study all the children present in the schools on the day of the examination - although it was continued in Kingston - was abandoned in Newburgh. Ast et al. (1955) stated: “Based on residence histories, the Newburgh study group was limited to those who had used Newburgh water since the introduction of sodium fluoride on May 2, 1945.” In the final report, also, only those children who had lived continuously in Newburgh were included, but “All the Kingston children examined are included in this report” (Ast et al., 1956).

Alterations in sample size. The sample size and the age distribution of the children were altered during the course of this study. The data included in the first three dental reports were obtained from the “entire elementary school populations” (Ast and Chase, 1953), except that in some years some of the children were excluded in Newburgh on residential grounds, and that in 1951-2, owing to the loss of an examiner, only half of the children in each city were included. However, in the 1953-4 series the age range was restricted to six to ten years, and the number of children examined was only a small fraction of those inspected in the same age groups during other examinations. Ast et al. (1956) said that the preceding report “dealt with rather small groups of children (about 375 children ages six to ten in each city), and there was considerable difference in age distribution.”

Sampling by selection. The method of sampling used in the 1953-4 examination must be considered to be unorthodox, and was described by Ast et al. (1955) in these words:
“The current series includes a limited number of schools which were chosen because of the availability of X-ray facilities. From previous data on DMF rates by school, it was determined that the selected Kingston school had a caries rate which was among the lowest in the city, while the rates for the three Newburgh schools were distributed through the range of rates for that city. This has the effect of minimizing the difference in the DMF rates between the two cities.”

A decrease in the “per cent difference”. In the final report (Ast et al., 1956, Table 1) the “per cent difference” between the DMF rate per 100 erupted teeth of children aged six to nine years in Newburgh and Kingston was given as 56.7. This is a smaller difference than any of those shown for the ages six, seven, eight and nine years (74.7, 68.3, 58.1 and 66.0 respectively), in the previous (1955) report, despite the fact that it was stated in that report, that the sampling method used had minimized the difference between the DMF rates in the two cities. A trial period of ten to twelve years was suggested by Ast (1943), and was mentioned in the authors’ first report (Ast et al., 1950). In view of the decrease in the “per cent difference” between the test and the control cities, which was revealed in the final report, it is unfortunate that the trial was stopped as soon as the minimum period proposed by the authors had elapsed.
DISCUSSION

The Expert Committee on Water Fluoridation of the World Health Organization (1958) stated that “Hundreds of controlled fluoridation programmes are now in operation in many countries. Some have been in progress for the past 12 years, so that conclusions are based on experience.” This statement suggests that there is a large amount of experimental evidence in regard to the process of artificial fluoridation. It is very doubtful whether this is the case. If hundreds of fluoridation programmes have been conducted with experimental controls, it is strange, and very unfortunate, that such a large body of data has not been published; for, except in the cases of the trials which have been considered here, published data concerning fluoridation trials are very meagre. It would seem, therefore, that the Expert Committee did not use the term “controlled” in its experimental sense, but in that of regulated measurement of the fluoride salt, such as in its statement that “The precision of fluoride application should be carefully controlled.”

The United Kingdom Mission (1953) which visited North America in 1952, in its report referred to “the Fluoridation Studies”, and enumerated only six study centres; and Jenkins (1955) mentioned “the six study centres on the American continent”. In addition to the four test cities which have been considered, the Mission referred to Sheboygan, Wisconsin, and to Marshall, Texas. No control city for Sheboygan was mentioned; and the Mission stated that in the latter study “The neighbouring town of Jacksonville with a fluoride-free water supply was selected as control, but although caries experience in the two areas was compared after 2 1/2 years of fluoridation, the most valuable basis for comparison is the baseline data of Marshall itself.” The Mission quoted two unpublished reports as the source of its information in regard to the latter study. An indication of the minor importance of these two trials is the fact that in the 240-page report of the New Zealand Commission of Inquiry (1957) no data from them were presented, the former city being mentioned twice and the latter only once. Furthermore, the directors of these studies, Doctors F A. Bull and E. Taylor, were not named in the report, and the extensive bibliography did not include any papers published by them.

The crucial importance, even at the present time, of the trials conducted
in Newburgh, Grand Rapids, Brantford and Evanston was demonstrated by the report made in 1957 by the New Zealand Commission, the hearings of which did not conclude until April of that year, and by the report of the Expert Committee of the W.H.O. (1958), which met during August 1957. The only evidence mentioned by the Commission with regard to the dental results of the addition of fluorides to water supplies was that obtained in those four cities. The Expert Committee referred to only the first three of those cities in the few lines of its report which mentioned dental results of fluoridation.

In discussing the general design used in fluoridation studies, the United Kingdom Mission (1953) said:

“In a fluoridation study, two nearby towns, comparable in all respects, are chosen, both having an almost fluoride-free domestic water supply, preferably from the same source. The water of one town is fluoridated while that of the other remains untreated, this town serving as the control. Before fluoridation is started the teeth of the children in both towns are examined in detail to ascertain if caries experience is similar and to determine its prevalence in the various age groups. Further examinations are carried out at yearly intervals and the dental condition of the children in the fluoridated town is compared with that of similar groups in the control town. The prefluoridation data also serve as a basis for comparison. The caries incidence may also be compared with that in a town where a similar concentration of fluoride occurs in the water naturally. In practice it is often difficult to obtain all these conditions and in some studies there is no independent control.”

The term “comparable in all respects” describes a theoretical ideal for a test and a control town rather than a practical possibility. In regard to the other matters mentioned in the design these studies exhibited numerous deficiencies. No control was employed in the City Council study in Brantford, and the Grand Rapids study lost its control in 1951 as a result of the fluoridation of the Muskegon water supply. In the extremely important matter of the water supplies, both the source and the composition of the Newburgh water is considerably different from that of Kingston. Further examinations were “carried out at yearly intervals” only in Grand Rapids-Muskegon, and in Newburgh-Kingston until 1952; if yearly examinations were made in the latter study after that year, the results for all years were not
published. In the Evanston trial, only two examinations were made in the control city, and few data from it have been published; in the test city only one age group was examined each year. In Brantford and in Evanston, and in the first and the last dental reports from the Newburgh-Kingston study, data from children of different yearly ages were added, thus introducing the possibility of “weighting”. In some instances, at least, the degree of “weighting” found indicated that the comparisons were not being made between similar groups in the test and the control cities. No pre-fluoridation data were gathered in Brantford by the Canadian Department of National Health and Welfare, for that study was not commenced until over two and a half years after the fluoridation of the city water supply.

In all of the studies that have been considered, it has been seen that fluoridation of the water supply of the test city was initiated before the initial caries rates in the control city were known. This late examination of the control cities, on first thought, may not seem to be of much consequence. However, it means that, in all of these studies, a matter of fundamental importance was disregarded—it could not have been established that the children of similar ages in the test and the control cities, prior to the commencement of the experiment, had reasonably comparable caries attack rates. Therefore, the statement of the United Kingdom Mission (1953) that “Before fluoridation is started the teeth of the children in both towns are examined in detail to ascertain if caries experience is similar and to determine its prevalence in the various age groups” appears to have been based on assumptions only.

Caries attack rates may be expressed as decayed, missing and filled teeth per 100 erupted teeth, or expressed as per 100 children or per child. The former method was preferred by the authors of the Newburgh trial “because individual teeth may be subjected independently to the hazard of caries” (Ast et al., 1956). In the Evanston study, the rate per 100 erupted teeth was given, but, curiously, only for children aged twelve to fourteen years. All other cases (the younger children in Evanston, and the other studies) in which the caries attack rates per 100 children or per child were given are based on the assumption, unsupported by published evidence, that in each age group the mean numbers of each category of erupted teeth per 100 children is very similar in the test and the control cities, and that little variation occurs from year to year. If this is not the case, comparisons between the rates prevalent in the test and the control cities, and those seen in different years, are not valid. Feltman (1956) gave fluorides—in tablet form—to pregnant women
and young children, and reported that “Many children in the study group showed a marked delay in the eruption of the deciduous teeth. This delay is in some instances a cause for alarm by the parents. The second incisor, second molars, and cuspids are the most frequently delayed, in many cases by as much as a year from the accepted average eruption dates.” Of course, if fluoridation results in the eruption rate of teeth being retarded, a decrease in caries experience would be expected due to the shorter time of exposure of the teeth to the risk of caries. It will be recalled that data were published in the Evanston study which were compatible with a continuous and marked decline in the rate of eruption of first permanent molars during the first four to five years of fluoridation, but that further comparisons could not be made because this type of data was not published for younger children in later reports.

In order to decrease the chance of misinterpretation, extensive use has been made of direct quotation from the original reports, and to avoid unnecessary repetition, consideration of the comments made on the results reported from these control cities, apart from those made by the authors of these studies, has been restricted to the statements of only a few writers.

It is felt that it is not necessary to discuss further the matters which have been noted above, for they are self-explanatory. It has been shown that the reports of the controls used in these fluoridation trials contain arithmetical and statistical errors, and that results and relevant data were omitted. Also, misleading statements were made which denied, ignored, or underrated the unexplained changes in caries attack rates which took place in the control cities, and which suggested that the pre-fluoridation data from the test cities, and those obtained during the basic examinations in control ones, were more closely comparable than was the case. Jean R. Forrest, the Senior Dental Officer, Ministry of Health, who was a member of the United Kingdom Mission and of the Expert Committee on Water Fluoridation of the World Health Organization, in 1957 contrasted “the emotional type of opposition” to fluoridation, to “the precise correct statements of scientists.” However, the situation which has been encountered is more aptly described by the words of Wade Hampton Frost “an outstanding American epidemiologist” (Bews, 1951). More than thirty years ago Frost (1925) said:

“It is frequently easy to exhibit some figures which, though not
really to the point, will nevertheless serve to impress an uncritical public, and the temptation may be great to give them, at least by implication, an unduly favourable interpretation. It is more difficult and more tedious to present the full argument, based on all the facts, and it is perhaps a little humiliating to admit that the statistical evidence is deficient because we have failed to collect it; but to do this is not only more scientific, it is in the end more convincing, and after all there is no free choice, because it is the only honest method, whether it be convenient or not. Finally, it is the only way of progress, for the first step towards collecting better evidence is to recognize the deficiencies of that which is at hand.”

More than eleven years after the initiation of the last of these five trials, the deficiencies of their controls still remain unrecognized. The endorsements of fluoridation by medical and dental associations, by public health authorities, and even the recent one by the Expert Committee on Water Fluoridation of the World Health Organization (1958), appear to have been based mainly on the opinions of the authors and of others. Indeed, in the report of that Committee, under the heading “Results of fluoridation”, instead of results being considered, comment was confined to: “Reports of the results after 10 years of controlled fluoridation in three cities”. Examinations of the data obtained in these trials, which have been published by other endorsing bodies, are also inadequate or absent. It is an understatement to term this failure regrettable.

In 1951, Appleton stated that in any future fluoridation trial: “The experiment should be genuine, and not one in name only. In designing such an experiment, a careful and competent analysis of those now in progress should first be made, in order to see how they might be improved or extended.”

In the early part of this paper some basic experimental considerations were mentioned. In cities in which it is intended to compare the caries attack rates of the children in a fluoridation trial, the three main factors which should be as closely comparable as is practically convenient are the composition of the water supply, the climate and the dental caries attack rates. Four trials having “fluoride-free” control cities have been considered. The composition
of Newburgh’s water supply is considerably different from that of its control city. There were gross differences between Evanston and its control city regarding the initial caries attack rates in the younger children. In the Department of National Health and Welfare study in Brantford, as the first examination was made over two and a half years after the commencement of fluoridation, it cannot be known what the pre-fluoridation rates in that city would have been, if assessed by those examiners; thus, it cannot be determined how closely the (1945) Brantford rates resembled those of Sarnia. In the Grand Rapids study, the fluoridation of the water supply of Muskegon in 1951 severely limited its usefulness as the control city.

In each trial both the test city and its control were selected. For instance, “Oak Park graciously offered to serve as the control community” for Evanston (Blayney and Tucker, 1948). Two cities which had agreed to participate in the experiment, after having been found suitable for comparison, should have been allotted at random to be test or control. It will be recalled that in at least two of the cities selected as test ones, Evanston and Brantford, “dental care was outstandingly good” (United Kingdom Mission, 1953).

It has been pointed out in Part One that in all these trials no attempt was made to devise a randomization procedure in the clinical examinations which would have eliminated examiner bias, nor were estimates made of examiner variability.

Two statements made by authors of these studies may be recalled. In 1950 Hill et al. said: “It is to be expected that the rate of caries in all teeth varies from year to year due to chance. A significant reduction of caries prevalence can therefore be assumed to exist only when the statistical analysis of the data provides almost absolute certainty that the observed differences are not due to chance.” However, as was mentioned in Part One this very important matter of random variation has been ignored in all these studies. Blayney and Tucker (1948) stated that: “A study of this nature must have an adequate control.” It has been seen that the controls used in these trials cannot be considered to be adequate.

It would appear that these shortcomings have not been recognized, for those who conducted these studies, and other writers, have expressed their satisfaction with the methods used. For instance, Ast and Chase, the authors of the 1953 report on the Newburgh-Kingston study, referred to “the carefully controlled studies such as the Newburgh-Kingston, Grand
Rapids-Muskegon, and the Evanston studies”; and Mather (1957) said: “This study at Brantford was most carefully set up and has been under the strictest control.”

Approval of the methods used in these studies was also expressed by the New Zealand Commission of Inquiry (1957), for it considered that: “All these investigations” in Brantford, Newburgh and Grand Rapids “were designed and executed with great thoroughness.” The Commission also said: “We have examined the statistical evidence brought forward by the advocates of fluoridation, and the conclusions they have drawn from that material ... We have found nothing to invalidate the statistics or cast doubt on their reliability.” It will be realized that many of the deficiencies of these studies can be noted only when different reports from the same study are compared. It seems that the Commission was handicapped in this regard, for although its “List of exhibits produced at public hearings” mentions over 250 items, such as papers, books, charts and letters, it includes only the final report, or the one which was the most recently published at that time, of the numerous reports showing the dental caries attack rates which were published in each of these studies. It would appear that none of the earlier accounts of these trials were shown to the Commission, nor were they mentioned in the “bibliography” of 144 references. None of the reports from the City Health Department trial in Brantford were listed as exhibits. The paper by Brown, Kohli, Macdonald and McLaren (1954a) which is mentioned deals only with gingival results. Although the Commission had the assistance of legal counsel in gathering the evidence, no mention was made of the employment of a statistician to assist its members in evaluating the numerical data.

The Expert Committee on Water Fluoridation of the World Health Organization (1958) also expressed its satisfaction with the methods used in these trials. Out of the hundreds of controlled fluoridation programmes which it stated have been set up, it mentioned only the Newburgh, Grand Rapids and Brantford (City Health Department) studies in the sixteen lines which allotted to the mention, one cannot say consideration, of the results of fluoridation on dental caries prevalence. Presumably these three trials were cited because the Committee considered that they were the most important and reliable studies, and it said that they were “carefully planned and controlled”. As this opinion of the Committee was made in referring to the three studies which it cited in mentioning results of fluoridation, it is
reasonable to assume, at least in this instance, that the term “controlled” was used in its experimental sense.

If this is the case, the inaccuracy of that statement of the Expert Committee is astonishing, for it will be recalled that, of the three studies which the Committee quoted, the Brantford (City Health Department) study, far from being carefully controlled, was not controlled at all. Furthermore, the control for the Grand Rapids study was abandoned after only six years, at the crucial stage of the trial when the first of the permanent teeth were erupting in the children of the test city who had ingested fluoridated water throughout their lives. Therefore the control, was abandoned before any assessment of caries activity in those teeth could be made. In regard to the remaining study mentioned by the Expert Committee, the Newburgh trial, after the unexplained decreases in the DF rates for deciduous teeth, which were shown as having occurred in Kingston, the control city, between 1945-6 and 1949, no further caries rates for deciduous teeth were published. Also, the erratic changes which were made in the methods used in this trial are not consistent with careful planning, nor is the choice as the test and control areas of two cities with water supplies which were of considerably different composition.

It has been acknowledged for many years that one of the fundamental procedures in planning an experiment is the establishment of a statistical design for the procedures before work is commenced. The deficiencies in the basic statistical requirements of a good experimental design are only too obvious in all these studies. Therefore, it is surprising that the Expert Committee did not point out these deficiencies, but, on the contrary, described the three studies which it mentioned as carefully planned ones. The importance of these matters is emphasized by the authoritative statement of Fisher (1951) that: “If the design of an experiment is faulty, any method of interpretation which makes it out to be decisive must be faulty too.”

This investigation of reports of these fluoridation trials was instituted when a preliminary examination of the methods used revealed disturbing facts, and solely because it was felt that, as Appleton (1951) expressed it: “Professionals and specialists have the duty of insisting upon a scientific demonstration of a high probability that a proposed method will be useful and safe, before it is recommended for general adoption. The maintenance of this attitude is of paramount importance.”

The deficiencies of these trials not having been recognized, many cities
have already fluoridated their water supplies on advice which is based largely on the results that have been considered. It is, therefore, an important and urgent matter that a more accurate assessment of the efficacy of this process should be obtained, but, unfortunately, it appears that little long-term experimental evidence is available. Therefore, despite the limitations imposed by the methods used in these studies, consideration should be given to a careful and competent examination of the whole of the original data obtained in them. The findings resulting from such an examination would be of assistance in designing future fluoridation trials, and would provide a far more adequate assessment of the results reported from these studies than it is possible to obtain from an examination of the very limited data that have been published.

At least until such a report is available for examination, it would be wise to maintain an open mind in regard to the efficacy of artificial fluoridation.
SUMMARY

1. Endorsements of the process of the mechanical fluoridation of public water supplies rely mainly on five experimental trials.

2. The controls used in these studies are considered.

3. The reliability of the results reported is affected by:
   
   (a) odd experimental and statistical methods;
   (b) failure to consider random variation and examiner variability, and to eliminate examiner bias;
   (c) omission of relevant data;
   (d) arithmetical errors;
   (e) misleading comments.

4. Controls were either doubtful or inadequate.

5. No control was employed in one trial.

6. The published data do not justify the statement that caries rates remained the same in control cities.

7. The sound basis on which the efficacy of a public health measure must be assessed is not provided by these five crucial trials.
Soon after the publication of the first edition of this monograph in September 1959, the author was informed by Dr K. T. Adamson, President of the Australian Dental Association, that copies had been sent to “all of the men who are in charge of the experiments” asking them for comments (personal communication). As a result the reviews published in the February 1960 issue of the Australian Dental Journal were contributed by Dr Donald Galagan, Dr J. R. Blayney and Dr I. N. Hill, and by Dr R. M. Grainger. The review in the New Zealand Dental Journal of January 1960 was written by Mr J. Ferris Fuller.

The aim of this monograph is to attempt to clarify some aspects of five crucial trials of artificial fluoridation, those conducted in Grand Rapids, Evanston, and Newburgh, U.S.A., and the two trials held in Brantford, Canada. Therefore, in this part, all these critical reviews will be quoted in full; and comments will be made on the points raised and indicated by the figures in brackets.

In order to allow the reader to appraise the criticisms more easily it will be necessary to refer to the statements made in the text, therefore page references are indicated for Parts One and Two of this volume.
The first review of this book in the *Australian Dental Journal*, by Dr Donald Galagan, Assistant Chief, Division of Dental Public Health, Public Health Service, Washington 25, D.C., was as follows:

My comments will be limited to the general qualities of Dr Sutton’s treatise and the conclusions he has reached. Individuals associated with the several fluoridation projects which he has purported to “analyse” will have provided their specific reactions to his “analysis” of their findings.

Although it is nothing new to see an accredited scientist mix fact and fancy, near truth with truth, and emotion with reason it is always shocking to realise that an intelligent individual in a responsible position can so baldly misinterpret scientific data. Actually, it would be extremely difficult for an objective scientist who knows anything at all about the data characterizing the relationship between dental caries and exposure to fluorides to reach the conclusion that the effectiveness of controlled water fluoridation in reducing dental caries has not been proved. This conclusion could only be reached by an armchair statistician who has chosen to ignore or does not know the great mass of information on the subject.

Thus, first and foremost among the *fundamental* errors(1) which Dr Sutton makes is expressed in the statement in the second paragraph of the monograph which says that “proposals to fluoridate domestic water are almost entirely based on the results of the Brantford, Grand Rapids, Newburgh, and Evanston projects”. Using this premise, the balance of the document is devoted to efforts to describe “errors and omissions” in these four projects. These errors are supposed to negate the whole fluoride-caries hypotheses(2), or at least to throw serious doubt on the fluoridation of water as a caries preventive.

Actually, the scientific basis on which this public health measure rests was established solidly before any of the above mentioned projects were started. The preventive effect of long term exposure to water-borne fluoride on caries experience was observed in literally thousands of children residing in many different communities where the water consumed had picked up the element as it coursed over or through the earth’s crust. The long series of investigations documenting the relationship are considered to be classic examples of good epidemiological method(3) - so much so that they are used as case studies in teaching the science of epidemiology in our schools of public health.
The fact is that the projects at Brantford, Grand Rapids, Newburgh and Evanston were designed primarily to evaluate the technical, financial and administrative problems associated with the controlled addition of fluorides to a municipal water supply, and, secondarily, to demonstrate the effectiveness of the procedure(4) to the profession and the public. To be sure, it was necessary to show that the procedure would reduce the caries attack rate in resident children, but as soon as the trend toward reduction was observed and corroborated, the “experimental” portions of the projects were completed for all practical purposes. The principal point, however, is that these projects emphatically are not the sole basis on which the widespread use (in the United States) of this procedure rests(5).

In short, it is preposterous to attempt to conclude that the basis for community water fluoridation is faulty because of some real or imaginary defects in the planning, execution and evaluation of data from the four community projects. The contents of the monograph, therefore, represent no more than an exercise in semantic and scientific dilettantism designed to serve some other purpose.

There are a good many other specific, but less important errors in judgment which the author has made, such as his suggestion that the variability of examiners in diagnosing dental caries has been overlooked(6). The truth is that this variability is well known, and is discussed at length by specialists in the fields of epidemiology and caries diagnosis. Because of this “human error” calibration of examiners is practised as a matter of course. Calibration reduces the variability among examiners, but even if it did not, the difference between the caries experience of children exposed and not exposed to fluoride is so great that even Dr. Sutton could recognise it(7).

There are several other examples of errors of judgment in the arguments contained in the monograph, indicating the author’s serious lack of understanding of the principles of statistics and epidemiology. For instance, the rather amateurish interpretation of adequate community “controls” for evaluating the effect of fluoride-bearing water on caries indicates that the author does not really know the manner in which caries occurs in a population(8).

The author’s use of innuendo to make a point is contained in his reference to some very questionable work of Feltman which indicated that the eruption of deciduous teeth might occur later in children exposed to fluorides. The author implies that it is likely that retarded tooth eruption in the children
residing in the fluoride communities reduces their exposure to caries attack, and thus their caries rate naturally is lower (9). Had Dr Sutton been familiar with the literature, he would have known that this was one of the first possibilities thought responsible for the low caries rate in children exposed to fluoride-bearing water (10). He would have known about the rather exhaustive report of Short on the relation between fluoride in domestic waters and tooth eruption which showed that fluoride in concentrations around the optimum used for caries control does not influence the eruption pattern of permanent teeth (11).

However, one suspects that further analysis of the details contained in the monograph will not yield much of value. From reading the document and from hearing him present part of it as a paper last February at your Adelaide Congress, I can only conclude that Dr Sutton has an intense and emotional drive to oppose fluoridation. Why he feels this way is not clear, but it seems likely to come from some motive other than a sincere concern for the statistical or scientific validity of the concept (12).

Commentary on the Review by Dr Donald Galagan

(1) Dr Galagan makes the charge that the “first and foremost among the fundamental errors” was made in the second paragraph of the monograph (p. 136). This paragraph stated: “Apart from these considerations, an examination reveals that there are aspects that call for a very careful appraisal of the figures presented in the reports of the experimental trials which have been conducted in Brantford, Canada, and in Grand Rapids, Newburgh and Evanston, U.S.A., and upon the results of which proposals to fluoridate domestic water are almost entirely based.”

Such proposals are based on two different sets of results—those reported from areas where fluorides occur naturally in the water supplies, and those from trials of the mechanical addition of fluorides to waters in which the fluoride content is very low or absent. The former reports are not considered in this investigation, but Dr Galagan says that they were gathered in such a way that they “are considered to be classic examples of good epidemiological method”. In any case, they are of practical importance only if it is known that the results reported will be obtained when commercially available fluorides are mechanically added to water supplies.

This knowledge can be obtained only by the use of experimental trials—the
results of which must then be considered to be of outstanding importance—unless it is accepted that it can be established, on theoretical grounds, that the results of artificial fluoridation will be identical with those seen in areas where fluorides are found naturally. If the latter case is accepted, the early artificial schemes which are considered here were held merely, as Dr Galagan suggests, “to demonstrate the effectiveness of the procedure” and cannot be considered to be true experimental trials.

The question to be answered is this: Were these trials mere demonstrations or were they set up as genuine experimental studies? If they were only demonstrations Dr Galagan’s charge is justified, but if these trials were conducted to determine the outcome of the process of mechanical fluoridation, then his accusation is without foundation and must be discredited.

In 1951 a report was issued by the ad hoc Committee on Fluoridation of Water Supplies of the National (U.S.A.) Academy of Sciences, National Research Council (Maxcy, Appleton, Bibby, Dean, Harvey, Heyroth, Johnson, Whittaker and Wolman, 1952). One of the members of this Committee was Dr H. Trendley Dean, who was closely associated with much of the earlier work concerning fluorides and was, at that time, the Director of the National Institute of Dental Research. The report included this statement: “In 1945, studies were begun to ascertain whether the adjustment of the fluoride content of a public water supply to the optimal level with commercially available fluorides would confer the same caries-inhibitory effects as do waters which carry the same concentrations of fluoride naturally.”

This statement makes it clear that Dr Galagan’s contention, that these trials were only demonstrations, is not correct. However, because of the importance of this matter and in case it is suggested that that Committee was misinformed concerning the intention of these trials, a quotation will be given from each of the five studies considered. Dr Galagan calls the process used in these test cities “the controlled addition of fluorides to a municipal water supply”, but the original term used by Dean et al. (1950) and by Brown (1951, 1952), “artificially fluoridated” drinking water, is preferable as it is free from ambiguity (p 63).

The quotations from the five trials are as follows:

(a) Brantford, City Health Department Study, Hutton et al., (1951): “It was recognized that fluorine in the public water supply was not a proven method
for the prevention of dental caries, and that it might take ten years to prove or disprove its preventive value.”

(b) Brantford, National Health and Welfare Study, Brown et al., (1954b): “The Brantford Fluoridation Caries Study was undertaken with a view to finding out whether or not the raising of the fluoride content of a previously fluoride-free water supply to part per million, by the mechanical addition of sodium fluoride, would reduce the incidence of dental caries to that which obtains where water supplies derive about 1 part per million of fluoride from deposits in the earth”

(c) Grand Rapids, Dean et al. (1950): “In 1945, three studies to determine the caries prophylactic value of artificially fluoridated drinking water were started in the United States and Canada.”

(d) Newburgh, Ast et al. (1950): “In 1943 it was proposed to determine whether we can translate the conclusions derived from the epidemiological studies in fluoride areas to a practical application in fluoride-free areas where the communal water supplies may lend themselves to treatment.”

(e) Evanston, Blayney and Tucker (1948): “After further deliberation of the project, both professional groups recommended to the Commissioner of Health that a carefully controlled study be developed to determine whether or not the addition of fluorine in minute quantities to the communal water supply would reduce the incidence of dental caries in Evanston and Skokie children.” Blayney and Tucker (1948) also said: “It was carefully explained to these Evanston citizens that nothing could be promised regarding the ultimate value in the control of tooth decay; that if such a program was to be undertaken it must be in the nature of an exhaustive study; and that it would be several years before data would be available which would even indicate the trend which we might expect.”

These statements, by the authors of all of the five studies considered, establish beyond question that in every case the studies were not designed “to demonstrate the effectiveness of the procedure” but to determine whether or not artificial fluoridation would be efficacious. Therefore Dr Galagan’s opinion is incorrect, and the statement made in Part One, which he termed the “first and foremost among the fundamental errors”, is a correct description of the situation.
(2) His suggestion that an attempt has been made to “negate the whole ‘fluoride-caries hypotheses’ is without foundation. The only reference to this matter is contained in the quotation of a statement, made in 1949, by the American Water Works Association regarding the experimental verification of “the fluoride-dental caries hypothesis” - that is, to the “fluoridation hypothesis”. Statements made in the preface to the first edition and on the first page of Part One and the first and last pages of Part Two show that consideration has been given only to five experimental trials of artificial fluoridation produced by mechanical means. The data from epidemiological studies in “naturally fluoridated” areas, on which the fluorine-dental caries hypothesis is based, have not been considered.

(5) Dr Galagan states that “The principal point, however, is that these projects emphatically are not the sole basis on which the widespread use (in the United States) of this procedure rests.” It can be seen, by reading the “free” quotation from paragraph two, page 136, given by this reviewer (1) and pages 140, 189, 190 and 196, that the word “sole” was not used nor implied.

No comment can be made on the other “fundamental errors” which Dr Galagan says (1) are present-for he has neglected to state their nature. Instead he continues:

(6) There are a good many other specific, but less important errors in judgment which the author has made, such as his suggestion that the variability of examiners in diagnosing dental caries has been overlooked.

Comment. This suggestion was not made. As Dr Galagan points out in his next sentence (7), “this variability is well known”. Therefore it is most unlikely that such a matter would be “overlooked” in studies employing statisticians. Indeed, attention was drawn, on page 180, to the fact that the importance of examiner variability was recognized by Ast et al. in 1950. However, in conducting the clinical examinations, in all the studies considered, this matter was ignored. Therefore, when speaking of this phenomenon, terms such as “not assessed” and “not estimated” were used.

(7) The truth is that this variability is well known, and is discussed at length by specialists in the fields of epidemiology and caries diagnosis. Because of this “human error” calibration of examiners
is practised as a matter of course. Calibration reduces the variability among examiners, but even if it did not, the difference between the caries experience of children exposed and not exposed to fluoride is so great that even Dr Sutton could recognise it.

Comment. As Dr Galagan says, this (examiner) variability is well known. It is precisely this fact that makes it so surprising that this factor was not assessed in these studies. It will be recalled that reference was made, on page nine, to two papers which investigated the matter of examiner variability in caries diagnosis.

The claim is made by the reviewer that “Calibration reduces the variability among examiners”, but he does not suggest that this process eliminates between-examiner variability—therefore it should have been taken into account. Of course, the use of the method of the “calibration” of the subjective judgment of several examiners with the subjective judgment of another is, to say the least, a poor substitute for a standard rigorous statistical procedure.

(8) There are several other examples of errors of judgment in the arguments contained in the monograph, indicating the author’s serious lack of understanding of the principles of statistics and epidemiology. For instance, the rather amateurish interpretation of adequate community “controls” for evaluating the effect of fluoride-bearing water on caries indicates that the author does not really know the manner in which caries occurs in a population.

Comment. The question may be asked: Who does?

(9) The author’s use of innuendo to make a point is contained in his reference to some very questionable work of Feldman (p. 192) which indicated that the eruption of deciduous teeth might occur later in children exposed to fluorides. The author implies that it is likely that retarded tooth eruption in the children residing in the fluoride communities reduces their exposure to caries attack, and thus their caries rate naturally is lower.

Comment. On page 192, reference was not made to the work of Feldman but to that of Dr Reuben Feltman who was an associate of Doctors D. E. Gardner and F. A. Smith, whose publications on fluorides are well known, and of Doctors H. C. Hodge and D. E. Overton, who were closely associated with the Newburgh trial (Gardner, Smith, Hodge, Overton and Feltman,
1952). Dr Feltman’s earlier (1951) work with fluoride tablets was referred to by the New Zealand Commission of Inquiry (1957) as “promising”. His 1956 paper, which was quoted (p.192), was a brief “progress report” only. Therefore the results mentioned in it were treated with reserve, the statement being made that “Of course, if fluoridation results in the eruption rate of teeth being retarded ....”.

(10) Had Dr Sutton been familiar with the literature, he would have known that this was one of the first possibilities thought responsible for the low caries rate in children exposed to fluoride-bearing water.

Comment. Dr Galagan has avoided the main point which was discussed in this paragraph. His insistence that one of the first possibilities considered was that the ingestion of fluoride-bearing water may retard tooth eruption, makes it even more strange that in only the Newburgh-Kingston study was mention made that this important matter had been investigated. Even in that trial, the only study of tooth eruption rate published was conducted after four years of fluoridation (Ast et al., 1951) in children who were six to twelve years of age, so that none of the subjects studied had been ingesting artificially fluoridated water throughout their lives.

(11) He would have known about the rather exhaustive report of Short on the relation between fluoride in domestic waters and tooth eruption which showed that fluoride in concentrations around the optimum used for caries control does not influence the eruption pattern of permanent teeth.

Comment. It is highly probable that Dr Galagan is referring to the paper by E. M. Short (1944) which is well known to those interested in fluorides, for this is the only paper concerning fluorides and tooth eruption listed under that name in the Index to Dental Literature in the English Language (published by the American Dental Association), the Quarterly Cumulative Index Medicus (to December 1956), or the issues of the Current List of Medical Literature which cover the subsequent period.

This “rather exhaustive report of Short” does not show whether or not the ingestion of fluorides at the “optimum” level has any influence on the eruption pattern of permanent teeth. This report was made on “selected 12-14 year old white school children” (Short, 1944) in whom almost all the permanent teeth had erupted. The data deal only with the total number of erupted permanent teeth and, despite Dr Galagan’s remark, do not give any
information regarding their eruption pattern—a factor which could be of considerable importance in the development of the DMF rate. Short’s Tables II and III show that, in all except three of the 4,453 children examined, at least twelve of the permanent teeth had erupted, the minimum number of erupted permanent teeth, for the remaining three children, being ten. His Table I (which excluded third molars) shows that, out of the possible twenty-eight teeth, the mean number of erupted permanent teeth per child in the various cities was between 25.22 and 26.81. In fact, in fifty-five per cent of the children all of the twenty-eight teeth had erupted.

Therefore, this study of Short (1944) gives no information regarding the ages at which the first ten permanent teeth erupted in these children, certainly none regarding the first permanent molars which, presumably, even in the youngest of these children, erupted about five or six years prior to the study. These molars are of outstanding importance in regard to the DMF rate, particularly in young children. Ast et al. (1956) said: “The first permanent molars are frequently used as an index of caries experience among children because this tooth accounts for the major incidence of caries in this group.”

It should be noted that neither Dr Galagan nor (as will be seen in their review of this book) Doctors Blayney and Hill, authors of the Evanston study, have commented on the suggestion of a decline in the eruption rate of first permanent molars in Evanston, between 1946 and 1951, which followed the introduction of fluoridation and which is depicted in Figure 4 (p. 27). Neither have they explained why, after 1951, whilst continuing to publish this type of data for the older children, they ceased publishing it for the younger

(12) However, one suspects that further analysis of the details contained in the monograph will not yield much of value. From reading the document and from hearing him present part of it as a paper last February at your Adelaide Congress, I can only conclude that Dr Sutton has an intense and emotional drive to oppose fluoridation. Why he feels this way is not clear, but it seems likely to come from some motive other than a sincere concern for the statistical or scientific validity of the concept.

Comment. Dr Galagan’s questioning of the motive behind this study should be considered in relation to statements which he made during his lecture to the Adelaide Congress (Galagan, 1959). He called those who questioned
fluoridation “the opposition” and said that this group “seems to be composed of four distinct kinds of people.” These he termed: “the hatemonger, the pseudo-health believer, and the person who opposes fluoridation for personal notoriety” and “the fourth, or rugged individualist, group”. As these are Dr Galagan’s views, it is not surprising that he doubts the sincerity of this attempt to investigate “the statistical or scientific validity” of these fluoridation findings.
The second review of this book in the *Australian Dental Journal*, by DR J. R. BLAYNEY, Director, Dental Caries Study, Evanston, and DR I. N. HILL, Zoller Memorial Dental Clinic, University of Chicago, was as follows:

Dr Sutton has much to say regarding the lack of comparability of the study and the control areas and the manner of selection of the children in each area to be examined. Oak Park is the suburb immediately to the west of Chicago and Evanston lies immediately to the north. Each community draws its water supply from Lake Michigan. Standard analyses for composition of the water are frequently run. A spectro-chemical analysis for 26 trace elements has been run on Lake Michigan water for a comparison of a similar analysis of water obtained from fluoride areas (unpublished data). Oak Park and Evanston receive their food from the same wholesale markets, each is chiefly residential and free from heavy industry. Each is composed of the same socio-economic level, as borne out by the United States census of population for 1950. In Evanston, 47,395 persons 21 years or over were native born. In Oak Park the figure was 42,454. In Evanston there were 6,049 foreign born persons 21 years or older while in Oak Park the figure was 5,081. In Evanston 41.2 per cent of the occupied dwelling units were owner occupied. In Oak Park 50.8 per cent were occupied by the owner. The median value of a one dwelling unit structure in Evanston was 19,499 dollars and in Oak Park the value was 16,259 dollars. The median value of gross monthly rentals in renter occupied dwelling units in Evanston was 72.53 dollars while in Oak Park the median value was 66.86 dollars. Both areas have comparable, climatic conditions and both are subjected to the same radio and television commercials regarding oral hygiene and dentifrices. Finally, the majority of the dental practitioners in the study and control areas are graduates of one of the three Chicago dental colleges.

For the baseline examination in Evanston we were committed to examine all school children within the selected age range regardless of the length of time they had resided in Evanston. Although an effort was made to include them, the Oak Park Parochial Schools did not find it convenient for us to examine their pupils. When we compared the caries prevalence rates of the two towns we found a difference. It is indeed fortunate that our records showed the school that each child attended. In this manner we could first eliminate the Parochial School group and then the Negro group from the total Evanston data. It is well known that coloured people have less dental
caries than whites living in the same population centres(15). The caries rates for the Evanston and Oak Park Public School white children compared favourably(16). All of this clearly indicates how hidden variables may exist in areas which otherwise appear to be comparable, and how important it is to be certain that comparisons are made between like groups(17).

Dr Sutton expressed astonishment that in 1955 the six and seven-year-old Evanston children had a lower caries prevalence rate than the Aurora children of like ages. We, likewise, did not anticipate this. However, the same critical evaluation both clinical and roentgenological, was made of every case. This difference was due to something other than fluoride. Possibly the presence of the dental team in the school, year after year, has stimulated the classroom teachers and the school nurses to place more emphasis on the teaching of oral health. Some unknown hidden variation not related to fluorides must account for the difference(18).

Dr Sutton was concerned that the control group was not examined annually. Neither we nor our advisers could see a reason to require an examination of the control group other than at the beginning and near the close of the study. This provides the baseline from which to measure the trend of the dental caries rate during the time interval (1947-1956). Should the rate in the last examination (1956) deviate materially from that of the initial baseline period (1947) that figure could be used as a correction factor on the Evanston findings. In fact, if we had not desired to measure the yearly decrement in the rate of dental caries under fluoridation and evaluate other factors only two examinations in Evanston would have been necessary, the first in 1946 (before fluoridation), the second and final in 1961(19).

Much has been made of the variations reported in the number of children examined. The baseline examination of 4,375 Evanston children and of 2,493 Oak Park children are correct. However, the data from those children who had not used Lake Michigan water all of their lives had to be excluded. It was also observed that some children below the age of 67 months and above the maximum of 174 months had been examined. Therefore these out of range children were not considered in the final determination of the caries rates. This explains the discrepancy between the Evanston 4,375 and 3,692 and the Oak Park 2,493 - 2,051 figures(20). It should be noted that when dental caries experience rates were compared, the same number of examinations, that is 1,991 for the six to eight-year-old children and 1,701 for the 12 to 14-year-old children, were used throughout the reports for
the combined Evanston school groups (Public White, Parochial and Public Negro)(21). Dr Sutton on p. 167 of his report (fig. 5, Statement C) calls attention to Evanston Dental Caries Study Report Number XVII. Here he points out that only 1,754 six to eight-year-old children and 1,556 12 to 14-year-old children were listed. This particular report was primarily concerned with differences in sex and race, as they influenced caries, rather than the effect of fluorides. Therefore a comparison was made of the dental caries experience rates of white girls to white boys; Negro girls to Negro boys; white girls to Negro girls; white boys to Negro boys; and white children as a group to Negro children as a group. The children were classified into male white, female white, male Negro and female Negro for comparison(22). As Dr Sutton calls attention to the difference in the number of examinations made in this report when compared with other reports, we wish to point out, in explanation, that it was necessary to not only insure that children of correct age be included but also it was necessary that every examination be clearly classified according to race and sex. There were 236 six to eight-year-old children and 245 12 to 14-year-old children excluded from this report because they did not fulfil the requirements for this comparison. These children were included in other reports as no distinction except age was made(23). It is also pointed out that this report, Evanston No. XVII as noted above, was primarily concerned with comparison of the caries rates of the coloured and white children and not with the effect of fluoridation on dental caries rates. Therefore in this light, this report, No. XVII, should not have been listed under the general heading of fluoridation as Dr Sutton has it listed in his critique(24).

It is true that a discrepancy in figures published in our paper XVI, Table I and in paper XVIII, Table I are at variance. This is due to the operator of the tabulating machine providing the wrong figures for the number of seven and eight-year-old children examined. This error was discovered after manuscript XVI was in press and therefore the corrections could only be made manually in the reprints supplied to readers who requested them(25).

Commentary on the Review by
Dr J. R. Blayney and Dr L N. Hill

(13) Reference to Part Two will show that no suggestion was made in it that there was a “lack of comparability” between Evanston and Oak Park; it was merely pointed out that the manner in which Oak Park resembled Evanston
was not stated (p. 16). Therefore this detailed exposition of the similarity
of the two cities is welcome, although it is unfortunate that, when speaking
of rental and dwelling values, the “mean” values were not given as well as
the “median” ones. It will be realized that the housing picture may be very
different in two towns and yet the “median” (that is, the middle) values
of the rentals and dwellings can be the same. In view of the data shown
here, regarding housing and the training of the dentists, it is surprising that
the members of the United Kingdom Mission (1953) should have singled
out Evanston for comment, remarking on its high economic level and its
“outstandingly good” dental care (p. 149).

These data also show how reasonable was the assumption of Dr Blayney
before assessing the caries rates in Oak Park—that that city was “the ideal
control community” for Evanston (Blayney and Tucker, 1948;). They also
emphasize how strange it is that such gross differences should be found
between the initial caries rates of the children aged six to eight years in
the two cities, and reported-after a delay of ten years—by these workers (p.
153; Fig. 3, p. 154). The fact that such gross differences can be found in
the caries rates prevalent in two cities which were so similar that one was
termed “the ideal control community” for the other (Blayney and Tucker,
1948), confirms the necessity for pre-fluoridation examinations in both test
and control cities. Unfortunately, as pointed out in Part Two this was not
done in any of these studies.

(14) For the baseline examination in Evanston we were committed to
examine all school children within the selected age range, regardless
of the length of time they had resided in Evanston. Although an
effort was made to include them the Oak Park Parochial Schools
did not find it convenient for us to examine their pupils.

Comment. The latter remark is welcome for it explains the absence of data
from the parochial schools in Oak Park.

(15) When we compared the caries prevalence rates of the two towns we
found a difference. It is indeed fortunate that our records showed
the school that each child attended. In this manner we could first
eliminate the Parochial School group and then the Negro group
from the total Evanston data. It is well known that coloured people
have less dental caries than whites living in the same population
centres.

Comment. Despite their statement that “It is well known that coloured people
have less dental caries than whites living in the same population centres”,
when conducting the initial examination in Evanston the authors combined
the data of the Negro children with those of the white children. It is now
clear, as deduced on page eighteen, that the racial and school groups were
taken into account only after it was found that there was “a lower caries rate
for school children of the control area” (Hill et al., 1951). Thereafter, when
comparing the test and control cities, the data of both the Negro and the
parochial school children were excluded from the Evanston data. No reason
has been given for this exclusion of the data of parochial school children
in Evanston-who had a high caries rate (Hill et al.) - from the data of the
main body of white children in that city. This could not be attributed to the
fording of a similarly high caries rate in the parochial school children in the
control city of Oak Park for they were not examined and, therefore, their
caries rates were unknown (see 14).

Neither has an explanation been offered for the extraordinary reversal of
this policy (the exclusion of the data of Negro and parochial school children
in Evanston) when compiling the XVIII Report (Hill et al., 1958). This
report published, for the first time, the initial caries rates for the permanent
teeth of the children aged six to eight years which were obtained, ten years
earlier, in the control city of Oak Park.

The rates for the deciduous teeth, which were obtained at that time, still
have not been published. This report provided the first opportunity to
compare the initial caries rates of the younger children in the test city and
its control (p. 153).

(16) The caries rates for the Evanston and Oak Park Public School
white children compared favourably.

Comment. This statement is interesting-for the caries rates for children aged
six to eight years in each of the three school groups have not been published
(p.151). It will be recalled that the mean caries rates for the six, seven, and
eight-year-old children in Evanston in 1946 were very much higher than the
mean rates for children of those ages obtained during the initial examination
in Oak Park. For the children aged six years the rate in Evanston was 46.85,
but it was only 26.89 in Oak Park (Hill et al., 1958). Only 0.1 per cent of the
Oak Park children were Negro (Hill et al., 1951), but exclusion of the data
of Negro children (who have a relatively low caries rate, see 15) from the
Evanston data would increase the rate of the remaining (white public and
parochial school) children so that in the six-year-old children, it would be
higher than 46.85. Therefore the difference between this rate and the Oak Park rate of 26.89, for children of that age, would be increased.

Hill et al. did not say how many of the younger age group of children attended each type of school, but only twenty-two per cent of the twelve to fourteen-year-old children, shown in their 1957a report as examined in Evanston in 1946, attended parochial schools. Therefore, the proportion of children aged six, seven, and eight years who were attending parochial schools in Evanston in 1946, and their caries rate, must have been very high to permit Doctors Blayney and Hill to state that “The caries rates for the Evanston and Oak Park Public School white children compared favourably.” Of course speculation is no substitute for data-and this still has not been published.

(17) All of this clearly indicates how hidden variables may exist in areas which otherwise appear to be comparable, and how important it is to be certain that comparisons are made between like groups.

Comment. The latter phrase is a reiteration of remarks made in the 1957a report from this study, that it is necessary “to make comparisons of like groups.” Why then, having realized this necessity, did Hill et al. ignore it in their 1958 report (p. 152)? In this report the data shown, for the year 1946, combined not only that of the white children attending both public and parochial schools, but the data of the Negro children as well. The resultant rate was then compared with that of children in Oak Park comprising, almost entirely, white children attending public schools. By ignoring the opinion they expressed in the previous year (1957a)-which they now reiterate-and comparing “unlike” groups of children, a more favourable degree of comparability was obtained between the initial caries rates of children in the test and the control cities.

(18) Dr Sutton expressed astonishment that in 1955 the six and seven-year-old Evanston children had a lower caries prevalence rate than the Aurora children of like ages. We, likewise, did not anticipate this. However, the same critical evaluation both clinical and roentgenological, was made of every case. This difference was due to something other than fluoride. Possibly the presence of the dental team in the school, year after year, has stimulated the classroom teachers and the school nurses to place more emphasis
on the teaching of oral health. Some unknown hidden variation not related to fluorides must account for the difference.

Comment. It is pleasing that Doctors Blayney and Hill should support the view expressed on page 187 (para. 4) that regular dental examinations may stimulate interest in the teeth and thus lead to improved oral health. In advancing the suggestion that “something other than fluoride” can affect the caries rates, they recognize the great importance which factors other than the fluoride concentration of the water supply may have on the caries rates. This extremely important matter was practically ignored by the authors of all these studies when preparing their reports.

(19) Dr. Sutton was concerned that the control group was not examined annually. Neither we nor our advisers could see a reason to require an examination of the control group other than at the beginning and near the close of the study. This provides the baseline from which to measure the trend of the dental caries rate during the time interval (1947 1956). Should the rate in the last examination (1956) deviate materially from that of the initial baseline period (1947) that figure could be used as a correction factor in the Evanston findings. In fact, if we had not desired to measure the yearly decrement in the rate of dental caries under fluoridation and evaluate other factors, only two examinations in Evanston would have been necessary, the first in 1946 (before fluoridation), the second and final in 1961.

Comment. This statement makes two things clear. The first is that, at the commencement of the study, neither the workers nor their advisers could have considered the possibility, which they now acknowledge(18), that “the presence of the dental team in the school, year after year” might have had a stimulating effect “on the teaching of oral health.” It is obvious that, if this effect is possible, not only the test town but also its control should have been examined “year after year”. The other point which is indicated by this statement(19) of Doctors Blayney and Hill is that despite their remark in 1950, the importance of random variation was not and, seemingly still is not recognized.

(20) Much has been made of the variations reported in the number of children examined. The baseline examination of 4,375 Evanston children and of 2,493 Oak Park children are correct. However, the data from those children who had not used Lake Michigan water
all of their lives had to be excluded. It was also observed that some
children below the age of 67 months and above the maximum
of 174 months had been examined. Therefore these out of range
children were not considered in the final determination of the caries
rates. This explains the discrepancy between the Evanston 4,375
and 3,692 and the Oak Park 2,493 - 2,051 figures.

Comment. This explanation of the difference between these sample sizes
in Evanston and Oak Park is welcome. It might have been deduced if the
decision to exclude “the data from those children who had not used Lake
Michigan water all of their lives” had been announced in one of the five
reports giving caries rates, issued prior to 1955 (p. 163).

In the XIX Report (Hill et al., 1959), the sample sizes shown for the two
age groups in Oak Park in 1947 (1,022 and 1,032) are almost the same as
those shown (1,020 and 1,031) in statement “E” of Figure 5. Comparison
with the statements for Evanston cannot be made for this (XIX) report
considered only “public school white children”.

(21) It should be noted that when dental caries experience rates were
compared, the same number of examinations, that is 1,991 for the
six to eight-year-old children and 1,701 for the 12 to 14-year-old
children were used throughout the reports for the combined Evanston
school groups (Public White Parochial and Public Negro).

Comment. It is surprising that the suggestion was made that this statement
should be noted-for it is not correct. The number of twelve, thirteen and
fourteen-year-old children examined in Evanston in 1946 was given in
Tables III, V, VI, VII, VIII, IX and X of the XV Report (Hill et al., 1957a)
as 418, 688 and 595, a total of 1,701. However, in Tables XI and XII of the
same paper different sample sizes for these ages were shown: 414, 692 and
617, a total of 1,723. The same discrepancies were noted between different
tables in the XI Report (Hill et al., 1955) Therefore the figure 1,701 was not
“used throughout the reports for the combined Evanston school groups”.

(22) Dr Sutton on p. 167 of his report (fig. 5, Statement C) calls
attention to Evanston Dental Caries Study Report Number XVII.
Here he points out that only 1,754 six to eight-year old children
and 1,556 12 to 14-yearold children were listed. This particular
report was primarily concerned with differences in sex and race, as
they influenced caries, rather than the effect of fluorides. Therefore
a comparison was made of the dental caries experience rates of white girls to white boys; Negro girls to Negro boys; white girls to Negro girls; white boys to Negro boys; and white children as a group to Negro children as a group. The children were classified into male white, female white, male Negro and female Negro for comparison.

Comment. A curious feature of this XVII Report is that although care was taken in regard to the age, race, and sex of the subjects, no attempt was made to “limit the examinations to continuous resident children” (Hill et al., 1957b). Thus it is reasonable to assume that some children were examined who were not “continuous” residents. Therefore the comparisons mentioned by Doctors Blayney and Hill were made on mixed samples of children some of whom had not ingested fluoridated water, those examined in 1946, and others who had done so for varying periods of up to about eight years. This disregard of the possible effect of the ingestion of fluorides on the caries rates, of some of the children examined, is inexplicable.

(23) As Dr Sutton calls attention to the difference in the number of examinations made in this report when compared with other reports, we wish to point out, in explanation, that it was necessary to not only insure that children of correct age be included but also it was necessary that every examination be clearly classified according to race and sex. There were 236 six to eight-year-old children and 245 12 to 14-year old children excluded from this report because they did not fulfil the requirements for this comparison. These children were included in other reports as no distinction except age was made.

Comment. This explanation why the sample sizes from Evanston shown in the XVII Report (1,754 six to eight-year-old children and 1,556 twelve to fourteen-year-old children) do not agree with those shown in other reports at first appears to be a reasonable one. However, before it is accepted, consideration should be given to two observations. Firstly, if the figures depicted in statements “B” and “C” of Figure 5 which were originally given by Hill et al. and are now confirmed by Doctors Blayney and Hill (20, 22), are accepted as correct, the numbers of children excluded in the two age groups (1,991-1,754 and 1,701-1,556) were 237 and 145, not 236 and 245 as stated by Doctors Blayney and Hill. It is possible that these errors could have arisen in typing the manuscript, but this could not be the case in regard
to the second observation.

This second observation is as follows: In the XVII Report (Hill et al., 1957b) it was stated that “in this report no attempt has been made to limit the examinations to continuous resident children.” Thus it is almost certain that data from both “continuous” and “non-continuous” resident children are included in the total of 3,310 subjects mentioned in that Report as examined in Evanston in 1946. Therefore, to determine the number of children who were excluded from the XVII Report, comparison must be made, not with the number of “continuous” residents of correct age that were examined in 1946 (3,692, see 20) and “were included in other reports”, but with the total number of children (“continuous” and “non-continuous” residents and “out of range”) that were examined in 1946, that is, 4,375.

If this is done, it can be seen that 1,065 of these children (4,375-3,310) were excluded from the XVII Report. This figure includes some “out of range” children, for it was stated that “some children below the age of 67 months and above the maximum of 174 months had been examined” (20) in the baseline examination of 4,375 Evanston children. Nevertheless, unless there were as many as 584 “out of range” children (1,065 = 584 + 481), the actual number of children excluded, because they were not of correct age or could not “be clearly classified according to race and sex”, must have been larger than the figure of 481 given here (236 + 245) by Doctors Blayney and Hill.

(24) It is also pointed out that this report, Evanston No. XVII as noted above, was primarily concerned with comparison of the caries rates of the coloured and white children and not with the effect of fluoridation on dental caries rates. Therefore in this light, this report, No. XVII, should not have been listed under the general heading of fluoridation as Dr Sutton has it listed in his critique.

Comment. The XVII Report from Evanston was not listed as a fluoridation study; it appears in the list of references (as do other papers not specifically concerned with fluoridation) because it was mentioned in the text. It was consulted in an attempt to investigate the confusing matter of the differences in the sample sizes for 1946 and 1947 shown in the various reports.

(25) It is true that a discrepancy in figures published in our paper XVI, Table I and in paper XVIII, Table I are at variance This is due to the operator of the tabulating machine providing the wrong figures for the number of seven and eight year old children examined.
This error was discovered after manuscript XVI was in press and therefore, the corrections could only be made manually in the reprints supplied to the readers who requested them.

Comment. If the errors contained in the XVI Report had been pointed out by providing an additional footnote to Table I in the XVIII Report (which was the next report in which this type of data was published) the reason for the difference between the two sets of figures would have been obvious. It should be noted that, although the source of the errors in the 1955 rates in Table 1, XVI Report was given, no mention has been made of the fact that, in the same table, there are errors in computing the rates for the six to eight-year-old age group in the years 1946 and 1948. Both of these errors were of long standing as they were shown, four years earlier, in the X Report (Hill et al., 1952). These errors were still contained in the XV Report (Hill et al., 1957a).

It can be seen that Doctors Blayney and Hill devoted a considerable part of their review to two matters. The first was the “comparability of the study and the control areas”—which was not questioned (see comment 13). The second was a lengthy description of the comparisons which they made between different groups of children in obtaining the data for the XVII Report. This information was given in almost the same words in that report, which, they stated (24), was not primarily concerned with the effect of fluoridation on dental caries rates.

However, they have not mentioned most of the matters which do directly concern fluoridation and caries prevalence and which were questioned. In fact their comments have touched on matters mentioned in only about a third of the sub-headings used in considering their study. Their meagre explanations have accounted for the presence of some of the errors in one table .... and have supplied a reason for the differences between the sample sizes for the year 1947 in Oak Park, and for the disparity between two of the three sample sizes for 1946 in Evanston (p 165) However, most of the matters mentioned in considering the Evanston study were ignored, even those illustrated by Figures 3 and 4.

It should be noted, therefore, that Doctors Blayney and Hill have not commented on the majority of the errors, omissions and mis-statements mentioned in considering the Evanston study, and almost all of them remain unexplained.
DR R. M. GRAINGER

The third review of this book in the Australian Dental Journal, by DR R. M. GRAINGER, Division of Dental Research, Faculty of Dentistry University of Toronto, was as follows:

Those whose work has been so unfairly criticized might well ask P. R. N. Sutton if he feels his own work is proof that the unimpeachable study can be done(26), or if he would welcome similar scrutiny of his publications.

While we do not claim to be able to answer every question to P R. N. Sutton’s satisfaction (or even our own), in order to help set the record as straight as possible(27) the following are specific comments on points raised by P. R. N. Sutton in his discussion of the Brantford Study. No attempt is made to rationalize why specific workers directed or restricted their research efforts or discussion in any areas other than to comment that they no doubt accomplished as much as they could under the circumstances in which they had to work.

Item 1: Reference to Hutton et al. (1951). The numbers of children of the same age examined in the years 1944 and 1945 were very similar with the exception of the nine-year age group. From the unpublished data released at annual meetings in Brantford the number of children examined in 1944 was 239, and in 1945, 319; making a total of 558 (not 608). The data in Table I (Hutton et al.) were apparently combined by pooling the two years’ results not by averaging the averages. However, the point is rather academic(28).

Item 2: Reference to Ontario Health Department report. The date of water-fluoridation in Tables I and II was given as 1946 through a typographical error but was twice correctly stated to be June, 1945, in the text referring to the Table. The small error in percentage reduction for seven-year-olds was also conceded. These points do not seriously underline the usefulness of the work(29).

Item 3: Re late commencement of National Health and Welfare study and detection of caries protection for young individuals born prior to commencement of fluoridation. Despite the fact that the Department of National Health and Welfare began its control study nearly three years after fluoridation began, much worthwhile information was obtained and the effect of late commencement, if any, was to result in underestimation of the fluoride protection(30).
Item 4: Reason for selection of control cities. It seems clear that Brown (1951) gave adequate reasons for selecting Sarnia and Brantford(31).

Item 5: Re superior dental care in Brantford. The difference in level of dental care between the cities is factual as recorded by Brown (1952). This variation of numbers of teeth classified as F. rather than D. or M. does not fundamentally influence the DMF rate(32).

Item 6: Comparability of rates. As stated under item 5, the dental condition of the children in Sarnia and Brantford differed in 1948 because a lower level of dental treatment in Sarnia resulted in higher tooth mortality. The tooth mortality rates thus differed but it does not follow that the DMF rates differed(33). Brown’s statement (1951) “by 1948 the Brantford data were not greatly different from those in Sarnia” is obviously referring to DMF rates and hence quite valid(34). The differences in oral hygiene are also only remotely related to the DMF rates under discussion(35).

Item 7: Concentration of fluorides. The fluoride content of the Brantford water supply was raised to approximately 1 ppm in June, 1945, and raised to 1.2 in February, 1949(36) The Stratford water fluoride content is believed to have been in the order of 1.3 to 1.6 ppm since 1917 when the wells were drilled. Naturally no analysis for fluoride was available prior to the beginning of the interest in fluoridation and early techniques for analysis were not as reliable as present methods(37). These facts have been recorded in the writings of the primary workers (Hutton et al., 1951; and Brown et al., 1951, 1952, 1953, 1954, 1956) and the differences in amounts from other writers might seem less “strange” if they were merely acknowledged to be minor misquotations(38).

Item 8: Re statement by Brown et al. (1956). The statement is substantially correct with the exception that Brown’s observational period did not begin until 1948 hence is less than 10 years(39). The decrease in mean df rates for the 9-11 years group in Sarnia between years 1948 and 1954 (Brown, 1956), did not continue into 1955(40). There was a highly significant decrease over the period 1948 to 1955 (2.37 to 1.93) in Brantford and no significant decrease in Sarnia (2.50 to 2.31)(41). In the same periods the mean df rates for this age [in] Stratford remained nearly equal (1.66 and 1.65) and increased for other ages (42).

Item 9: Re Table II: Ontario Department of Health Report. The printing of dashes rather than percentages for the control cities was to avoid confusing the table with “negative reductions” and in the case of the 9 to 11 df figure
to avoid emphasising what was considered to be a spurious decrease(43). This judgment was borne out by the 1955 figures(44).

Item 10: Differences in reported rates between examiners. Different examiners give characteristically higher or lower rates upon examining the same individuals due to differences in skill, training the physical condition. Thus the differences quoted are no reflection on the design of the experiment or the care taken in the work. The strength of the double examinations comes through corroboration of caries trends in Brantford over the years and not through interchangeability of data(45).

Item 11: Significant fluctuations in controls. The important point is that for the controls the inter-year changes were upward trends or mere fluctuations (even though in some cases calculated to be beyond change), whereas in Brantford the change took the form of a highly significant continual downward trend(46).

Item 12: Larger percentage changes in control. There is no definite explanation as to why rates increased in Stratford and also in Sarnia over the ten years, but this may be a reflection of a general post-war increase in dental caries which has been seen in other areas. However, it is significant that in the various fluoridation experiments, e.g. in Brantford, Newburgh, Grand Rapids, etc., the shift has always been significantly downward in the fluoridated cities whereas the control city rates have remained about the same or in the case of Stratford, increased(47).

Item 13: Smaller percentage decrease after longer fluoridation. As pointed out by Sutton himself on page 168 (middle paragraph) the fluoride protection for permanent teeth of the children aged six to seven seemed to occur within two or three years after fluoridation began. Thereafter the yearly DMF rates were subject to random fluctuation and the differences in percentage decrease of 69 per cent and 51 per cent are most likely a reflection of this inter-year variation(48).

Item 14: The quotation from the Ontario Government Report is taken out of context from a series of summary statements. The previous statement was to the effect that in Brantford there had been a significant decrease of about 60 per cent in DMF rates. In the statement following, as picked out by Sutton, it was stated that “no change” occurred in Stratford and
Sarnia. It should be clear enough from the context that the words “no similar downward change” were inferred.

Item 15: Possible weighting effect. The critic is referring to a possible shift in age distribution within the group, e.g. a possible sampling shift within the six to eight-year age group so that certain years had disproportionately higher or lower numbers of eight-year-olds and hence higher or lower average caries scores. This is rather remote in that selection methods used by Dr Brown were the same each year, moreover very large shifts in age distribution would be needed to produce the significant differences to which P N. R. Sutton refers.

Commentary on the Review by Dr R. M. Grainger

(26) Dr R. M. Grainger raises the question as to whether “the unimpeachable study can be done”. This is, of course, unlikely. It is precisely for this reason that all papers (and these include my own) which set out to present new knowledge should be examined, in order to reduce the chance that findings which are not soundly based will be accepted at their face value. This is particularly necessary in those studies which may involve the health of the public.

(27) The result of Dr Grainger’s attempt “to help set the record as straight as possible” will be judged after considering his other remarks.

(28) Item 1, Reference to Hutton et al. (1951); The numbers of children of the same age examined in the years 1944 and 1945 were very similar with the exception of the nine-year age group. From the unpublished data released at annual meetings in Brantford the number of children examined in 1944 was 239, and in 1945, 319; making a total of 558 (not 608). The data in Table I (Hutton et al.) were apparently combined by pooling the two years’ results not by averaging the averages. However, the point is rather academic.

Comment. The phrase “making total of 558 (not 608)” suggests that the figure 608 was an error in this monograph. This is not the case, in fact this figure was not mentioned. It was published by the authors of this study, Hutton et al., in 1951 (Table 1, column 2). Dr Grainger, therefore, is suggesting that
the total 558 children (derived from the unpublished figures of 239 and 319) is correct, and that the figure of 608 children examined, published by the authors of the study, is incorrect. It should be noted that, five years after this figure of 608 was first published, in Table II, column 2, of their final report Hutton et al. (1956) again published their figure of 608. In both the tables in which it appears it has been used in computing the def and the DMF rates. Also, if one accepted Dr Grainger’s figure of 558 as the correct number of nine-year-old children examined in these two years, the impossible situation would also have to be accepted in which the number of these children with decayed, missing or filled teeth, which Hutton et al. (1951) gave as 595, would exceed the number of children examined.

(29) Item 2: Reference to Ontario Health Department report. The date of water-fluoridation in Tables I and II was given as 1946 through a typographical error but was twice correctly stated to be June, 1945, in the text referring to the Table. The small error in percentage reduction for seven-year-olds was also conceded. These points do not seriously undermine the usefulness of the work.

Comment. The “small” error in percentage reduction, which, Dr Grainger said “was also conceded”, was the showing of 51 per cent instead of 66 per cent (p. 167). Dr Grainger does not mention here the substitution of dashes for figures in the two cases of reduction in the caries rate in the control cities (pp. 4, 37, 44). Several types of errors are present in Tables I and 11: (a) two omissions, which Dr Grainger implied-Item 9 of this review(43)-were made deliberately; (b) two typographical errors; (c) two arithmetical errors (Table I, age 7, “% Reduction Since 1944-45” in the caries attack rates should be 66, not 51, and in Table II, age 10, the “% Reduction Since 1944” in the caries attack rates should be 18, not 61); and if, as appears likely, the figures given by Dr Grainger in Item 1, of this review(28), are incorrect and were used, (d) four incorrect mean figures.

Dr Grainger contends that the points which he mentioned “do not seriously undermine the usefulness of the work”; but the occurrence, on one page alone, of all the errors and omissions which have just been mentioned certainly undermines confidence in the care taken in the preparation of this official report by the anonymous “statisticians” of the Division of Medical Statistics, Ontario Department of Health.
Item 3: Late commencement of National Health and Welfare Study and detection of caries protection for young individuals born prior to commencement of fluoridation. Despite the fact that the Department of National Health and Welfare began its control study nearly three years after fluoridation began, much worthwhile information was obtained and the effect of late commencement, if any, was to result in underestimation of the fluoride protection.

Comment. Dr Grainger does not state the nature of this “worthwhile information” but, whatever it was, it could not compensate for the lack of a pre-fluoridation caries assessment in this study. Its late commencement could be justified only if it was known that the caries rates in Brantford had not been affected by the ingestion of fluorides prior to the baseline examination (p. 168). However, the results from the City Health Department study, if taken at their face value, indicated that there had been marked and erratic changes: at first a considerable rise in the DMF rates after about one year of fluoridation, followed by a marked fall during the second year. It is surprising, therefore, that, out of all the cities in Canada, Brantford was chosen as the location of two long-term studies, for it should have been obvious that the value of the second study would be severely limited by the fact that the very important data showing the pre-fluoridation caries rates could never be obtained.

Item 4: Reason for selection of control cities. It seems clear that Brown (1951) gave adequate reasons for selecting Sarnia and Brantford.

Comment. As Dr Grainger notes, this paragraph refers to the selection of the control cities, which were Sarnia and Stratford—not “Sarnia and Brantford”.

The sole reference to the selection of control cities which Brown (1951) gave is as follows: “The Ontario Dental Division, under Dr Frank Kohli, volunteered assistance, as did Dr G. L. Anderson, Medical Officer of Health for Sarnia, and Dr H. B. Kenner, Medical Officer of Health for Stratford, and both these cities entered the study as controls. (The water of Sarnia is fluorine-free, and that of Stratford contains 1.3 ppm. of fluorine from a natural source.)” Dr Grainger considers that “Brown (1951) gave adequate reasons” for the selection of the control cities—but few would agree with him.
(32) Item 5: Re superior dental care in Brantford. The difference in level of dental care between the cities is factual as recorded by Brown (1952). This variation of numbers of teeth classified as F. rather than D. or M. does not fundamentally influence the DMF rate.

Comment. Increased dental care usually includes some prophylactic treatments and, as noted by Doctors Blayney and Hill (18), even regular examinations may be accompanied by “more emphasis on the teaching of oral health.” This statement by Dr Grainger implies that he considers that such increased dental care has no influence on the total DMF rate.

(33) Item 6: Comparability of rates. As stated under Item 5, the dental condition of the children in Sarnia and Brantford differed in 1948 because a lower level of dental treatment in Sarnia resulted in higher tooth mortality. The tooth mortality rates thus differed but it does not follow that the DMF rates differed.

Comment. It does not state “that the DMF rates differed; it points out, as its title states, the “Doubtful comparability of rates” owing to the delay in setting up this study. Dr Grainger’s comments suggest either that he has not understood the meaning of the first sentence of the paragraph, or that he is seeking to distract attention from the presence of this important deficiency in the study-its late commencement.

(34) Brown’s statement (1951) “by 1948 the Brantford data were not greatly different from those in Sarnia” is obviously referring to DMF rates and hence quite valid.

Comment. This quotation does not appear in Brown (1951) but a similar statement was made by Brown et al. in 1953 and 1954 (b) and is given on page 169. The fact that it was “obviously referring to DMF rates” was not questioned. The implications of this remark were discussed.

(35) The differences in oral hygiene are only remotely related to the DMF rates under discussion.

Comment. Brown et al. (1954b) said that “marked differences in oral hygiene as between the test and control groups might conceivably affect the findings”. Such “marked differences” were reported-but were disregarded (p 41).
(36) Item 7: Concentration of fluorides. The fluoride content of the Brantford water supply was raised to approximately 1 ppm in June, 1945, and raised to 1.2 in February, 1949

*Comment.* This statement of Dr Grainger is welcome because it provides the answer to the question: Which of the statements regarding the concentrations of fluorides in the Brantford water, which were reported on page forty-two, are accurate and which ones are not?

(37) The Stratford water fluoride content is believed to have been in the order of 1.3 to 1.6 ppm since 1917 when the wells were drilled. Naturally no analysis for fluoride was available prior to the beginning of the interest in fluoridation and early techniques for analysis were not as reliable as present methods.

*Comment.* This statement is most revealing for it indicates that the wells at Stratford have been analyzed to determine their fluoride content only since “the beginning of the interest in fluoridation”. If this is the case, the statements of Brown et al, (1953, 1956), concerning the “continuous” use of water containing fluorides in concentrations of 1.3 ppm or 1.6 ppm since 1917 are not founded on data and are, therefore, merely different guesses.

(38) These facts have been recorded in the writings of the primary workers (Hutton et al., 1951; and Brown et al., 1951, 1952, 1953, 1954, 1956) and the differences in amounts from other writers might seem less “strange” if they were merely acknowledged to be minor misquotations.

*Comment.* It was pointed out that the “facts” regarding fluoride concentrations were stated differently in these papers. In regard to the concentration in the Stratford supply, a comparison of the statements made by Brown et al., in 1953 and 1956 suggests that the concentration of fluorides in this supply may have increased from 1.3 to 1.6 in this three year period. The important admission that the fluoride concentration in Stratford was obtained only relatively recently, is not contained in any of the six “writings of the primary workers” mentioned by Dr Grainger. Therefore his statement is not correct.

As Dr Grainger suggests, it is not unlikely that the statements regarding fluoride concentration of the “other writers”, the New Zealand Commission of Inquiry (1957) and the Ontario Department of Health (1956), were “minor misquotations”.

(39) Item 8 Re statement by Brown et al (1956). The statement is substantially correct with the exception that Brown’s observational period did not begin until 1948, hence is less than 10 years.

**Comment.** Dr Grainger suggests that the phrase “more than ten years” is incorrect. It was inserted into the quotation of a statement by Brown et al. (1956), but enclosed in square brackets to indicate that it was not a part of the quotation. However, in the sentence which immediately precedes that quotation Brown et al. (1956) said: “Brantford has had more than 10 years of experience with 1 part per million of fluoride in its water supply. During that time... “It is clear that they were not referring to “Brown’s observational period” of about seven years, but to the period of fluoridation in Brantford which commenced in June 1945 (Hutton et al., 1951; p. 173) and was, therefore, “more than 10 years”.

(40) The decrease in mean df rates for the 9-11 years group in Sarnia between the years 1948 and 1954 (Brown, 1954), did not continue into 1955.

**Comment.** The 1955 rate of 2.31 df was still below the 1948 and the 1951 figures of 2.50 and 2.41 respectively.

(41) There was a highly significant decrease over the period 1948 to 1955 (2.37 to 1.93) in Brantford and no significant decrease in Sarnia (2.50 to 2.31).

**Comment.** The decrease mentioned by Dr Grainger (2.37 to 1.93) was reported in Brantford between 1948 and 1954 (Brown et al., 1954b) not “over the period 1948 to 1955”. In 1955 this rate rose to 1.99 (Brown et al., 1956), and the difference between 1948 and 1955 was no longer said to be “highly significant” (Brown, 1955)

The rates quoted by Dr Grainger for Brantford are for the years 1948 and 1954 (see 63). It should be noted that it was between these two years that the maximum “decrease” was reported in the rates in that test city (2.37 to 1.93). Furthermore, in mentioning Sarnia, instead of giving the figures for the same period (1948-54), 2.50 to 2.11, he cited the figures 2.50 to 2.31, which cover a different period (1948-55) and do not reveal (Brown, 1955) the significant “decrease”, in the rate in this control city, which was shown in the previous report (Brown et al., 1954b). By the use of these figures,
the reviewer exaggerates the contrast between the test city and this control. Thus, this statement by Dr Grainger is both inaccurate and misleading.

(42) In the same periods the mean df rates for this age [in] Stratford remained nearly equal (1.66 and 1.65) and increased for other ages.

Comment. The rates for the four examinations were: 1.66, 1.76, 1.58, 1.65 (Brown, 1955). (Throughout this monograph caries rates have been given in the form in which they appear in the original papers although it is recognized that, in cases such as these, the practice of showing caries rates with two places of decimals is, probably, not warranted.) Dr Grainger mentions the least variable of the ten caries rates in the control cities—that for the deciduous teeth of children aged nine to eleven years in Stratford. He omits to mention the DMF rates which show the remarkable situation, in this control city, in which each of the inter-year changes occurring in this age group, and in five out of the six inter-year changes in the rates of the “other ages”, were said by Brown (1955) to be statistically significant.

(43) Item 9: Re Table 11, Ontario Department of Health Report. The printing of dashes rather than percentages for the control cities was to avoid confusing the table with “negative reductions” and in the case of the 9 to 11 df figure to avoid emphasizing what was considered to be a spurious decrease.

Comment. This astonishing explanation, for the printing of dashes in this table, implies that these omissions were made deliberately because the results did not conform to those expected. Why should a decrease of 0.44 df (18 per cent) in the test city be accepted and published, but a very similar one of 0.39 df (16 per cent) in a caries rate in the control city of Sarnia be considered “spurious” and not published—a dash being shown in the appropriate position in the table? By printing these dashes, the “statisticians” of the Ontario Department of Health could have misled their Minister into thinking that there were no changes in these caries rates in Sarnia and Stratford (particularly as the Summary of the report said so) but that there had been a decrease of eighteen per cent in the corresponding rate in Brantford due to fluoridation.

Talk of “negative reductions” cannot disguise the fact that nothing is more
calculated to confuse a table than (as Dr Grainger implies) the deliberate omission by “statisticians” of figures giving the percentage changes in rates (one of which was said to be significant).

(44) This judgement was borne out by the 1955 figures.

Comment. Dr Grainger tries to justify this “judgment” by implying that these so-called “spurious” decreases were not seen in the 1955 figures. However, small “decreases” were still shown in that year, the “reduction” in Sarnia being 7.6 per cent.

In any case, this so-called “judgment”, regarding the omission of data, has no place in the preparation of an unbiased report.

(45) Item 10: Differences in reported rates between examiners. Different examiners give characteristically higher or lower rates upon examining the same individuals due to differences in skill, training the physical condition [sic]. Thus the differences quoted are no reflection on the design of the experiment or the care taken in the work. The strength of the double examinations comes through corroboration of caries trends in Brantford over the years and not through interchangeability of data.

Comment. Dr Grainger refers to the differences between examiners in the assessment of caries rates. This important matter has already been considered. The aim of the paragraph mentioned was to show that, as the rates obtained by the examiners in the two Brantford studies were different, data from the City Health Department study could not be used to decrease “the deficiency in the data of the National Health and Welfare study, owing to its late commencement”. The admission, which is implicit in Dr Grainger’s remark, that “interchangeability of data” was not permissible between the two studies in Brantford confirms the point made. The degree of reliance which can be placed on the “corroboration of caries trends in Brantford over the years” must be considered in the light of the widely divergent results obtained in these studies, which were discussed in the second paragraph of page three.

(46) Item 11: Significant fluctuations in controls. The important point is that for the controls the inter-year changes were upward trends or mere fluctuations (even though in some cases calculated to be beyond change) [sic], whereas in Brantford the change took the form of a highly significant continual downward trend.
Comment. It was pointed out that in the control city of Stratford five out of the six comparisons made between the permanent teeth rates of successive years were said by Brown et al. (1954b) to be significant changes (four of them being at the three standard error level). In the permanent teeth rates in the other control city, Sarnia, there were four highly significant and one significant change in the nine comparisons made. Brown et al. (1953, 1954b) and Brown (1955) said that the odds relating to the occurrence by chance or sampling variation of a difference of the magnitude of three standard errors (such as were reported in eight of these changes) “are 369 to 1 against”. Therefore, when Dr Grainger terms these unexplained changes “mere fluctuations” he is rejecting that remark of Brown et al. and denying the meaning of statistical significance.

Dr Grainger neglected to mention that the “highly significant continual downward trend” in the caries rates in Brantford occurred only in children who were aged twelve to fourteen years. In the two other age groups, in both the deciduous and the permanent dentitions, there was an upward trend in the caries rates in the fluoridated city during the last year of the study, the rise from 0.44 DMF to 0.69 DMF, in the youngest age group, being said to be a highly significant rise (Brown, 1955). Therefore this statement by Dr Grainger, that there was a “continual downward trend” in Brantford, is incorrect and is misleading.

(47) Item 12. Larger percentage changes in control. There is no definite explanation as to why rates increased in Stratford and also in Sarnia over the ten years but this may be a reflection of a general post-war increase in dental caries which has been seen in other areas. However, it is significant that in the various fluoridation experiments e.g. in Brantford, Newburgh, Grand Rapids, etc., the shift has always been significantly downward in the fluoridated cities whereas the control city rates have remained about the same or in the case of Stratford, increased.

Comment. Dr Grainger’s statement, that the “rates increased in Stratford and also in Sarnia over the ten years”, is inaccurate for, as he pointed out in his Item eight (39), “Brown’s observational period did not begin until 1948, hence is less than 10 years.” Sarnia was first examined in March 1948 and Stratford in October of that year (Brown, 1952). Therefore the caries rates of the children in both those towns were known for a period of about seven
years, not one of ten years. Furthermore, in the deciduous teeth in Sarnia, the younger age group showed a higher rate in 1955 than in 1948, but in the older children the final rate was lower than the initial one. In this city the DMF rate rose between 1948 and 1953 but between that year and 1955 there was a decrease in this rate in each of the three age groups.

The suggestion that there has been “a general post-war increase in dental caries” is not supported by these studies, for such a rise it was not seen in any of the unfluoridated control cities considered. In Muskegon and Oak Park there was no definite trend. At the time when (as a result of their being fluoridated) these cities ceased to serve as controls, the rates for the children of some ages were higher, and for other ages they were lower, than during the initial examination. The trend in Kingston cannot be investigated owing to the method of presenting the data in the Newburgh study. The contention that “the shift has always been significantly downward in the fluoridated cities” can be accepted only if the many deficiencies pointed out in this monograph are ignored and the figures from these trials accepted at their face value.

(48) Item 13: Smaller percentage decrease after longer fluoridation. As pointed out by Sutton himself on page 168 (middle paragraph) the fluoride protection for permanent teeth of the children aged six to seven seemed to occur within two or three years after fluoridation began. Thereafter the yearly DMF rates were subject to random fluctuation and the differences in percentage decrease of 69 per cent and 51 per cent are most likely a reflection of this inter-year variation.

Comment. No specific mention was made of “the children aged six to seven” when pointing out the reductions in the DMF rates which were reported to have occurred in the early years of the City Health Department study (Hutton et al., 1951). Some implications of this reported early decrease in caries rates were discussed on pages two and thirty-eight.

When he makes the remark that “the differences in percentage decrease of 69 per cent and 51 per cent are most likely a reflection of this inter-year variation”, Dr Grainger shows that he could not have noted that the increase in the DMF rate in these children, in the fluoridated city, from 0.44 in 1954 to 0.69 in 1955 (so that the “percentage decrease” dropped from 69 per cent
to 51 per cent), was shown by Brown (1955) to be, statistically speaking, a highly significant (three standard error level) rise in the rate in the test city during the final year of the study. Therefore, when he makes this suggestion, he is expressing an opinion which contradicts the notation given by Brown (1955), in his Table IV, which indicates that the rise in rates which produced this percentage “decrease” is statistically significant at the three standard error level.

Dr Grainger may be right—but if he is, Brown’s (1955) indication of statistical significance in this case is incorrect, and the methods used in the National Health and Welfare study for calculating statistical significance must be questioned. In consequence, all statements made in the study regarding significant changes in the caries rates, both in the test city and its controls, become doubtful.

It is of interest to note that the “1955 Report” from this study was released in two different publications. The first, a booklet, was dated November 1955, and was “prepared by H. K. Brown . . . with the assistance of H. R. McLaren... G. H. Josie... and Barbara J. Stewart”. The second publication is a paper by Brown, McLaren, Josie and Stewart published in 1956 in the Canadian Journal of Public Health, no reference being made to the previous publication. The ten tables and the two figures in the body of these reports are the same and the text of both is practically identical. However, there is one important difference: that part of the discussion dealing with the unexplained rise (shown as significant) in the DMF rate of children aged six to eight years in Brantford (and also in the control city with the “optimum” concentration of fluorides in its water), was omitted from the later report (Brown et al., 1956).

The questions should be asked: Why was this very important small section of the original report omitted when it was published in the Journal? Why, in both these 1955 reports, was the smaller percentage “decrease” between the initial and the final caries rates of the youngest age group in Brantford not published?

(49-50) Item 14 The quotation from the Ontario Government Report is taken out of context from a series of summary statements. The previous statement was to the effect that in Brantford there had been a significant decrease of about 60 per cent in DMF rates. In the statement following, as picked out by Sutton, it was stated that “no change” occurred in Stratford and Sarnia(49). It should be
clear enough from the context at the words “no similar downward change” were inferred(50)

Comment. The charge that the quotation mentioned was “taken out of context” is meaningless unless it implies that the sense of the original statement has been altered. So that the reader can judge this matter, the whole of paragraph three (the “previous statement” referred to by Dr Grainger) and paragraph four are reproduced. The quotation given on page 176, which was the one criticized, is shown here in italic type to distinguish it from its context.

The evidence produced by the investigators of the Brantford City Health Department and of the Department of National Health and Welfare, independently show that since the introduction of the fluorine in the water there has been a significant decrease, amounting to approximately 60 per cent in the number of decayed, missing and filled teeth suffered by Brantford school children.

At the same time, it has been established that there has been no change in the already low dental caries attack rates in Stratford which has 1.2 ppm of natural fluoride in its domestic water, or in the relatively high rates for Sarnia which has had virtually no fluoride in its water.”

Dr Grainger suggests that the writers of this report, when they used the expression “no change”, really meant “no similar downward change”. However, as they were members of the Division of Medical Statistics and, presumably, were trained in the very precise science, of statistics, it is unlikely that they would use such an inexact expression. Also, the term “downward change” is a rather clumsy substitute for the word “decrease” which was used in the previous paragraph (para. 3 above). Both these points suggest that Dr Grainger’s interpretation is incorrect.

(51) Item 15 Possible weighting effect. The critic is referring to a possible shift in age distribution within the group, e.g. a possible sampling shift within the six to eight-year age group so that certain years had disproportionately higher or lower numbers of eight-year-olds and hence higher or lower average caries scores. This is rather remote in that selection methods used by Dr Brown were the same each year; moreover very large shifts in age distribution would be needed to produce the significant differences to which P N. R. Sutton refers.

Comment. After reading Dr Grainger’s remarks it may be supposed that it
was said that the inter-year significant changes in caries rates, reported from the control cities, were due to a “weighting” effect. However, if the original paragraph is consulted, it will be found that it is headed “Unexplained Significant changes in controls.” A “weighting” effect was mentioned as a possible explanation for these unexplained changes. This suggestion was made following the failure of the authors (Brown, 1951, 1952; Brown et al., 1953, 1954b, 1956) to advance even a suggestion why these changes occurred. Dr Grainger’s comment leaves these significant changes in the caries rates of the control cities as the authors of this study left them - unexplained.

This reviewer has made it clear that the statements regarding the “continuous” use of water containing fluorides in concentrations of 1.3 or 1.6 ppm since 1917 in Stratford are not based on data (comment 37); and that the misleading omission of figures from the Ontario Department of Health Report (1956) was made deliberately (comment 43). However it has been seen that, although Dr Grainger said (27) that his aim was “to help set the record as straight as possible”, most of his comments, if they had been accepted at their face value, would have had the reverse effect.
Apart from the reviews already quoted above, the only published criticism known to the author is that contained in the Book Reviews section of the January 1960 issue of the *New Zealand Dental Journal*. This was contributed by MR J. FERRIS FULLER, a member of the Dental Research Committee of the New Zealand Medical Research Council and a member of the Fluoridation Committee of the Department of Health, whose submissions to the New Zealand Commission of Inquiry (1957) are mentioned in over twenty paragraphs of its report.

Mr Fuller’s review stated:

“Everyone is out of step except our Albert,” or so the author would have us conclude. Altogether an extraordinary book; clever but unfortunate; skilfully contrived and yet-stripped of its finery—rather slender. It could be ignored if the matter rested within the Sciences; but since by the very nature of the subject it takes us into the public forum, some of the errors must be stated.

Part I of this book (Fluoridation: Errors and Omissions in Experimental Trials) is a reprint of a paper by Sutton and Amies(*) that appeared recently in the Medical Journal of Australia criticising the Brantford-Sarnia-Stratford study in Canada(52). But the authors have omitted to read the literature(53), and their criticisms therefore are not based on the known facts. This is a serious matter especially when the comments come from two critics who exalt themselves above fellow scientists of at least equivalent status in other parts of the world. They accuse the Canadian workers of failing to devise a randomisation procedure that would eliminate bias(54), of deliberately omitting vital information in some of the tables(55), and finally of displaying bias in the presentation of results(56). Their comments are based on a report of the Ontario Department of Health (1956) to the Ontario Minister of Health, a report obviously written in simple abbreviated terms for public consumption(57). Sutton and Amies failed to read two official publications readily available(58), namely, a 51-page booklet “A suggested methodology for fluoridation surveys in Canada” and the 35-page detailed report of the Department of Health and Welfare, of November, 1955. These two booklets together show that great care was taken to introduce a well-designed randomisation procedure(59), that examiner variability was eliminated as far as humanly possible by the employment of one examiner only throughout the whole period of the study(60), and that the information

* See footnote, p. 1
alleged to have been omitted is in fact shown in detail in the tables in the 1955 report (61), together with the standard error for each of the indices used. In short, the more important criticisms that appear so damaging are in fact without foundation. Thus, when the authors say that “what must be eventually a statistical study does not appear to have been designed as such” and “no attempt at statistical evaluation has been considered” their comments are absurd and, indeed, irresponsible (62). The full official report on the Brantford study was available in New Zealand, incidentally, when the Commission of Inquiry held its hearings (63), and three of its tables are included in the published report of the Commission.

In Part 2 of the book Sutton continues in the same vein. He complains that misleading comments are made in some reports, yet his own book contains many misleading statements. For example, he claims that a proper evaluation of examination errors at Grand Rapids has not been carried out (64), and he doubts the accuracy of caries attack rates in test and control areas because X-ray examinations were incomplete or absent (65). It is significant that he omits to refer to a report by Hayes, McAuley, and Arnold published in the U.S. Public Health Report in December, 1956, which is a key reference in this subject (66). This report met the specific point that “some observers have suggested that X-rays are essential to determine the efficacy of caries control measures” and an investigation was undertaken “to determine whether or not supplementing direct observation with X-ray examinations would affect the conclusions based on direct observation alone.” The conclusion was that supplementary X-ray examinations supported the clinical findings and did not change the basic observation that substantial decreases in dental caries occurred during the test period. The very standard errors that Sutton demands for a proper statistical evaluation were available in this report (67).

He quotes a subsequent (1957) paper by McAuley that suits his book and, in the light of his criticisms and allegations, this makes the omission of any reference to the 1956 report more damaging (68). To borrow his own phrase, omissions of this nature render his work “open to doubt.” Sutton criticises his overseas colleagues for their inability to examine children in control towns prior to fluoridation (69). With personal experience of a study of this nature he would appreciate that where on the one hand the interests of a large number of people and their local bodies and institutions are concerned as compared with only one or two examining personnel on the other, it is almost impossible to operate a plan to the exactitude dreamed of at the statistician’s desk. In any event, the criticism is rather meaningless as far as the Grand Rapids study (70) is concerned when we realise that the baseline
examination in the control city of Muskegon showed that caries prevalence in that city is of the same order as in Grand Rapids.

In attacking the Evanston-Oak Park study, Sutton bemoans the lack of information about the design of the study and phrases such as “It is not clear...”, “It is not understood...”, (It) was not stated...” give the lead to questions and speculations that follow. But why not adopt the simple expedient of writing to the workers concerned and so finding out instead of speculating? This attitude is typical of the book(71). And typical also is the quibbling over details that do not detract one iota from the part that fluoridation has played in these areas in reducing dental decay(72). “The total tooth surfaces considered... should be 58,325, not 58,352” says the author, and also... the mean of these values for 1946... is 150.09, not 149.76”(73). Dear me, Dr Sutton, how dreadful.

And then we come to the Newburgh-Kingston study. Prominence is given to the different composition of the waters at Newburgh as compared with the control city of Kingston(74), and this is cited as the reason why the latter is unacceptable as a control. But once again Sutton omits any reference to a key report, that by Dean, Arnold, and Elvove of August, 1942, listing caries prevalence rates in communities where the variables in the domestic water mentioned by Sutton varied to a greater degree than between Newburgh and Kingston without caries prevalence being markedly affected(75).

The author complains of bias in the manner in which some results are presented but, as can be seen, he displays bias himself in the choice of articles he quotes(76) and in his omission to read others. It is not surprising, therefore, to see him fall into the familiar pattern of the anti-fluoridationist. Those who question fluoridation are given the familiar title of “eminent authorities,” a distinction not afforded anyone else(77). It is surprising, however, to see him serve his ends by quoting Feltman’s study on the use of fluoride tablets. This study lacks the very control that one would expect Sutton to consider essential(78).

As one would expect, there are no bouquets for the New Zealand Commission of Inquiry, one complaint being that “no mention was made of the employment of a statistician to assist its members in evaluating numerical data.” Had the author inquired, he would have been told that the Professor of Biochemistry on the Commission was well versed in biometrics, and that scientific witnesses quickly discovered that tables were unacceptable unless they contained complete details including standard errors, so that he could evaluate data statistically for himself and the Commission(79).
Finally, a warning to those reading this book, lest they be misled by the polemics and the array of figures. Please note that Sutton’s conclusions in part 2 (which forms the greater part of the book) are confined to variations in the prevalence of dental decay in control cities and not to the cities where fluoride has been added(80). What of the places where fluoridation has been adopted? Sutton does not dispute the fact that the prevalence of dental decay has been substantially reduced in the fluoridation cities of Grand Rapids, Newburgh, Brantford, and Evanston(81), nor does he mention that these good results have been confirmed by several independent studies in the U.S.A., and also in Tasmania, Brazil, Japan, Germany, Sweden, and at Hastings in New Zealand(82). The validity of the results from Hastings, incidentally, has been checked by the Applied Mathematics Laboratory of the New Zealand Department of Scientific and Industrial Research(83).

The anti-fluoridationists will rejoice with fresh ammunition to replenish their stocks; but it is unlikely that this work will serve any useful purpose in scientific circles despite the author’s rather pretentious hopes. The performance is almost as old as Time: “The mountains are in labour, there will be born a ridiculous mouse,” said the ancient poet.

Commentary on the Review by Mr J. Ferris Fuller

(62) The charge made by Mr Fuller that ‘their comments are absurd and, indeed, irresponsible’ will be considered first, partly because of its serious nature and partly because it sets the standard for his criticisms.

This charge is made by misquoting parts of two sentences (para. a, p. 4). In the second misquotation the word 'comprehensive' was omitted by Mr Fuller, thus completely distorting the meaning of the original sentence.

Fortunately, the fact that Mr Fuller gives several other 'quotations' from the first edition which are not completely accurate permits the interpretation that the omission of the word 'comprehensive' is due to Mr Fuller having read the monograph only superficially prior to publishing his review. If this is not the case, the more unfortunate conclusion must be fared: that he deliberately made this omission in an attempt, by the use of misquotation, to discredit the statements of those whose findings contradict his own beliefs.
Despite the fact that a study of Parts One and Two of this monograph will show the true nature of most of the remaining points raised by Mr Fuller, in order to avoid the possible suggestion that the objections which he raised have not been refuted, some comments will be made on them.

(52) Part One mentions the experimental trials which have been conducted in Brantford, Canada, and in Grand Rapids, Newburgh and Evanston, U.S.A. (p. i). The comments made in it were not confined, as this reviewer infers by his comments and by here ignoring the other studies, to 'the Brantford. Sarnia-Stratford study in Canada'.

(53) In regard to the remark of Mr Fuller that 'the authors have omitted to read the literature' it may be noted that the brief paper (Part One), which he is criticizing in this paragraph of his review, contains references to seven of the original papers which deal with the caries rates from these studies-more than were mentioned in the entire 'bibliography' of the report of the New Zealand Commission of Inquiry (1957).

(54, 59) Mr Fuller said 'They accuse the Canadian workers of failing to devise a randomisation procedure that would eliminate bias'. The original statement (p. i) refers to 'bias on the part of the examiners' and is not restricted to the studies conducted by 'the Canadian workers'. This reviewer says (59) that 'These two booklets together show that great care was taken to introduce a well-designed randomisation procedure'. However the only randomization procedure mentioned in these booklets was related to the sampling process and was used to determine whirls children should be included in the study-it had nothing to do with the elimination of examiner bias. In order to eliminate such a bias it is necessary that the examiner does not know whether each of the children he is examining belongs to the test or to a control city (p. 9). As the examinations in Brantford and its control cities were conducted at different times (p. 43) it is obvious that suitable precautions were not taken to eliminate examiner bias.

(55) The only omissions of information from tables which were mentioned in Part One (p. 4) were the printing of dashes in the Ontario Department of Health Report (1956). Care was taken (p. 4) not to make the suggestion that these Canadian workers were 'deliberately omitting vital information in some of the tables', but Dr Grainger's remarks (Item nine; 43) indicate that
the omission of these figures from the Ontario Department of Health Report (1956) was deliberate.

(56) As these omissions were made deliberately, the accuracy of the statement, made on page two of this monograph, that 'Bias is suggested by the presentation of some results', is confirmed.

(57) In considering the studies in Brantford, reference was made to two of the original papers as well as to the figures contained in the tables of the Report of the Ontario Department of Health (1956). This Report was 'A Report to the Minister of Health' and was 'Prepared upon his request by The Division of Medical Statistics'—it was not, as Mr Fuller submits, written 'for public consumption'. Even if it had been, does he suggest that basic figures presented 'for public consumption' should be different from those shown to any other class of reader?

(58) Thanks are due to Mr Fuller for drawing attention to this 1955 booklet of Brown, for it was not realized that two slightly different reports, both termed '1955 Report', were issued from the Department of National Health and Welfare study in Brantford. Reference has already been made to this booklet (Brown, 1955) when discussing Item 13 of Dr Grainger's review (48).

(60) Mr Fuller says that reports from the National Health and Welfare study in Brantford show 'that examiner variability was eliminated as far as humanly possible by the employment of one examiner only'. However, within-examiner variability remains, and (p. 2) neither within nor between-examiner variability was estimated in this or any of the other studies considered.

(61) Mr Fuller does not specify the 'information alleged to have been omitted' to which he refers here. As he is speaking of Part One, it is assumed that he means the statement made (p. 2), in regard to the five trials considered, that 'The importance of random variation in the D.M.F. rate (decayed-missing-filled permanent teeth rate) does not appear to have been recognized, or else it has been ignored.' (Other 'omissions' have just been considered under comments 54, 55 and 60.)

In 'the tables in the 1955 report' (Brown, 1955), mentioned by Mr Fuller, the mean caries rates and the standard errors of the mean rates were shown, with notations which indicated the 'levels of statistical significance'. (Similar data were shown by Brown et al., 1953, 1954b.) In the test city,
nine out of the twelve 'Inter-Year' changes in the D.M.F. rates were said to be significant. However, the strange result was indicated that most of the changes in the control cities were also said to be significant (Brown, 1955). In Sarnia, six out of twelve, and in Stratford, no fewer than eight out of nine changes between successive examinations were said to be significant (ten of the fourteen significant changes in these control cities being indicated as being at the three standard error level). In the first '1955 Report' (Brown, 1955) these changes in the caries rates in the control cities were mentioned, but no reference was made to the fact that most of them were considered to be significant. And as was mentioned (comment 48) in referring to Dr Grainger's Item thirteen, in the second '1955 Report' (Brown et al., 1956) all mention of these changes was deleted.

(63) Mr Fuller said that 'The full official report on the Brantford study was available in New Zealand, incidentally, when the Commission of Inquiry held its hearings' (in 1956-7). In that case, and in view of the importance that he appears to attach to this 1955 booklet of Brown (53, 58-61), it is surprising that, as recently as 1959, he ignored it when giving a lecture to the New Zealand Institution of Engineers (Fuller, 1959). On that occasion he said: 'In this age group there has been a 69% reduction in dental decay. The enamel of the teeth of these children has developed under the complete influence of fluoridation and we have a situation the same as that found at Sarnia [air], a situation that verifies the caries/fluorine hypothesis' (Fuller, 1959).

This '69% reduction in dental decay' was shown in the 1954b report of Brown et al., but this very impressive result was a transitory one (pp. 46-7). The chart depicting the D.M.F. rates in this study, which was shown to the Institution, was also taken from that report. Why did Mr Fuller rite the most favourable result from this study and ignore the final report (the booklet by Brown, 1955) which shows that, in these children in Brantford, there was considered to be a highly significant rise in the caries rate during the final year of the study?

(64-7) In Part a of the book Sutton continues in the same vein. He complains that misleading comments are made in some reports, yet his own book contains many misleading statements. For example, he claims that a proper evaluation of examination errors at Grand Rapids has not been carried out, and he doubts the accuracy of caries attack rates in test and control areas because x-ray examinations
were incomplete or absent(65). It is significant that he omits to refer
to a report by Hayes, McAuley, and Arnold published in the U.S.
Public Health Report in December, 1956, which is a key reference
in this subject(66). This report met the specific point that 'some
observers have suggested that x-rays are essential to determine
the efficacy of caries control measures' and an investigation was
undertaken 'to determine whether or not supplementing direct
observation with x-ray examinations would affect the conclusions
based on direct observation alone.' The conclusion was that
supplementary x-ray examinations supported the clinical findings
and did not change the basic observation that substantial decreases
in dental caries occurred during the test period. The very standard
errors that Sutton demands for a proper statistical evaluation were
available in this report(67).

Comment. Mr Fuller seems to suggest that(64) examiner errors for the
Grand Rapids study were given in the paper of Hayes, McCauley and
Arnold (1956)-this is not the case. In speaking of that paper, he stated (67)
that 'The very standard errors that Sutton demands for a proper statistical
evaluation were available in this report.' In the first place, standard errors
were not demanded, indeed they were not even mentioned in the discussion
on the Grand Rapids study.

Secondly, the paper by Hayes et al. (1956) deals with clinical and X-ray
examinations made on small numbers of children, from 'four selected
schools', in 1946, 1947 and 1953. The children were grouped into three
age ranges-five to seven, eight to ten, and twelve to fourteen years. Out of
the 11,012 'continuous resident' children of those ages examined in Grand
Rapids in those three years, only 736 (less than seven per cent) were X-rayed
and included in this study.

This paper showed the caries rates as D.M.F. permanent teeth per child for
only the age range of twelve to fourteen years, a range which was not used
in the other reports from the Grand Rapids study (Dean et al., 1950; Arnold
et al., 1953, 1956). Hayes et al. said that 'Left and right posterior bite-wing
radiographs were made for every pupil. For each fourth-grade child (8-10
years of age), one anterior bite-wing X-ray was made to show the central
incisor teeth'. However, despite that statement, they did not publish any
results for the premolars, canines, and incisors of children under the age of
twelve years.
The three main reports from Grand Rapids (Dean et al. 1950; Arnold et al., 1953, 1956) were concerned with two types of caries rate: the D.M.F. permanent teeth per child and the d.e.f. deciduous teeth per child at each year of age, from four to thirteen years for the deciduous teeth and from six to sixteen years in the case of the permanent ones. None of these rates, nor their standard errors, were shown by Hayes et al. (1956), so that this study was disregarded. It is clear, therefore, that Mr Fuller’s suggestion (61), that the standard errors for these Grand Rapids reports were shown by Hayes et al. (1956), is incorrect and misleading.

Mr Fuller stated (65) that the author ‘doubts the accuracy of caries attack rates in test and control areas because x-ray examinations were incomplete or absent.’ This remark refers to the statement made on page sixteen: that the lack of X-ray examinations ‘must throw considerable doubt on the accuracy of the caries attack rates’. This remark is borne out by results published by Blayney and Green (1952) and by Ast et al. (1956). Also, in the paper cited by Mr Fuller (Hayes et al., 1956) it was stated that ‘The combined technique, direct observation plus bite-wing roentgenography, consistently yields a higher estimate of caries prevalence than direct observation alone’. This remark, far from disagreeing with the statement made on page sixteen to which Mr Fuller takes exception, in fact confirms its accuracy.

Mr Fuller (66) is confusing two different matters: assessment of changes in caries rates and accuracy of caries rates. He says that it is ‘significant’ that no reference was made to the paper by Hayes et al. In this, attention was drawn to the fact that observers had ‘suggested that X-rays are essential for dental surveys designed to determine the efficacy of caries control measures’. In order that the results from the Grand Rapids study, which were based essentially on clinical examinations, could be regarded as reliable, it was necessary to establish that the absence of X-ray examinations did not invalidate the findings. The aim of the investigation by Hayes et al. was to determine whether different conclusions regarding changes in caries rates in a study would have been reached if each examination had been supplemented by an X-ray assessment instead of using clinical examination methods alone. This is a different matter from that of the accuracy of caries rates which was mentioned in comment 65 and which was the subject under discussion in the second paragraph of page sixteen.
(68) He quotes a subsequent (1957) paper by McAuley that suits his book, and, in the light of his criticisms and allegations, this makes the omission of any reference to the 1956 report more damaging. To borrow his own phrase, omissions of this nature render his work 'open to doubt.'

Comment. Mr Fuller refers to a 1957 paper by McAuley'. It is thought that he means the 1957 paper of McCauley and Frazier which reports on a caries survey in Baltimore. This paper was mentioned because it provided a recent example demonstrating the marked differences in caries rates which may be attributable to examiner variability (p. 9), and because it provided a recent opinion which had a bearing on the action of fluorides (p. 8). Mr Fuller refers to 'the omission of any reference to the 1956 report' of Hayes, McCauley and Arnold. Although a photostatic copy of that report was obtained, prior to preparing the comments on the Grand Rapids trial (pp. 10-15), the data given in it were not mentioned for the simple and, it would have been thought, obvious reason that the type of data considered in this monograph is not mentioned by Hayes et al. (1956) (see comment 67). Therefore it is ludicrous to refer to this omission as 'damaging' and as rendering the work 'open to doubt.'

(69) Sutton criticises his overseas colleagues for their inability to examine children in control towns prior to fluoridation. With personal experience of a study of this nature he would appreciate that where on the one hand the interests of a large number of people and their local bodies and institutions are concerned as compared with only one or two examining personnel on the other, it is almost impossible to operate a plan to the exactitude dreamed of at the statistician’s desk.

Comment. The incorrect statement (pp. 64-5) of the United Kingdom Mission (1953), that ‘Before fluoridation is started the teeth of the children in both [test and control] towns are examined in detail’, was criticized but not the ‘inability to examine children in control towns prior to fluoridation.’ Mr Fuller suggests that the failure of these workers to conduct prefluoridation surveys in the control cities was due to their ‘inability’ to do so. It will be recalled that the consequences of this failure were particularly obvious in the Evanston trial (pp. 16-21; Fig. 3, p. 22). As Mr Fuller must be aware, in his own country there was a similar failure to conduct an examination in
the control city of Napier prior to fluoridating the water supply of Hastings. In the first report from that project (Ludwig, 1958) the caries attack rates in Napier were not published, but it was stated that 'the two cities were not comparable'; as a result, the original plan of this project, to use Napier as the control city, was abandoned.

The Dental Health Division and Research Division of the (Canadian) Department of National Health and Welfare (1952) said that, using only one examiner, 'the examination of, say, 1,600 children spread over, say, 20 schools can be accomplished in a matter of, at most, 4 weeks, including follow-up.' Therefore it is obvious that it was not a question of their 'inability to examine children in control towns prior to fluoridation' but of a lack of appreciation of the necessity for such a procedure (p. 65). If this necessity had been recognized, it would have been illogical to jeopardize such long-term experimental studies by failing to delay the commencement of fluoridation by the short period required to assess the caries rates in the children in the control area.

(70) In any event, the criticism is rather meaningless as far as the Grand Rapids study is concerned when we realize that the base-line examination in the control city of Muskegon showed that caries prevalence in that city is of the same order as in Grand Rapids.

Comment. Mr Fuller mentions the Grand Rapids trial—the trial which showed the best comparability of caries rates between the test and the control cities. He ignores the 'smoothing' of the initial rates in the Newburgh-Kingston trial (pp. 54-5) and the fact that, at the time fluoridation was instituted in Brantford, the degree of comparability of the rates in that city and in Sarnia, its 'fluoride-free' control city, cannot be established (pp. 39, 44). He also ignores the gross differences found in caries rates during the initial examinations in Evanston and its control city (p. 21; Fig. 3, p. 22).

(71) In attacking the Evanston-Oak Park study, Sutton bemoans the lack of information about the design of the study and phrases such as 'It is not clear - - .', 'It is not understood - - .', '(It) was not stated - - - ' give the lead to questions and speculations that follow. But why not adopt the simple expedient of writing to the workers concerned and so finding out instead of speculating? This attitude is typical of the book.

Comment. The ten reports from this study which dealt with caries rates, a
total of more than sixty-five pages in journals, provided ample space for
the authors of this study to publish details both of their methods and of the
results they had obtained. It was felt, therefore, that data which they had not
published during the twelve years which had elapsed since it was obtained—
such as the caries rates of the younger children attending the different types
of school (p. 34) and the caries rates of the deciduous teeth in Oak Park
(p. 24)—would, almost certainly, not be disclosed in correspondence. The
reasonableness of this assumption has been borne out by the failure of
Doctors Blayney and Hill, in their review of this monograph, to mention
these, and other, omissions which were pointed out.

(72-3) And typical also is the quibbling over details that do not detract
one iota from the part that fluoridation has played in these areas
in reducing dental decay(72). ‘The total tooth surfaces considered … should be 58,325, not 58,352’ says the author, and also ‘… the
mean of these values for 1946 ... is 150.09, not 149.76’(73), Dear
me, Dr Sutton, how dreadful!

Comment. Errors in the tables of a research report may inadvertently appear,
but should be rare, and they should be reported and corrected as soon as
is practicable. To refer to such errors as 'quibbling over details' indicates
that Mr Fuller does not realize, or in this ease does not admit, the need for
accuracy in a research report.

The first of the numerical errors mentioned was present in Table XII (Hill
et al., 1957a). Mr Fuller omits to mention that, in the same table, the D.M.F.
rate per hundred surfaces for the fourteen-year-old children in 1946, which
was shown in a previous paper (Hill et al., iy) as 15.09, was altered to 15.92.
As a result of this small change in the caries rate, the percentage 'Differences
from 1946' were increased from 1.78 to 6.8 (6.8 should read 6.91) in 1949,
and from 7.62 to 12.44 in 1952 (p. 23). No explanation for this change was
given—indeed, the fact that a change had been made was not mentioned.

In spite of Mr Fuller's opinion to the contrary, this is a clear example
why even small errors in the caries rates should be noted. Small errors or
changes in these rates may produce marked changes in the results reported
when these are expressed as percentage changes—as is the case in all these
studies.

The second error referred to by Mr Fuller, the incorrect rate of 149.76,
was originally pointed out on page thirty-one of this monograph. It is the
smaller error of two which arose when the authors of this study altered their original results (p. 30), and is an error in computing the amended rates (p. 31) in the 1952 report of Hill et al. This incorrect figure was repeated in the 1954, 1956, and 1957a reports.

(74-5) And then we come to the Newburgh-Kingston stud'. Prominence is given to the different composition @1 the waters at Newburgh as compared with the control riss of Kingston(74), and this is cited as the reason why the latter is unacceptable as a control. But once again Suitos omits any reference to a key report, that by Dean, Arnold, and Elvove of August, 1942, listing caries prevalence rates in communities where the variables in the domestic sealer mentioned by Sutton varied to a greater degree than hitween Newburgh and Kingston without caries prevalenie being markedly afferted(75),

Comment. Mr Fuller suggests that the variables in the domestic water are of little importance in a fluoridation study, and, therefore, that the differences (p. 48-9; Fig. 7, p. 49) between the water supplies of Newburgh and Kingston are unimportant. In support of this contention he cites the findings of Dean, Arnold and Elvove (1942). However it is clear that his opinion was not held by Dr Ast, the senior author of the Newburgh study, in 1943 (p. 49) when he emphasized the importance of the comparability of the ‘chemical composition of past and present water supply’. Nor was it shared by the American Water Works Association (1949) when they stated that the experimental verification of the fluoride-dental caries hypothesis ‘obviously necessitates the use of a nearby “control” city with a water supply comparable in all respects to that to which fluoride is being added.’

It should be noted that not only was the composition of the Kingston water considerably different from that of Newburgh (pp. 48-9; Fig. 7, p. 49) but the authors (Ast et al., 1950) ignored this fact and said (p. 48) that the waters ‘were comparable and have remained so, except for the addition of sodium fluoride in Newburgh's supply.’

(76) The author complains of bias in the manner in which souse results are presented but, as can be seen, he displays bias himself in the choice of articles he quotes and in his omission to read others.

Comment. Mr Fuller has made the assertion that bias was displayed by the
choice of articles quoted. It is assumed that he meant a bias against literature
in favour of fluoridation, but the unreasonable nature of this accusation is
obvious when the following facts are considered: There were sixty-seven
references given in the first edition of this monograph. Thirty-one of these
were to original papers from fluoridation trials, eleven were to papers by
authors of fluoridation studies or by strong proponents of this measure, and
six were official reports. Of the remaining nineteen, thirteen references were
made to statistical or other 'neutral' studies, and only six references were
made to papers which could conceivably be considered to question any
aspect of fluoridation. Moreover, only brief mention was made to these six
papers, about half a page in all, of the seventy-two pages of the text being
devoted to them.

It can be seen, therefore, that the observations made in this study of
fluoridation trials are founded, almost entirely, upon statements made in the
original reports from these trials and by those who advocate this measure.

Mr Fuller's charge, that bias is shown in this monograph, is in direct contrast
to the opinion of the reviewer for the Journal of the Dental Association of
South Africa (15 March 1960) who said that 'The author proves himself to
be completely without bias; although he exposes numerous errors, omissions
and misstatements in this evidence, he does not condemn fluoridation out
of hand.'

(77) It is not surprising, therefore, to see hint fall into the familiar
pattern of the antifloridationist. Those who question fluoridation
are given the familiar title of 'eminent authorities,' a distinction not
afforded anyone else.

Comment. Those termed 'eminent authorities' (p. 5) were: (a) Sir Stanton
Hicks, who, for many years, was Professor of Physiology and Pharmacology
in the University of Adelaide, and Scientific Advisor on Foodstuffs and
Feeding to the Australian Military Forces. (b) Dr Hugh M. Sinclair, Vice-
President of Magdalen College, Oxford, and formerly Professor of Human
Nutrition at that University, and his co-author, Dr Dagmar C. Wilson, the
author or co-author of many original papers on dental caries and fluorides. (c)
The remaining authority mentioned as questioning the safety of fluoridation
(p. 5) was the late Professor Harold K. Box who was, correctly, described
in paragraph 119 of the Report of the New Zealand Commission of Inquiry
(1957) as 'an international authority on periodontal disease'.

It is of interest to note the next paragraph in that New Zealand Report where
mention is made of the paper of Professor Box from which the quotation on page five was taken (Box, 1955). It states: ‘120. Dr Cunningham, Head of the Department of Periodontology at the Otago University Dental School, produced an article published by Dr Box in 1955 in which he stated: “I have never made a survey of gingival and periodontal diseases in any area where the water was naturally fluoridated ... and I have written or published nothing on this subject.” (Dental Digest. 61: 172-April 1955.)’

That statement, read without reference to its context, suggests that Dr Box did not express an opinion regarding the possible effects on the periodontal structures of the ingestion of fluorides. However, in the concluding paragraph of that paper, he stated: ‘At the present time, the available findings on gingival and periodontal diseases, as revealed by survey, are totally inadequate. It is my considered opinion that the artificial fluoridation of water supplies, on a wholesale basis, should not be advocated or adopted until fully sufficient findings show that there are no harmful sequelae from a gingival or periodontal standpoint.’

This paper was entitled ‘Fluoridation and periodontal disease’. It occupied only one page, and the opinion which Professor Box expressed in the concluding paragraph was also shown, in almost the same words, in a summary, its large type, which preceded his paper. As this paper was ‘produced’ and, presumably, read, the opinion which he expressed in such strong terms could not have escaped the attention of the New Zealand Commission of Inquiry (1957). Therefore it is surprising that that Commission, instead of giving prominence to the opinion of Professor Box, whom it recognized as ‘an international authority on periodontal disease’, should fail even to mention his ‘considered opinion’. (78) It is surprising, however, to see him serve his ends by quoting Feltman’s study on the use of fluoride tablets. This study lacks the very control that one would expect Sutton to consider essential.

Comment. Mr Fuller disparages the work of Feltman on the use of fluoride tablets. However, the New Zealand Commission of Inquiry (1957) said that ‘certain preliminary controlled studies by Held & Piguet (1954) in Switzerland and by Feltman (1951) in the United States are promising.’ The paper by Feltman (1956), which was quoted on page sixty-six, was stated to be a ‘progress report’. His findings (p. 66) were mentioned because
data from the Evanston trial were compatible with a continuous and marked decline in the rate of eruption of the first permanent molars during the first four to five years of fluoridation (pp aG-fl). Because Feltman's results were only progress ones, and because the authors of the Evanston study, Hill et al., failed to publish this type of data after 1951 (pp. 27, 66; Fig. 4, p. 27) the suggestion inherent in both these results was treated with reserve, when preparing this monograph, and it was stated: ‘Of course, if fluoridation results in the eruption rate of teeth being retarded . . .’ (p. 66).

It should be noted that no comment was made by Doctors Blayney and Hill (in their review of this book) on this suggestion of a decline in eruption rate, even though it was illustrated in Figure 4 (p. 27).

(79) As one would expect, there are no bouquets for the New Zealand Commission of Inquiry, one complaint being that “no mention was made of the employment of a statistician to assist its members in evaluating [the] numerical data.” Had the author inquired, he would have been told that the Professor of Biochemistry on the Commission was well versed in biometrics, and that scientific witnesses quickly discovered that tables were unacceptable unless they contained complete details including standard errors, so that he could evaluate data statistically for himself and the Commission.

Comment. If this is so, it is surprising that the Commission (New Zealand Commission of Inquiry, 1957) stated (p. 69) that ‘We have found nothing to invalidate the statistics or cast doubt on their reliability.’

(80) Finally, a warning to those reading this book, lest they be misled by the polemics and the array of figures. Please note that Sutton's conclusions in part 2 (which forms the greater part of the book) are confined to variations in the prevalence of dental decay in control cities and not to the cities where fluoride has been added.

Comment. The conclusions in Part Two were not 'confined to variations in the prevalence of dental decay in control cities'. Those 'reading this book' will, no doubt, realize-without this 'warning'—why emphasis was placed on these cities, for it is unlikely that they would not have read (p. 5) the title of Part Two: ‘Fluoridation trial controls: errors, omissions and misstatements’.

(81-2) What of the places where fluoridation has been adopted? Sutton
does not dispute the fact that the prevalence of dental decay has been substantially reduced in the fluoridation cities of Grand Rapids, Newburgh, Brantford, and Evanston (81), nor does he mention that these good results have been confirmed by several independent studies in tin U.S.A., and also in Tasmania, Brazil, Japan, Germans, Sweden, and at Hastings in New Zealand (82).

Comment. The claim that 'the prevalence of dental decay has been substantially reduced' in these test cities, as a result of fluoridation, was questioned in the concluding statements of both Part One and Part Two (Pp. 4, 71). Mr Fuller's statement that these claims were not disputed is incorrect. The fact that he made such a statement supports the conclusion reached in the first comment on his review—that he had read this monograph only superficially.

In December 1958, a list of cities with fluoridation schemes in operation was supplied by the Dental Health Officer, World Health Organization (F. B. Rice, personal communication). In this, no control cities were shown for the fluoridation projects in four of the countries mentioned by Mr Fuller, namely Brazil, Japan, Germany, and Sweden. No control was attempted in the Tasmanian scheme (Brothers, 1956). The control for the Hastings project was abandoned, the reason given for this action being that 'the two cities were not comparable since the basic soil type in the Napier area is different to that in the vicinity of Hastings' (Ludwig, 1958). As all these artificial fluoridation projects were conducted without controls, it should be obvious why they were not mentioned in Part Two for, as its title states, it considers fluoridation trial controls. The meagre results published (p. 6) from these and other projects cannot be said to 'confirm' the results of the main studies on artificial fluoridation—in any case uncertain results cannot be confirmed.

(83) The validity of the results from Hastings, incidentally, has been checked by the Applied Mathematics Laboratory of the New Zealand Department of Scientific and Industrial Research.

Comment. It is frequently not recognized that the validity of results depends not only on the accuracy of the mathematical computations but also on the design of the experiment. In discussing the mechanism of the Evanston trial, Blayney and Tucker (1948) said that 'A study of this nature must have an
adequate control.' The Hastings project has no control for, soon after it was commenced, the control was abandoned.

Sir Ronald Fisher's statement on this subject is of very great importance in considering the results published from all these fluoridation projects (Fisher, igyt): 'If the design of an experiment is faulty, any method of interpretation which makes it out to be decisive must be faulty too.'
CONCLUSION

In this second edition, consideration has been given to criticism published by five men who, by their close association with fluoridation investigations, should be exceptionally well equipped to comment. It should be noted that these criticisms make practically no relevant comment on the points raised concerning the Grand Rapids and the Newburgh trials, nor on most of the matters mentioned by the author in discussing the Evanston trial.

The Editorial in the February 1960 issue of the *Australian Dental Journal* inferred that the first edition was essentially an 'unearthing' of 'typographical errors, slips in arithmetic and minor inconsistencies'. In the light of the comments made on the criticisms published in these book reviews, the reader must decide whether this inference is a true one. It is pertinent to mention that, in an 'Occasional Survey' published in the *Lancet* (March 1960) entitled 'Fluoridation: the present position', it was stated that the first edition showed 'that the American trials claim more and prove less than the published results at first suggest.'

In the final paragraph of Part One, the opinion was expressed that 'It is possible that a case for fluoridation can be solidly based'. However, investigation of the published criticisms that have been reprinted here has considerably strengthened the conviction, which was expressed in the Summary of Part Two, that 'The sound basis on which the efficacy of a public health measure must be assessed is not provided by these five crucial trials.'
REFERENCES


REFERENCES

Canad. dent. Ass. 18, 200-4.
INDEX

Adamson, K. T., 73
Age: correct, 86, 92, 93 distribution (altered) 61, (shift in) 98, 109; group, see Group; not mentioned, 46; range, 1, 87, (changed) 50, 52, 53, 8, (restricted) 61, (selected) 84, 118, (varied) 52
Age of children: 5 years, 6, 57-8; 6 years, 2, 3, 9, 12, 13, 20-31 passim, 52, 57-8, 59, 6, 8, 88, 89, 97, 107; 7 years, 3, 2031 passim, 51, 52, 55-6, 57-8, 62, 8, 86, 88, 89, 94, 95, 97, 99, 107 8 years, 20-31 passim, 52, 55, 56-7, 8, 62, 86, 88, 89, 94, 98, 109; 9 years, 37, 51, 52, 55, 6, 8, 62, 95, 98, 99; so years, 2, 55, 56; 11 years, 12-13, 51, 52, 55, 6; 12 years, 13, 20-31 passim, 52, 55, 56-7, 8, 62, 86, 88, 89, 94, 98, 109; 13 years, 20-31 passim, 52, 5, 6, 103, 104, 105, 106, 107, 108, 109; 'out of range', 85, 91, 93; under 67 months, 85, 91, 93
Aim of fluoridation, 8, 42
American Water Works Association (19), 7, 50, 79, 253
Appleton, J. L. T. 67, 70
Armitage, P. (15), 9
Arnold, F. A. et al. (1953), 2, 10-ii, 12, 13, 20, 28, 118, 119 (1956), 13, 14, 15, 46, 118, 119 Ast, D. B. and Chase, H. C., 48, 0, 52, 55, 57, 60, 61, 68
Ast, D. B. et al. (1950), 2, 38, 48-58 passim, 60, 62, 78, 79, 123; 20, 50-60 passim, 81; (1955), 1, 50, 55, 52, 53, 6, 8, 61-2; (1956), 50, 51, 52-3, 6, 8, 60, 61, 62, 65-82, 119
Aurora (control city), 13, 28-9, 8, 89 Authorities: 'eminent', 113, 124; 'international', 1245; public health, 67; question safety of fluoridation, 124
Averaging of caries rates, 3, 37, 95, 98
Baltimore, 38, 120
Bews, D. C. (iss), 67
Bias: elimination of, 9, III, 115; examiner, r, 2, 9, 51, 68, 72; in choice of articles quoted, 113, 123-4; in presentation of results, iii, 113, 523; not eliminated, I, 68, 72, 115
Black, A. P. (955), II, 13
Blayney, J. R. and Greco, J. F. (1952), 16, 21, 29, 119
Blayney, J. R. and Hill, I. N. (1960), 73, 82, 84-94, 101, 122, 126
Box, H. K., 124; (1955), 125
Brantford (test city), I, 3, 5, 7, 37-47, 64, 6, 68, 69, 73-8 passim, 95-116 passim, 121, 127
Brantford City Health Department, 3, 7, 37-8, 42, 44, 64, 69, 70, 77, 100, 105, 107, 109
Brothers, P. C. (1956), 127
Calcium content of water, 49
'Calibration' of examiners, II, 75, 7980
Canada, 1, 5, 37, 44, 73, 76, 78, 100, 111, 115; free dental services, 39
Canadian Department of National Health and Welfare: Dental Health Division, 73; fluoridation study, 3, 38-47, 6, 68, 69, 78,
INDEX

95, 100, 108, 109, 111, 115, 116, 127

Care, dental, in test city: 'outstandingly good', 16, 39, 68, 87; superior, 96, lot

Caries (dental), 28; and individual teeth, 6; control of, 78, 8, 112, '8; diagnosis of, 75, 79, 80; experience, see Caries attack rates; incidence, 12, 13, 38, 78, 80; prevention of, 5, 77; marked decrease not established, 4, 47, '7, 127; pit and fissure, 26-7, 28, 51; prevalence, 46, 69, 114, 127; prevention of, I; protection, 95, 100; proximal, 29; reductions, 6, 68, 74, 114, 127; risk of, 66; studies, need for analysis of water, 8

Caries (dental) attack rates: absolute drop in, z; accuracy, iiz, 117, (doubtful) 16, 'p; addition of different data, 20, 29, 31, 37, 47, 52, 53; 'adjusted', 52, 6; alterations, 30, 45, 46, izi; amended, 31, 123; and structural theory, 38; appraisal, 9, 16; assessment, 68, io, 121; Aurora, 13, 28, 29, 8, 89; averages of, 3, 30, 37; 'beyond change', 97, 105; changes in, 2, 3, 12, 23, 29, 35, 38, 45, 46, 6, 97, 104, 105, 116-17, 121, 522, (in controls) 12, 20, 24, 44, 55, 66, 85, 97, 100, 106, 110; comparable, 8, 88; comparability, 96, lot, 121, (doubtful) 39, 101, (not known prior to fluoridation) 6; comparisons of, 16, 57, 18, 19, 25, 25-6, 28, 29, 51, 64, 67, 84, 8, 86, 88, 91, 93, (prevented) 5, 21, 51, 53; computation of, 99; considered in selection of schools, 62; contrasts exaggerated, 54, so; 'crude', 52; decimal places and, 104 decreases, 2, 24, 30, 46, 47, 66, 97, 100, 104, io6, 107, .08, 509, 112, 118, (in control), 6, 57, 70, 96, 99, 103, 104, 107, (spurious) '97, 104, 105; delay in publishing, 18, 21, 22, 88; depicted, 57, ii; differences between groups, 2, 3, 12, 17, 19, 24, 25, 29, 31, 35, 36, 41, 44, 45, 46, 47, 52, 75, 80, 84, 87, 88, 92, 96, 98, 101, 103, 109, (initial) 18, 25, 22, 55, 68, 121, (not due to fluorides) 8, 89, 9o; different (computed from the same data) 24, 121, (reported by different examiners) see Variability; divergent, zo; 'downward change' 98, 109; 'downward trend' in, 6o, 97, 105, io6; effect of fluorides on, So, 8, 92, 93, 94, 104; errors in, see Errors; eruption of teeth and, see Eruption of teeth; examiner variability in, see Variability; factors affecting, 7, 41, (unknown) 8, 90 final, z, 25, 8, 91, 107, io8; fluctuations, 46, 97, 105, ,o6, (in controls) 14, 55, 25, 45, 55, 56, 97, io5; fluoride concentrations and, see Fluorides; high (in one school) 21, (in parochial schools) 88; higher with X-ray, '9; identical, 37; in control groups, ii, 16, 19, 20, 21, 24, 25, 28, 29, 43, 45, 53, 54, 55, 57, 8, 87, 88, 89, 96-ITO passim, 113, 121, 126, (not published for ten years) 18, 25, 22, 88, (remained at 'same level') 4, 43, 44, ('unchanged') 12, 13, 14, 15, 47, 104; in deciduous teeth, 24, 35, 5!, 70, 119, izi; in different localities, z; in migrants, s; in school groups, zo, 25, 26, 62, 8, 88, 89, 92, 93; in selection of study groups, 7, 62, 67; incompatible statements, i; increase (ignored) z, (in controls) 96, 97, 104, 106, (postwar) 97, TO; increased by exclusion of Negro data, 89; initial (baseline), 2, 3, II, 13, 17, 59, 21, 22, 25, 28, 29, 39, 53, 54, 8, 87, 88, 89, 90, 107, 108, 113, 121, 122; late determination in control, II, 20, 39, 48, 6; low, 8i; lower (in control) 88, (in Evanston than in Aurora) 28-9, 85, 89, (in fluoridated communities) 76, (in Negro children) 8, 87, 88; mean values, 4, 10, 13, 30, 31, 37, 54, 88, 113, 116, iz; means of mean rates, 30, 31; methods used in determining, see Method; 'minimizing the difference', 62; 'minute differences' in, 24, 25 misleading statements regarding, 4, 24, 43-7 passim; 'nearly comparable', 26; 'no change', 46, 47, 55, 56, 97-8, 104, 108-9; non-publication, 20, 22, 26, 55, 56, 57, 88, 121, see also Data; numerical values, 17, 21, 23, 26, 28, 29, 31, 44, 45, 88, 89, 98, 103, 104, so6, 507, 109, 113, 121, 122, sz; of 'the population', 18; omitted for deciduous teeth, 24; original, 16, 17, 46, (replaced) 30, 123; parity expected, 28; per child, 9, 14, 15, 48, 6, 118, 119; per 100 children, 22, 23, 33, 35, 48, 6, z; per 100 erupted teeth, 9, 33, 48, 52-6 passim, 6; per 100 tooth surfaces, 23, 33, 121, percentage differences, 2, 31, 46, 5, 62, 97, 105, 106, 109, 121, (altered statement) 23, 24, 122; percentage reductions, 3, 4, 12, 13, 21, 23, 26, 37, 44, 46, 57, 97, 99,
136

INDEX

104, 107, 108, 117, ('small’ error in) 95, 99; pit and fissure, 26, 51; postwar rise in, 97, 106, 107; prefluoridation, 12, 13, 16, 59, 20, 21, 29, 37, 100, (cannot be known) 39, 43-4, 66, 68, 100; reduction, 3, 4, 5, 8, 53, 21, 26, 29, 30, 38, 43, 44, 46, 74, 75, 107, 517, 127, ('negative’) 97, 10; reliability of, so; results of, 6, is, 37, 55, (omitted) 46, ('reports of’) 67; rise (initial) 24, 30, ’00, (in test city) 107, inS, 117; similarity in test and control, 8, 17, 39, 64, (not investigated) 6; ‘slightly higher’, 46; 'Smoothing’ of, 54-5, 121; statistically significant changes and differences, 17, 18, 19, 25, 35, 36, 43, 44, 46, 97, 98, 103-10 passim, 516-17, (in controls) 24, 30, 45, 47, 103-10 Passim, 117; trend in, 60, 78, 8, 90, 97, 505, so6, so7; unbiased estimate of, 19; ‘unchanged’, 12, 13, 14, 15; variations in, 3, 9, 23, 30, 35, 47, 68, 97, 507, 116, (in controls) 12, 13, 24, 25, 45, 114, ’26; variability, 2, 9, 13, 14, 15, 30, 45, 6, '04; water variables and, 113, 123; 'weighted’, 29, 47, 52, 53-4, 98, 109, so; X-ray assessment, see X-ray; yearly decrement, 8

Chemical composition of water supplies, 8, 48-9, 70, &53, 123, see also Water supply

Children, 88, 106, 107; age composition, 47, 61; born (prior to fluoridation) 95, ion, (since fluoridation) 46, 47' 53 changes in age range, 50, 52, 53, 58; classification of, 92; continuous resident, 32, 33, 34, 61, 91, 92, 93, ’18; data of, see Data; dental condition in test and controls, 41, 75, 80; developing teeth of, 117; distribution of, and caries rate adjustments, a; eruption of teeth, see Eruption of teeth; excluded from study, 17, iS, 60-61, 84, 85, 87, 91; given fluoride tablets, 66, 80, 113, 125; in 'fluoride-free’ city, 7, 121; movements of, 60; Negro, 17, 18, 20, 25, 26, 31, 32, 33, 34, 84-93 passim; not examined, 84, 87, (prior to fluoridation) 6, 112, 120; number examined, 30-7 passim, 8a, 8, 86, 90-4, 99, 121; of different ages, 52, 53, 56; oral hygiene of, 39, 41, 60, 84, 96, ‘or; 'out of range’, 8, 91, 93; school, 16, 17, 18, 19, 21, 25, 26, 32, 59, 84, 87, 89, 109; selection of, 60, 84-5, 87, 1 15; sex and race, 86, 91, 92, 93; see also Age of children

Cities: fluoridated, 97, 106, 114, 126, 127, see also Test city; fluoride-free, 7, 10, 38, 48, 68, sal; selection of (control) 7, 63, 64, 68, 100, (test) 7, 64, 68, son; socio-economic status, 7, 16, 39, 48, 84, 86-7; test and control, 7, 90, miS, 119, 120, (caries attack rate differences) 21-2, 68, 8, 89, 96, 101, (climate of) 7, 8, 10, 48, 67, 84, (compared) 48, 54, 64, 8, 88, 89, 104, 121, (different soils) 127, (lack of comparability) 21-2, 68, 84, 94, 121, (similarity) 48, 64, 87, 112, 113, 121, (water supplies of) 7, 48, 50, 64, 68, 70, 78, 84, 113, 123; with fluorides from natural sources, 7, 53, 38, 42, 64, 74, 76, 77, 81, 84

City: control, see Control city; test, see Test city; Health Department, see Brantford City Health Department

Climate: and salts ingested, 8; of test and control 7, 8, 10, 48, 67, 84; variations in, 19

Comparisons: abandoned, 45; between like and unlike groups, 89; 'Intercity' and 'Inter-year', not valid, 66, prevented, 51, 66; requirements of, 92; test and control, 18, 39, 51, 85, 86, 88, 89, see also Cities

 Constituents of natural waters, 8, see also Water supply

Control: abandoned, 11, 70, 107, 121, 127, 128; area, see Control city; inadequate, 25; independent not used, 64; late examination in, ill 20, 43, 48, 95, 100, lao; measures, 69; must be adequate, 7, 25, '27-8; necessary, 7, 25, 50, 68, 127, 128; not employed, 37, 64, 72, 113, 123, 527, 128; poor choice, 25; requirements, 7, 49; selection of, 7, 10, 63, 64, 68, 96, 100, 127 suitable, 11, 68; 'the ideal’, 16, 19, 87; unsatisfactory, 49

Control city: caries attack rates in, see Caries attack rates; cited by W.H.O., 127; examinations in, see Examinations; fluoridation of, 11, 12, 14, 15, 20-5, 107, (limits usefulness) 68; 'fluoride-free’, 4, 7, 50, 54, 15, 38, 48, 68, 109; las; 'ideal', 16, 59, 87; late examination, II, 20, 38, 43, 48, 6, 68, 95, 100, 120; 'naturally fluoridated’, 13, 38, 42, 64; necessity for, 7, 25, 50, 68, 527, 128; Negro children in, 31, 32, 88; only white children considered, 32; selec-
INDEX

137

tion, 7, 10, 63, 64, 68, 96, 100; termina-
tion, 7, 70, 107; water composition of, see Water; 'weighting', 29, 53-4; see also Aurora, Control, Kingston, Muskegon, Napier, Oak Park, Sarnia, Stratford
Controlled: defined, 63, 69; fluoridation, 63, 67, 69, 70, 74, 75, 77
Controls, 72, 106; 'adequate community', 80; assessment of adequacy, 6; deficiencies not recognized, 67; doubtful or inadequate, 72; evaluation of; experimental, 63, 75; necessity for, 7, a, 68; not adequate, 68; not used, 70, 527 selection of, 7, its, 63, 64, 68, 96, 100; see also Control, Control city
Cox, G. J. and Levin, Margaret M. (1942), 38
Data: arbitrary selection, 19; baseline, 8, 90; combined, (in age groups) 29, 47, 52-5, 6, 6, see also Age of children, 'Weighting', (Negro and white children) 57, 59, 20, 88, 89; 'correction' of, 18, 8; defects in, s; disagreement between, 94; exclusion of, 17, 18, 19, 61, 84, 8, 86, 89, 90-1, 92, 93, (Negro) 17-18, 84-5, 87, 88, (parochial school children) 17-18, 84-5, 87, 88, (policy of) 17, 88, (unexplained) 17, 19, 88; from epidemiological studies, 79; housing, 87; incomplete presentation, as; 'interchangeability' of, 97, 105; 'judgment' and withholding of, 105; meagre, 22, 24, 33, 6, 63, 6, 71, 127; 'misinterpretation' of, 74; nonpublication of, 18, 24, 27, 34, 44, 51, 55, 56, 57, 63, 66, 82, 87, 88, 89, 121, 522, 'a6; omitted, 17, 19, 61, 72, 82, 96-9 passim, 504, 108, 111, 112, 522, ('deliberate') 104, 505; 'pooled', 95, 98; statistical evaluation of, 4, 112, 1' 4, 118, see also Caries attack rates, Statistics; ten-year delay in publication of, 18, as, aa, 88; unpublished, 21, 63, 88, 95, 98, 99
Dean, H. T., 28, 77
Dean, H. T. et al. (1939), 8; (1942), 113, 123 (1950), 7, 10-11, 28, 38, 77, 78, 118, 119
Deatherage, C. F. (1942) 8, 43
Decay, tooth, see Caries
d.e.f. rate: baseline higher in control, 13; comment on, 58; defined, 12; initial rise in test city, 24; omitted, 24; per child, 14,

119; per 100 deciduous teeth, 57; pre-fluoridation lower in test city than in Aurora, 13; unexplained decreases in control, 57; variations in, 12-14; see also Caries attack rates
Department of National Health and Welfare, see Canadian Department of National Health and Welfare
Design, experimental, for fluoridation study, 7-9, 64, 67, 69, 70, 97, 105, 127, 128, (abandoned) 121
d.f. rate: defined, 44; per 100 deciduous teeth, 55-6; see also Caries attack rates
Disease, periodontal, 124-5
D.M.F. rate: defined, 2; development of, 82; effect of topical fluoride, 2; see also Caries attack rates
Dunning, J. M. (1950), 9

Enamel: formation, a; mottled, 8, 4
Errata in tables, 93, 95, 99, 113, 122, 123; corrected, 13, 94; or errors in computing, 37; see also Data
Error: examiner, is, 97, 99, 105, 118, see also Variability; 'human', 75, 7; sampling, s; standard, 45, sn6, so8, 112, 113, ,s6, 117, 118, 519, 126; 'weighting' as source of, 29
Errors: and omissions, 6, 7, 99, 524, safi; arithmetical, 23, 24, 35, 37, 6, 66, 72, 94, 95, 99, 522, 523, 129 examination, Isa, 517, 118; 'fundamental', 74, 76, 78, 79; in amended rates, 35, 123; in computing, 37, 86, 93, 94, '22, 123; in reports, 6, of judgment, 75, 79, 80; repeated, 31; statistical, 66; typographical, 23, 24, 92, 95, 99, 129; see also Method, Results
Eruption of teeth, 28, 70, 76; and caries attack rates, 80; and fluorides, 76, So, 8, 'a6; delay in, 54, 66, 75, So, 8s, 82, sa-6; odd method of assessment, 28, 54; pattern of, 76, 81, 82; rate of, 65, 82, 126, (progressive decline in) 26, 27, 66
Evanston (test city), i, , 56-36, 47, 48, 52, 58, 59, 64-9 passim, 73-8 Passim, 84-93 passim, 113, 114, 115, 120, 121, 126, 127, 129
Examinations, 45, 52, 61, 8z, 97, 105; annual, 64, 8, (not in control) 25, 65, 90 baseline, 53, 55, 8, 8, 87, 90, 93, 100, io6,
INDEX

112, 113, 121; clinical, I, 9, 12, 16, 28, 29, 45, 50, 51, 53, 5, 8, 79, 84, 8, 89, 112, 118; date commenced, ii, 16, 20, 38, 43, 48, us; final, 8, o; in controls, 25, 32, 45, 6, 90, 115, 121, (late) I,, 20, 38, 43, 48, 6, 68, 95, 100, 120; initial, 52, 16, 35, 38, 39, 40, 41, 50, 54, 6, 88, 107; methods, 9, 16, 121, (changes in) 50, 5s; none made for nine years, 25; not limited to continuous residents, 32, 35, 92, 93; number of, 86, 91, 92, 99, see also Sample size; 'only two necessary', 90; pediatric, 60; post-fluoridation, 20, 21, 24, 27, 31-7 passim, 40, 41, 43, 50-1, 52, 53, 8, 59, 64, 90, 9'; pre-fluoridation, 8, 20, 24, 27, 28, 31, 33, 34-5, 36, 37, 39, 48, 50, 52, 53, 64, 6, 8, 89, 90, 91, 120, see also Examinations, baseline, initial, (necessity for) 8, 65, 87, 121, (not done) 6, 87, 500, 112, 120, 121 regular, 90, 10,; successive, 45, ii; time taken to conduct, 121; X-ray, see X-ray Examiners, 112, ,20; 'calibration' of, 11, 75, 79, 80; changed, 2, 10-11, 50-1, 52; dental hygienists used, 2, 50, 51; dental team, 89, 90 different, 11, 50, 97, 105; only one employed, 111, 116, 121; results reported by, 3, 97, 104, 105, see also Results; several used, 2, 10, 13, 50, 51; subjective judgment of, 9, 80; two used, 50; variability of, see Variability; variations in, 5, Experiment: design of, 7, 9, 64, 67, 68, 69, 70, 97, 505, 121, 527, 128, see also Design; fluoridation, , 6, 67-9, 7, io6, see also Trials; interpretation of, 128; methods, i, 61, 68-72 passim, 95, 98, 122, 127; planning, 112, 120, (requirements) 70 Feltman, R., 80; (1951), 80, 125; (1956), 66, 75, 81, 113, 123, 125, X-ray Examiners, 112, ,20; 'calibration' of, 11, 75, 79, 80; changed, 2, 10-11, 50-1, 52; dental hygienists used, 2, 50, 51; dental team, 89, 90 different, 11, 50, 97, 105; only one employed, 111, 116, 121; results reported by, 3, 97, 104, 105, see also Results; several used, 2, 10, 13, 50, 51; subjective judgment of, 9, 80; two used, 50; variability of, see Variability; variations in, 5, Experiment: design of, 7, 9, 64, 67, 68, 69, 70, 97, 505, 121, 527, 128, see also Design; fluoridation, , 6, 67-9, 7, io6, see also Trials; interpretation of, 128; methods, i, 61, 68-72 passim, 95, 98, 122, 127; planning, 112, 120, (requirements) 70 Feltman, R., 80; (1951), 80, 125; (1956), 66, 75, 81, 113, 123, 125, X-ray Fluoridation: and caries-free teeth, 8; and developing teeth, su; and eruption, see Eruption of teeth; and periodontal diseases, u; artificial, 5, 28, 42, 63, 71, 72, 73, 75-9 passim, 81, 95, 100, 103, 125, (object of) 78, (superiority of) 28-9, (validity of) 76, 8i, 8z; basis of, 74, 75, 76, 79, 129; benefits, 113, 122, (apparent drop in) 3, (requirements for maximum) z, (to deciduous teeth) 57-8; commencement of, I, ii, 50, 54, 57, 64, 95, 97, 100, 103, 107, 120, 121, (date stated incorrectly) 37, 95, 99, 100, (examinations prior to) 6, 120, see also Examinations, (in Baltimore) 38, (in Brantford) 38-9, 43, 68, 103, (in Evanston) 27, (in Newburgh) 48, 6, (in Muskegon) 107, (in Oak Park) 20-I, 107, (initial caries rate in control not known before) II, 20, 38, 48, 65; 'controlled', 63, 64, 67, 70, 74, 75, 77, see also Trials; effect of, 86, 92, 93, 94; efficacy of, 74, 77, 129, (assessment of) 529, ('demonstration' Of) 75, 77, 78, (trials to determine) 77, 78; endorsement of, , 67, 72; experiment, , 65, 67-9, 77, see also Experiment; experimental evidence for, ; hypothesis, 5, 79; in Marshall, 63; literature on, 76, 80, 123, 124, 127; little early effect of, 39; mechanical, 5, 42, 72, 75-9 passim; natural, 4, 13, 38, 64, 78, 79, 109; of control city, ,, 12, 14, 15, 20, 107, (not realized) 11; only dental benefits claimed, 5; other factors ignored, 90; period of, 103; preventive value not proven, 78; principle of endorsed, in; projects, see Projects; questioned, 83, 113, 124; results of, 74, see also Caries attack rates, Eruption of teeth, Fluorosis; safety questioned, 5, 524; shorter period and lower caries rates, 28; see also Trials Fluoride: communities, 76; -dental caries hypothesis, see Hypothesis; experience, 50, 79; protection, 95, 97, 100, 107; salt, measurement of, 63; water-borne, 74 Fluorides: action of, 8, iso; added to water supplies, 7, 12, 13, 16, 42, 48, 50, 61, 64, 75, 76, 77, 123, see also Fluoridation; and eruption, see Eruption of teeth; and fluorosis, 8, 43; application of, 63, 109; commercially available, 76, 77; concentrations of, 5, 42, 90, 96, 102, 103, 109,
INDEX

2, 3, 46, iaa; of interpretation, faulty, 70; of planning an experiment, 70, iia, san; of presentation of results, 5, 45, 46, 53, 107, 122; of selection of children, 84-5, 87, n5; random allocation of test and control, 8, 68; randomization, 1, 18, 111, 115, (not devised) III, iii; sampling, see Sampling method; statistical, a, 3, 4, 6, 8, 9, 10, 12, 20, 23, 28, 29, 44, 46, 48, 52, 56, 72, 80, 95, 98, 112, 114, 118; see also Methods

Methods: altered, 19, 45, 52, 70; examination, 9, 16, 121, (‘calibration’ of examiners) 75, 78, 80, (changes in) 50, 52; experimental, 1, 61, 68-72 Passim, 95, 98, 122, 127; of calculating significance, 108; of treatment of data, see Data; selection, 98, 109; variations in, 50

Migrants, 59, 60, 6

Misquotation, 112, 114, (alleged) 97, 108-9

Mis-statements, , 6, 11-12, &4, 15, 17, 18, 19, 20, 21, 23-4, 25-6, 30, 32-6, 37, 39, 4150 passim, 66, 72, 74, 75, 76, 79-82 passim, 8, 86, 91-109 passims, 111-27 passim

Molars, 26, 27, 45-6, 51, 52, 58, 66, 82

Mortality, tooth, 39, 41, 60, 84, 96, 101

Muskegon (control city, initially, for Grand Rapids), 10-15, 20, 25, 37, 64, 68, 107, 113, 121

Napier, 120, 121, 127

National Health and Welfare Study, see Canadian Department of National Health and Welfare

Nesin, B. C. (1956), 5 (1957), 1

Newburgh (test city), 1, 5, 48-65, 68, 69, 70, 73-81 passim, 97, 106, 107, 113, 114, 115, 121, 123, 127, 129

New Zealand, 114, 127; Commission of Inquiry on the Fluoridation of Public Water Supplies (1957), 38, 42, 63,64,69,81, 103, 111, 112, 113, 115, 117, 124, 125, 106

Oak Park (control city for Evanston), 16-18, 19-22, 24-5, 28, 32, 34-5, 36, 68, 84-91 passim, 107, 113, 121, 122

Omissions: from fluoridation reports, 6, 8, 64-5, 74, 99; from quotation, 114; of composition of water, 8; of data, see Data; of figures, 110, (dashes substituted) 4, 37, 44, 96, 104, 115, (‘deliberate’) 99, 114, 115, 116; of information, 155, 116; of reference to report, 112, 120; of results, 26, 27, 46, 55, 66, 110; to read articles, 113, 115, 123


Opinions: altered, 24, 35, 89; as basis of endorsement of fluoridation, 67; gingival and periodontal disease, 5, 125

Oral: health, 8, 90, 101; hygiene, 39, 41, 60, 84, 96, 101

Population, 18; caries in, 8, 75, 80, 87; centres, 88; of Evanston, 19, 84; of Oak Park, 84; of the U.S.A., 19; meaning of, 19; movements of, 59-60; ‘relatively stable’, 59, 6n, 61; sampled, 19; tooth, 52; unnamed, 61

Procedure: fluoridation, see Fluoridation; method of, see Method

Programmes: educational, 25; fluoridation, 78, (‘controlled’) 63, 69; see also Trials

Projects, fluoridation, 74, 75, 78, 79, 121, &27, 128; see also Trials

Quenouille, M. H. (1952), 8

Questionnaires, 32, 59, 60

Radusch, Dorothea F. (1941), 9

Report: adjustment of caries rate in final, in Newburgh, 5a; final, in Brantford, 46, 62, io8, (ignored) 117; from Brantford, ‘1955’, io8, is6, 117; of ad hoc Committee U.S.A., 77; of Expert Committee of W.H.O., 46, 63-70 passim; of N.Z. Commission, 38, 42, 63, 64, 69, 81, 124, 125; of Short, 76, 8’; omission of data from, see Data; research, 122; to Minister of Health, Province of Ontario, 3, 44, 109, 110, III, 116

Reports: appraisal of, i, 76; errors in, see Errors; examination of, 6; misleading comments on, 112; of results of fluoridation trials, 67, 76, 90; omissions from, see Omissions; preliminary examination of, 6; progress, 50, 125-6; unpublished, 63; ‘weighting’ in, see ‘Weighting’

Residence: continuous, 32, 59, 84, 85, 87, 91, 92, 93; not continuous, 87, 90-1, 92, 93
Residents: continuous, 32, 33, 34, 61, 91, 92, 93, 118; new, in Kingston, 60; not continuous, 34, 35, 90-1, 92, 93; of correct age, 86, 92, 93

Results: accepted at face value, 107; assessed, i; averaged, 37; basic, in control, not known until after fluoridation, ii, 20, 38, 48, 6; combination of (different ages) 29, 52, (prefluoridation) 37, 95, 98; comments on, 6, 66; computation of, 93-4; disagreements between, 23, 86, 93, 94; divergent, 3; false confidence in, 36; gingival, 69; grouping of, ss; interpretation of, 39; most favourable cited, 103-4, 117; non-comparability of, 8, 80; not compatible with theory, a; not published, 8, 64-5, 88, 108, see also Data; of fluoridation, 64, 74, 76, 122, (artificial and natural) 77, (not considered by Expert Committee of WHO.) 67, 69, (questioned) 126; omission of, 26, 27, 46, 55, 66, 110, (pit and fissure) 27, 82; original altered, 30, 123; presentation of (bias suggested) 2, 116, (different methods) 50; progress, 126; reliability of, 6, 72; significance, 35, 36; transitory, 117; uncertain, 127; unexpected, 104; unreliability of, 36; validity of, 114, 127; 'weighting' of, see 'Weighting'

INDEX

Rice, F. B., 127
Russell, A. L. (5956), 13

Safety of fluoridation questioned, 5, 124
Sample size, 32, 86, 91, 92, 94, 95, 98, 99; altered, 61; differences, In, 34, 90-4 passim; discrepancies in, 32-3, 85, 86, 91, 92, 94; statements of, 99, (different) 32-6, 86, 91, 92, ( incompatible) 33-4, 36, 86, 91, 92, 93; variations in, 10, 85, 90
Sampling, 31; by selection, 61-2; error, 13; random, 8; shift in, 98, 109
Sampling method: by selection, to, 61; changed, 8; different in test and control cities, 10, o, 8; in Brantford, 115; in Oak Park, 32; minimized differences, 62; mixed data, 92; unorthodox, 61; variations in, 50
Sarnia (control city for Brantford), 3, 4, 20, 38-47 passim, 68, 96, 97, 98, 100-11 passim, 115, 117, 121
Schlesinger, E. R. et al. (1950), 48, 60; (1956), 60
Schools, 32, 34, 59, 6', 62, 09, las; Negro, &7, 18, 20, 25, 26, 33, 8, 8, 89; of public health, s; parochial, 16, 17, 18, 20, 25, 26, 34, 84, 8, 87, 88, 89; selected, 50, 62, 1,8; white public, 17, 19, 20, 25, a6, 32, 8, 88, 89, 9; see also Groups of children
Selection: in sampling, 61-2; of children, 60, 84-5, 87, 115; of controls, 7, so, 63, 64, 68, 96, ion; of data, 7, 19, 64, 68; of schools, 50, 62, 68; of test cities, 7, 64, 68, 100
Sequelae, periodontal, 5, 125
Short, E. M. (1944), 81, 82
Significance, statistical, 3, 17, 24, 25, 26, 29, 30, 35, 36, 43-7 passim, 54, 68, 96, 97, 98, 103-10 passim, 117; see also Error, standard
Sinclair, H. M. and Wilson, Dagmar C. (1955), 5
Socio-economic: considerations of fluoridation, 5; status, 7, 16, 39, 48, 84, 86-7
Sodium fluoride, see Fluorides
Statisticians, 74, 79, 104, 109, 112, 120 anonymous, 99; changed, o, 52; deliberate omission of figures by, 104, 105; not employed by N.Z. Commission, 69, 113, 126
Statistics, 69, jog; and evaluation of data, 4, 30, 112, 113, 114, 18, 126; and probability, 21, 24, 30, 35, 36; and validity of fluoridation, 76, 82, 83; biometrics, 113, 126; evidence from, 69; methods used in, see Methods; principles of, 75, 80; significance of results, see Significance; terminology used in, 36
Stratford (control city for Brantford), 3, 4, 38-47 passim, 96-III passim, 115, 117
Studies, fluoridation, see Trials
Survey: caries, see Examinations; fluoridation, see Trials; X-ray, see X-ray
Tablets, fluoride, 66, So, 153, 125
Teeth: caries-free, 57-8; caries in, see Caries; Caries attack rates; congenitally missing, 26; development of, s; erupted, a, ii, aS, 53, 54, 56, 60, 61, 70, 8s, 82; eruption of, see Eruption of teeth; individual, exposed to caries, 6; 'resistant' as a result of fluoridation, 2
Test city, 4, 8, 9, 37, 47, 68, 77, 87, 116, 117; see also Brantford, Evanston, Grand
Rapids, Newburgh

Trials, fluoridation: aim of, 42, 77-8; commenced, II, 20, 38, 59, 78, 90 controlled, see Controlled, Controls; data from, see Data; deficiencies not recognized, x; 'demonstrations', 75, 77-8; design of, see Design; experimental, x, 7, 67, 70, 72, 76, 77, 79, I 06, I 15, 121; examiner variability in, see Variability; independent, 114, 127; (in Brantford) 3, 37; late commencement of, 38, 95, 100, I0s, 105; long-term, I00; methods used in, see Methods; no control used, 7, 128; results of, see Results; see also Brantford, Evanston, Grand Rapids, Newburgh

United Kingdom Mission (1953), 16, 20, 25, 28, 32, 37, 39, 40, 42, 45, 48, 60-8 passim, 87, 120

United States of America, 1, 5, 19, 73, 75, 76, 78, 79, 84, 114, 115, 127, 129; ad hoc Committee on Fluoridation of Water Supplies, 77

Variability, examiner, 44, no; and 'calibration', II, 75, 79-80; betweenexaminer, 2, 9, II, 51, 68, 80, 97, 105, 116; elimination of, six, 1,6; evaluation of, it; 'human error', 75, 79; importance of, 9, 5!, 79; not assessed, 2, 11, 51, 52, 68, 79, 80, .6; not considered, 13, 72, 79; 'overlooked', 75, 79; within-examiner, 9, II, 51, 116

Variation: causes and measurement, 8; in age groups, 50-7 passim; in diagnosis, 51, 96, lot; in examiners, see Variability; in methods, 50, s; random, 97, 107, (ignored) 2, 30, 68, 1,6, (importance not recognized) 2, 90, 116, (not considered) 72; sampling, 106; unknown, 8, 90

Water: analysis, 49, 84, 96, 102; and mottled enamel, 8, 43; composition, 8, 84, (different in Newburgh and Kingston) 48-9, 64, 68, 70, 113, 123; domestic, s, 76, 509, 113, 123, (in Stratford) 42-3; finished, 42; fluoridated (artificially) 5, 28-9, 42, 58, 64, 72, 75, 76, 77, 78, 79, (in control city) 11, 14, 20, 21, 68, 107, (in test city) see Fluoridation, (ingestion of) 2, 11, 53, 54, 61, 70, 74, 8o, 81, 92, 503, (naturally) 28-9, 42, 64, 74, 75, 76, 77, 8o, 100, 109, 125 fluoride-bearing, 8o, 8; fluoride content of, 42, 78, 102, 108, 110; fluoride-deficient, 6, log; 'fluoride-free', 4, 7, 10, 14, 15, 38, 48, 63, 64, 78, 100, 109; from wells, 43, 96, 102, Iso; Lake Michigan, 32, 84, 6s; proposals to fluoridate, 74, 76; sources for test and control cities, 8, 10, 48-9, 64, 84; surface, 48; unchanged, ss; untreated, 64, variables in, 523; variations in, 49; volume consumed, 8

Water supply, 7, 76; composition of, 7-8, 67, 77, 123, (calcium content) 49, (different in Newburgh and Kingston) 48-9, 64, 68, 70, 113, &23. (fluoride content) 42, 78, 90, 96, 502, (magnesium content) 49; fluoridation of, 1, 5, 7, 8, 42, 8, 64, 71, 72, 74, 75, 76, 77, 78, 81, 99, 103, 107, 109, 114, 123, 125, 526, 527, see also Fluoridation; 'fluoride-free', 4, 7, 10, 14, 15, 38, 48, 60, 63, 64, 76, 109, 121hardness of, 49; ingestion of salts from, 8; of Arizona communities, 43; of Baltimore, 38; of Brantford, 38, 41, 42, 47, 65, 96, 102, 103, 109 of control city fluoridated, xi, 14, 20, 2s, 68, lo; of Evanston, 16, 20; of Grand Rapids, it; of Hastings, no; of Jacksonville, 63; of Kingston, 48-9, 113, 123; of Muskegon, 10, II, 3, 14, 64, 68, 107; of Newburgh, 48-9, 64, 68, 70, 513, 523; of Oak Park, 20-1, 107; of Sarnia, 38; of Stratford, 38, 42-3, 96, 102, (so); of test and control cities, 48-9, 68, 70, 78, 84, 153, (importance of close comparability) 7, 49, 50, 64, 123; public, 5, 77, 78; soft, and mottled enamel, 8 'Weighting', 29, 47, 52, 53, 54, 6, 98, 109, IsO

World Health Organization, 46, 63-70 passim, 127

X-ray: and accuracy of caries rates, ss; and choice of schools, 62; examinations, 2, II, 28, 50, 51, 53, 6, 58, 8, 89, (essential) 112, 118; (importance of) 16, (incomplete or absent) 16, 112, 118, 119, (proximal) 29
ADDENDUM TO SECOND EDITION
(Published in The Greatest Fraud: Fluoridation, by
to accompany the reprint of the earlier—1960—book)

Further criticisms and comments.

In the second edition of the monograph Fluoridation. Errors and Omissions in Experimental Trials it was stated that copies of the first edition were sent by the Federal President of the Australian Dental Association to all the men in charge of the trials which had been considered.

As has been mentioned, criticisms by the authors of the Evanston and the Brantford (Canadian Department of National Health and Welfare) studies, were published as “book reviews” in the February, 1960, issue of the Australian Dental Journal.

After the second edition was “in press”, the June 1960 issue of Nutrition Reviews was received, containing (Vol. 18, pp. 161-165) a paper by Dr J.M. Dunning entitled “Biased criticism of fluoridation. This paper quoted some passages from “… letters to Dr Kenneth Adamson, President of the Australian Dental Association” from the senior author of the Grand Rapids trial, Dr F.A. Arnold Jr., and from the senior author of the Newburgh trial, Dr D.B. Ast, and some criticisms by Dr J.R. Blayney of the Evanston trial, which had not been published in the above-mentioned “book reviews” in the Australian Dental Journal.

The following pages contain all the passages from those letters which were published by Dr Dunning. In view of the title he gave to his paper, it is considered likely that he cited from those letters the quotations which he considered to be the most important criticisms advanced by the authors of those fluoridation trials.

These comments were prepared in 1960 and had a very limited circulation in roneoed [Gestetnered] form. They have not been otherwise published until now because of the refusal of many editors to accept comments which question fluoridation.

Dr F.A. ARNOLD JR

Dr Dunning said that: “Dr F.A. Arnold, Jr., Director of the National Institute of Dental Research and principal investigator at Grand Rapids, writes in
part as follows (Arnold to Adamson. October 16. 1959)“.

The quotation from Dr Arnold’s letter which will be considered first is the following accusation (as reported by Dr Dunning):

(84) “Although he [the author of the monograph] did not publish his material until 1959, he (apparently intentionally) overlooked the report of the tenth year of the study which appeared in 1956. As was originally planned, it was this year that we obtained “complete” age groups of adequate size.”

Comment. It is difficult to believe that Dr Arnold could have made this extraordinary accusation for, if the monograph is consulted, it can be seen that “...the report of the tenth year of the study which appeared in 1956” the paper by Arnold et al. (1956) - was (a) listed under Dr Arnold’s own name in the references, (b) shown, again under his name, in the Index, which indicates that this paper was mentioned on four pages, and (c) was given as the source of the data from which Figures 1 and 2 were compiled (pages 147 and 148). These two figures depict the caries rates reported for each age group in each year in the city of Muskegon up to the time when, as a result of its water supply having been fluoridated, it ceased to be the “fluoride-free” control city for Grand Rapids. (See explanatory notes to Figures 1 and 2, pp. 147 and 148).

If Dr Arnold is correctly quoted by Dr Dunning, it would appear that, before criticizing it, he read the monograph only superficially, even that part of it which relates to his own study. Furthermore, it is clear that, before making the accusation that “apparently intentionally” this 1956 report from the Grand Rapids study had been “overlooked”, he failed to check both the list of references and the Index.

If this is not the case, one is forced to conclude that Dr Arnold made this accusation deliberately, knowing that it was untrue, with the intention of misleading the President of the Australian Dental Association.

That author was also reported to have written (85-91):

(85) “He [Sutton] overlooks the fact that one examiner has been with the study throughout.”

Comment. This fact was not “overlooked”. This can be seen by referring to the monograph, the top of page 144 where Arnold et al. were quoted as saying that: “There have been changes in the dental examiners with the exception of one officer who has participated in each series of examinations.
Each new examiner has been calibrated against this one officer to standardize diagnostic criteria” (Arnold et al., 1953).

(86) “If we used his findings, we would come up with the same general result.”

Comment. As it is unlikely that the findings of this examiner were not used in this study, it is concluded that Dr Arnold is referring to the situation which would have arisen if the data from this study had been confined to those obtained by that one examiner. However, no comment can be made in this matter as in the published data the findings of the examiners were combined.

(87) “Also, we could call attention to the fact that two more of the four examiners used throughout the first ten years of the study started examining during the third year of the study and have participated each year since.”

Comment. Dr Arnold refers to “the four examiners used throughout the first ten years of the study”. However, in a note published on the first page of his report of the tenth year of the study (Arnold et al., 1956), he stated that: “The following dental officers of the Public Health Service conduct the annual dental examinations”, and named five examiners: Doctors Likins, Russell, Scott, Singleton and Stephan. In addition, he mentioned the names of four other dentists who “also participated as examiners” in the study: Drs Loe, McCauley, Ruzicka and Short. In his 1953 report also (Arnold et al., 1953) he had acknowledged the participation of the same nine examiners not four as Dr Arnold stated in his misleading letter to the President of the Australian Dental Association.

(88) “He criticizes our selecting samples by school grade. If he would realize it, and probably he does, this strengthens the study.”

Comment. It is evident that Dr Arnold has changed his views on this matter. When, in 1953, he described the method used (Arnold et al., 1953), he acknowledged that “choosing examinees by grade in this manner will, in some instances, not give well-distributed specific age groupings.” But now he says that “... selecting samples by school grade ... strengthens the study.”

(89) “In the first place this gives us a random sample.”

Comment. In order to be satisfied that the children examined constitute a random sample of those in the city, it is necessary to know if the children
were a random sample of those in their school and, also, that the schools in which the examinations were made were a random sample of those in each city. The method of sampling, as described by Arnold et al. (1953), was stated on page 153. The meagre description that “on the basis of available information” (which was not disclosed) “25 representative schools were selected” - no description of the method of selection being given - does not permit the reader even to attempt to determine whether or not the schools selected constituted a random sample of those in the city. In the next report (Arnold et al., 1956) it was not stated how many schools were selected, merely that “The annual samples of the school population of Grand Rapids and Muskegon are taken from schools selected as representative of each city as a whole.” (See comment 91 below for an independent report on the sampling methods used in this study.)

(90) “Also, it permitted us to examine all the children of a grade without the examiners knowing whether the child belonged to the “continuous resident” group or not.”

Comment. This fact was mentioned by Arnold et al. in 1953. However, it is of little consequence, for no comparisons were published between the caries rates in the “continuous resident” group and the other children in Grand Rapids. This statement by Arnold indicates that he realized the need for “blind” examinations. However, he made no attempt to incorporate this vital point in experimental design when he arranged for the examination of the Grand Rapids children and their comparison with those of the control city of Muskegon. The desirable aim of eliminating unintentional bias on the part of the examiners would have been achieved if the children in the test and the control cities had been examined on the same occasions “without the examiners knowing whether the child belonged to the “continuous resident” group in Grand Rapids or the “continuous resident” group in the control city of Muskegon. Unfortunately this was not done.

(91) “The planning of the study and the analysis of the data were done by a group of people all of whom are more knowledgeable in this field of research than is Dr Sutton.”

Comment. No comment will be made on this remark (except to say that Dr Arnold has never met me) but it is pertinent to quote another opinion. T.M. DeStefano (Bull. Hudson County Dent. Soc, 23: 20-31, Feb. 1954) quotes from the critique of the report of the “seventh Year of Grand Rapids-Muskegon Study” (Arnold et al., 1953) that “... had been sought and paid
for by a group of general practitioners from a reliable statistical firm” (the Standard Audit and Measurement Services, Inc., 89 Broad St., New York 4, N.Y.). DeStefano quotes this critique as stating:

“The authors appear to have demonstrated an unfortunate disdain for some of the pre-requisites of valid research.” Also that “In the first place, the sampling design of the experiment is embarrassingly conspicuous by its absence.

Such a brief description as: “On the basis of available information the 31 school districts in Grand Rapids were classified on a socio-economic basis. From the 79 schools in those districts, 25 representative schools were selected and the examiners assigned ... etc.” leads one to suspect that the drawing of the sample was dangerously amateurish. This suspicion makes one feel that either the results of fluoridation are so dramatic as to force themselves through the veil of poorly selected samples or “at the other extreme” that the reported results are merely the fiction of a biased sample. From work other than that reported by the authors, one tends to discard the latter possibility but the lack of sophistication shown in selecting the sample leads to complete bewilderment as to the precise effects or the extent of the effect of fluoridation.”

This critique by the Standard Audit and Measurement Services continues:

“With a pre-listed population (such as a school enrolment) there would appear to be no excuse for not using modern sampling tools and procedures. Employment of these devices would enable not only a more certain statement of the effects of fluoridation but (perhaps more importantly) a precise estimate of the error inherent in such statements.”

DR D.B. AST

Dr Dunning then said that “Dr David B. Ast, Director, Bureau of Dental Health of the New York State Department of Health, makes the following comments (Ast to Adamson, March 3, 1960)”.

Dr Ast is reported to have written (92-7):

(92) “Sutton criticizes the comparability of data among the four studies because in Newburgh and Kingston we used the rate based on DMF per 100 erupted permanent teeth instead of DMF per child.”
Comment. Contrary to this statement by Dr Ast, “the comparability of data among the four studies” was not criticized. However, it was pointed out that it is very difficult to compare the results shown in the five reports from Dr Ast’s Newburgh trial because of the different methods of presentation of data that were adopted by Dr Ast and his co-workers. Nor was criticism levelled at the use of “the rate based on DMF per 100 erupted permanent teeth”

(93) “We explained why we used the permanent tooth population as the universe considered.”

Comment. The paper giving this explanation (Ast et al., 1956) was referred to on twelve pages of the monograph.

(94) “However, in order to make our data comparable to other study data, in the reports for 1953-54 and 1954-55, the Newburgh-Kingston data were given both ways—DMF per 100 teeth, and DMF per child.”

Comment. If the aim of Dr Ast and his co-workers was to make the data from their study “comparable to other study data”, it is unfortunate that they did not examine the methods used in publishing the data obtained in other studies and publish some tables in which the Newburgh-Kingston data were presented in the form used in these other studies. Owing to this omission, they prevented comparisons being made with the results published in the other studies considered in the monograph by: (a) not disclosing any caries rates for deciduous teeth except in their 1951 report, (b) confining the rate “DMF teeth per 100 children” in 1953-54 to those aged six, seven, eight, nine and ten years (Ast et al., 1956); (c) combining the 1954-55 caries data into four groups children aged six to nine years, ten to twelve years, thirteen to fourteen years, and sixteen years of age (Ast et al., 1956). In the other main studies, although the DMF rates were shown per child or per 100 children, either clinical examinations only were used, or the data were reported for individual yearly ages or for age ranges which were different from those used by Ast et al. Thus, comparison of these rates with those published from the Newburgh study cannot be made.

(95) “What is significant and had escaped Sutton is the fact that the percentage differences in Newburgh and Kingston were almost the same for both methods used.”

Comment. Dr Ast, no doubt, did not mean to suggest that the results were
almost the same in the test and the control cities, but intended to refer to the percentage differences (in caries rates) between Newburgh and Kingston.

It is surprising that Dr Dunning should have published this remark of Dr Ast, for a paper which he wrote almost ten years earlier (Dunning, 1950) showed that he realized the inadequacy of results stated merely as percentage reductions. In the summary of that paper he pointed out that “Interpretative and other examining errors in DMF studies may be large, easily exceeding 100 per cent differences between samples.” He said also that:

“Illustrations of actual data indicate that the standard deviations of observations about the means (averages) in DMF studies are large even where examining errors are reduced to a minimum.” Dr Dunning then said that: “These two sources of variability imply that human DMF studies should be subjected to close scrutiny as to the validity of the data and statistical significance tests applied and reported wherever possible. Mere statements that “caries was reduced by x per cent” are not sufficient.”

It can be seen that it is precisely this method of presenting data, that Dr Dunning criticized in 1950, which was used by Ast et al. to report the results from the Newburgh trial: “Mere statements that “caries was reduced by x per cent” (differences between the test and the control cities) without “statistical significance tests applied and reported.”

(96) “Another criticism made is that baseline data were collected in Kingston a year after the Newburgh survey. I can’t believe Sutton really believes this to be valid criticism. He must be, or should be aware of the fact that caries is not an acute disease of short duration, but a slowly developing one ...

Comment. Dr Ast is wrong in his assumption - it certainly is considered to be valid criticism to point out that the initial examination was not made in the control city until after the fluoridation of the test one. By writing about the obvious fact “that caries is not an acute disease of short duration, but a slowly developing one” Dr Ast avoids the significant point: that he and his co-workers assumed that the caries rates in the children in the control city would be similar to those in the test one, and that they omitted, prior to starting the experiment, to test this vital matter.

(97) “The baseline data in Newburgh and Kingston based on the examination of all the school children age six to 12 in both cities
were almost identical. All of the examinations were made by the one examiner. Could Sutton really believe that the DMF rate of 20.8 for Kingston, and the 21.0 for Newburgh could have been significantly different if both examinations were made exactly at the same time?...” [end of published quotation]. “... this type of criticism questions not the research but the professional acumen of the critic.”

Comment. Dr Ast and his co-workers were fortunate that they were able to present figures for caries rates which were comparable, although the fact should not be forgotten that they improved the comparability between the initial caries rates in the test and the control cities by combining the data from children of different ages.

The workers who conducted the Evanston study made the same assumption and failed to examine the children in the control city until after the fluoridation of the test one (Blayney and Tucker, 1948; p. 153). They were not as fortunate as were Ast et al., for they found “...a lower caries rate for school children of the control area” (Hill et al., 1951). In the younger children, there were gross differences between the initial caries attack rates in Evanston and its control city. The same omission was made in the trial in Hastings, New Zealand. As a result, the control was abandoned, for its caries rates were lower than in Hastings (Ludwig, 1958).

DR J. R. BLAYNEY

Dr Dunning then said that “J.R. Blayney, Director of the Evanston Dental Caries Study, comments thus (Blayney to Adamson, November 23, 1959)”. Dr Blayney is reported to have written (98-100):

(98) “Dr Sutton ....states, “the arbitrary selection of the data which is then termed “ representative”, instead of making the ultimate findings to be considered valid and reliable, would render a report based on this selective data unfit for serious consideration.”

Comment. This “quotation” is inaccurate. Dr Blayney has omitted the words “a section of and refers to “selective data” instead of to “selected data”. The original paragraph was: “However, the process which they described—the arbitrary selection of a section of the data, which is then termed “representative”—instead of making “the ultimate findings to be considered valid and reliable”, would render a report based on this selected data unfit for serious consideration.”
Comment. This comment by Dr Blayney to the President of the Australian Dental Association is misleading. The statement by Hill et al. (1950), which was quoted on pages 151 and 152 and to which the comment made by the author of the monograph refers, made no mention of the “separation of white and Negro, public and parochial school children”, but instead, stated the intention of including “... only those groups of children which are representative of the population, with respect to dental caries experience”,

Hill and Blayney originally did not intend to separate the children into racial and school groups. It was not until their 1951 report that mention was made that they contemplated such an action, that is, not until a year after they published the statement mentioned above. They decided to separate the Evanston data into racial and school groups when they found that the initial examinations “... indicated a lower caries rate for school children of the control area” (Hill et al., 1951). They have not explained why it was necessary to exclude from the main body of white children those who happened to attend the parochial school, rather than the public one.

As they consider that Negroes have less dental caries than white children it is, of course, reasonable to consider the data of white children separately from those of Negro children. However, Hill et al., first included the data of Negro and parochial school children (with those of the white children attending public schools), then excluded these (Negro and parochial school) data for several years, and then, despite their statement that such a process was necessary, reversed their policy and included these data with those of the white children attending public schools. By this reversal of policy they were able to present initial caries rates for the test city which were more comparable to those in the control city than would have been the case if they had not disregarded their previously-stated policy of comparing “like with like”. This cannot be considered to be a reasonable course of action.

(100) “We have gathered no secret or concealed data” ...[end of published quotation].
Comment. This assertion by Dr Blayney should be considered in the light of the numerous instances, in his study, in which relevant data were not published, in some cases even for as long as twelve years after they were obtained.

The failure of Dr Blayney and his co-workers to publish these relevant data has, without question, concealed them from readers of their reports.

Dr Dunning quoted two further paragraphs from Dr Blayney’s letter. These were printed in the “Book Review” published in the Australian Dental Journal in the February, 1960, issue.

DR J.M. DUNNING

The criticisms made by Dr Dunning himself will not be considered, for his attitude to the monograph and the lack of care in the preparation of his critique are evident from even one example:

He stated that “In discussing requirements for a control, Sutton adopts the position that the control city should be “comparable in all respects” to that where fluoride is being added.” That phrase was not used by the author of the monograph, but was quoted by him from two sources (pp. 141, 178, 179,190). His, considerably different, views on this matter were stated on pages 190 and 193.

Therefore, by attributing to the author an opinion which he did not express, and that he actually criticized (p. 190), and by omitting the different opinion that the author did express, Dr Dunning misleads his readers.

Furthermore, the fact that Dr Dunning has elected to publish these extracts from letters written by authors of fluoridation trials, indicates either that he has chosen to ignore or has failed to detect errors in them which should be obvious to a careful investigator.

In 1984, twenty-four years later, Dr Dunning was still criticizing papers which questioned fluoridation. He continued his former technique of misleading his readers by inventing false statements, attributing them to the author of the article he was criticizing, then disputing his own false statements. He stated that Colquhoun (1984):

“... mentions an increase in periodontal disease as if it might have been caused by fluoridation.”

In fact, Colquhoun (1984) stated that: “... water fluoridation does not affect” periodontal disease.
Dr Dunning also wrote that:

“Colquhoun continues to quote Sutton on the subject of defects in early studies of fluoridation, stating that he (Colquhoun) has seen no convincing refutation of this. I offer him my article, “Biased Criticism of Fluoridation” in which I quote the views of several of the leading fluoride researchers of the day. The studies Sutton criticizes most harshly have survived as pioneer efforts and been confirmed not only for their conclusions but for their methodology.”

This was the paper in Nutrition Reviews, mentioned above, which has remained unchallenged in print until now, years after it was written, because of the difficulty in having accepted for publication any material which questions fluoridation.

It is interesting that Dr Dunning (1984), after so many years, can call the authors of the original studies:

“... the leading fluoride researchers of the day”.

and say that their methodology had been accepted, when he himself (Dunning, 1950) condemned the method they used of expressing caries changes as percentages without the use of statistical tests.

That Dr Dunning now accepts their methods, such as that used in the Evanston study which led to the authors admitting that they had made gross errors in stating the number of children seen during one examination, one of which was a discrepancy of more than 1000 children shows that he should be included with those described by Professor John Polya (1964) as:

“... unreliable witnesses before a jury either of scientists or of lay common sense.”

It is clear that Dr Dunning’s criticisms can be disregarded. He is one of those critics whose intense pro-fluoridation opinions have made them muddled thinkers, and he is one who intentionally manufactures incorrect statements about those he criticizes to try to attack work which he cannot find grounds to fault by legitimate means.
ADDITIONAL OBSERVATIONS ON THE EVANSTON, GRAND RAPIDS AND NEWBURGH TRIALS

1. Gross numerical errors in statements of the number of children examined.

The Evanston Trial

These comments on the Evanston trial were published in 1980 in the present author’s book Fluoridation Scientific Criticisms and Fluoride Dangers. It was stated:

“Additional Errors in the Evanston Trial Data.

In January, 1967, which was the twentieth anniversary of the commencement of the Evanston Trial, an entire special issue of the Journal of the American Dental Association was devoted to a report on that study (Blayney and Hill, 1967). In this, the original tables, complete with their gross numerical errors, were reproduced, despite the fact that these [errors] had been pointed out eight years earlier (Sutton, 1959) and some of them had been acknowledged by the authors (Sutton, 1960). In addition, several faulty tables were published for the first time.

The tables [in this issue of the J. Amer. Dent. Ass.] then showed three different statements regarding the number of children aged 6-8 years who were examined in Evanston during the 1946 examination:

(i) 1991 children - see Tables 10, 11,30,40 and 47.
(ii) 1985 children - see Tables 7,8,16,18,21 and 32.
(iii) 1754 children - see Tables 24 and 25.

There were also no fewer than six different statements in that article of the number of children aged 12-14 years examined in Evanston in 1946:

(i) 1703 children - see Tables 15 and 32.
(ii) 1702 children - see Table 47.
(iii) 1701 children - see Tables 11,30,41,44 and 45.
(iv) 1697 children - see Tables 7,9,12,13,17,19,22 and 31.
(v) 1556 children - see Table 26.
(vi) 1146 children - see Table 46.

Between the sum of the two highest statements of the number of children examined in Evanston in 1946, and the sum of the two lowest statements of children examined in the same year in the same study in the same city, there
is a difference of 794 children (1991 + 1703 - 1754 - 1146 = 794).

The number of children stated to have been examined in Evanston is even more divergent in the original papers than in this special article. Blayney and Tucker (1948) and Hill et al. (1950) both gave a figure of 4375 children, compared with the number of 3310 in Hill et al. (1957b), a difference of 1065 children.

It was these differences which the medical journalist Anne-Lise Gotzsche, in a letter to the Lancet in 1975, said that she had showed to workers in other fields, and that they had “simply laughed” at the statistics (see Fig. 5, p. 167).

In that book (Sutton, 1980)—prepared as a submission to the Committee of Inquiry into the Fluoridation of Victorian Water Supplies (1980)—it was stated (p. 203):

“These errors were mentioned [by the present author] 12 years ago to the Tasmanian Royal Commission on Fluoridation. Since that time I have not heard of any mention of them or of a criticism having been made of the numerical data published in that report.”

It appears that, in the manner common in fluoridation trials, those erroneous tables have been accepted at their face value, without investigation.

More than thirty years ago it was pointed out (Sutton and Amies, 1958b) that:

“This uncritical attitude to these studies is rife.” “Also it has been assumed that associations and individuals that ... accepted the responsibility of publicly advocating fluoridation, have undertaken independent examinations of the data, and not merely repeated the opinions of others.”

This situation was referred to by Professor John Polya (1964) in his book Are We Safe? He wrote:

“It is immaterial that other evidence in favour of fluoridation is not always false; the point is that persons, bodies and arguments that knowingly or in simplicity acquiesce in one blatant falsehood are unreliable witnesses before a jury either of scientists or of lay common sense.”

He continued:

“The scandal created by the exposure of this absurdity resulted in
the admission that the first figure (4,375) was correct. In defence of
the other claims it was explained that “out of range” children were
eventually excluded from the survey, but then further critical check
revealed more numerical inaccuracies, not to speak of the magnitude
of a correction exceeding 1,000. In better examples of scientific
work the author sticks to his experimental group; discarding on
the scale quoted strongly suggests that the experiment had to be
altered to fit pre-conceived results. This is one of the common
consequences of working without control of observer bias.”

It is pertinent to point out that, in the Foreword to that article in the special
edition of the Journal of the American Dental Association, in January 1967,
Dr F.A. Arnold, Jr., the Assistant Surgeon General, Chief Dental Officer,
U.S. Public Health Service (and formerly the chief experimenter in the
study in Grand Rapids) stated:

“Here, in a single report, are data on the effect of water fluoridation
on dental caries so completely documented that the article is
virtually a text book for use in further research. It is an important
scientific contribution towards the betterment of the dental health
of our nation. It is a classic in this field.”

It is indeed a classic - a first-class example of the errors, omissions and
misstatements which abound in the reports of these fluoridation trials.

2. False information in the Abstracts of papers

The abstracts of reports on fluoridation trials are unusually important, for
it is likely that lay people, and politicians in particular, will confine their
reading of the report to the Abstract, assuming that it accurately reflects the
findings, and will base their opinions and actions on its statements.

The Grand Rapids Trial. The final report of the Grand Rapids study was
published in 1962. Reading the Abstract which preceded the body of the article it would seem that, at last, the authors (Arnold et al., 1962) had come
to realize the necessity for comparing the results from the test city with those
from the control one for they stated that the results had been “...compared
with the caries attack rates in the control group of children in Muskegon,
Mich.” This claim was not made in the body of the article, which included
the statement that: “... fluorides were introduced to this [Muskegon] water
supply in July, 1951” Therefore at that time Muskegon ceased to be a control
city, some eleven years before this final report (Arnold et al., 1962) from Grand Rapids.

How then, in 1962, could the final result from the test city be compared with data from a non-existent control one?

The claim of Arnold et al. (1962) that they compared the Grand Rapids caries rates with those in the “control group of children in Muskegon, Mich.” is shown to be false by their statement that: “...in subsequent [after 1954] analyses of Grand Rapids data, comparison has been made with the original Grand Rapids findings and with those for Aurora.”

This is confirmed by the statement in the Abstract that:

“Caries attack rates were lowered by 57 per cent in children 12 to 14 years old in 1959.” This figure of 57 per cent is obtained by averaging the figures of 57.0, 63.2 and 50.8 per cent for the ages of 12, 13 and 14 years shown in their Table 2 to be the “per cent reduction in DMF teeth (1944-1959)” in Grand Rapids (not between Grand Rapids and its control city of Muskegon).

*The Newburgh Study*. Similar mis-information regarding comparisons being made between test and control cities was published in the same year (1962) by Dr David Ast, the senior author of the Newburgh study. In the Abstract of that paper (Ast and Fitzgerald, 1962) he wrote:

“Among children 12 to 14 years old in the four study areas, reductions in the DMF rates as compared to the rates in control cities ranged from 48 to 71 per cent.”

Table 2 is the only one in that paper showing DMF rates for children aged 12-14 years (in one case 13-14 years). In the first two studies listed, Grand Rapids and Evanston, no reference is made to a control, the “reduction” in Evanston, shown as 48.4 per cent, is obviously the 48 per cent mentioned in the Abstract. This “difference” is between the rates in Evanston in 1946 and 1959, not between Evanston and a control, as stated in the Abstract. The Grand Rapids rates are also shown between that city in 1944-45 and 1959, no control data being used. Indeed Ast and Fitzgerald stated in the main text:

“In the Grand Rapids and Evanston studies the control cities were lost before the study was completed, so that the current data have been compared with the base line data.”
Not with control cities, as they stated in their Abstract.

There should not have been any confusion regarding the use of the term “control”, for the co-author of that paper, Bernadette Fitzgerald, was described as the “senior biostatistician, division of special health services, New York State Department of Health.” Therefore the authors’ incorrect statement that they compared the caries rates “in the four study areas” with rates in control cities is unlikely to have been made inadvertently.

3. Continuing publication of false statements.

It has just been shown that Dr Ast (the senior author of the Newburgh study) and Dr Arnold (the senior author of the Grand Rapids study) continued to disseminate false statements regarding their studies many years after those ten-year studies were concluded, Also, the arrogance of Drs Blayney and Hill (the authors of the Evanston study) in publishing an article in 1967, which repeated, in a special issue of the Journal of the American Dental Association, figures which they had acknowledged seven years earlier were faulty (Sutton, 1960), indicates the reckless disdain of all those authors for the truth, and for the members of the scientific community (which normally trusts statements made in established journals by senior scientists, for it is not used to being misled by such readily-verified deceptions).

Their false statements do not engender confidence in the reliability of the data published and the statements made by those senior scientists in their original reports of what are still regarded by fluoridation advocates as three of the four main fluoridation studies on which the case for fluoridation mainly relies - those in Newburgh, Grand Rapids and Evanston in U.S.A.

Commenting on the Grand Rapids study, Ziegelbecker (1983) pointed out that the experimenters had examined “all” children from 79 schools in Grand Rapids at the commencement of the trial, but that:

“After 5 years in 1949 they selected children at only 25 schools in Grand Rapids for their investigation and observed children at the same time at all schools in Muskegon (the control city).”

For instance, the number of children aged 12 to 16 years who were examined in Grand Rapids at the commencement of the trial was 7,661, but only 1,031 were examined in 1959 (Arnold et al., 1962).

In 1988, Colquhoun stated:
“In the control city of Muskegon all children were examined throughout the period. From the year-by-year figures for six-year-olds which were published three years later in 1953, it is revealed that an impossible 70.75% reduction was recorded in the first year of the trial (Arnold et al., 1953) and that there was then an increase and no overall reduction in the following years. Examination of similar data for other age groups shows that the sample of 25 schools could not have been representative of the population being studied.”

He pointed out that:

“The reported DMF of several of the age groups in this sample, approximately one year after the initial examinations, was lower than that of the same children when they were a year younger.”

He concluded:

“Fluoridated water cannot turn decayed, missing or filled teeth into sound ones. It follows that the caries experience of the children had not been reduced as claimed. The large recorded reductions, which were mostly in the first year only, were a result of selection of data.”

4. Fictional results?

In 1954 De Stefano reported the findings of professional statisticians regarding the Grand Rapids study. They raised the question whether “...the reported results are merely the fiction of a biased sample.”

Ziegelbecker (1983) also, studied this situation. He stated:

“We must conclude from this result that the sample in Grand Rapids was not representative for all children and with respect to the basic examination. In the following years from 1946 to 1949 (and later to 1954) the 25 schools in the sample were the same each year and we see that the caries experience in the sample was not reduced by fluoride in 1946-1949.

If we accept that the sample was representative for the children, aged 6, in the 25 schools in those years then we must conclude that fluoride in the drinking water had not reduced the dental caries experience of children, aged 6, in Grand Rapids in the years before the US Public Health Service
released the policy statement [endorsing fluoridation] to the American Dental Association.”

He concluded:

“We must conclude from these results that a fluoride content of 1 ppm in the public water supply does not reduce dental caries experience.”

Colquhoun stated in 1988:

“In their final study in Grand Rapids, published in 1962 after 15 years of fluoridation, American health officials [including the director of the U.S. National Institute of Dental Research, Dr F.A. Arnold, Jr.] wrote: “... no such dramatic and persistent inhibition of caries in large population groups had ever been demonstrated by any other means than fluoridation of a domestic water supply.”

Colquhoun commented:

‘That statement, which could be described as the dogma of fluoridation, is now considered by an increasing number of critics to be unscientific and untrue.”

In view of the disclosure of the types of error which have just been mentioned, such a grandiose claim, although it was widely accepted at the time, can no longer be considered to be true.

More than thirty years ago Sutton and Amies (1958a) commented on this sudden initial decrease in caries reported from Grand Rapids (and from other studies considered). It was stated that the results reported were not those which would be expected if the hypothesis was correct that fluoride “strengthens” developing teeth and makes them more resistant to attack by caries. Despite the fact that the results published from fluoridation studies do not support this hypothesis, it is still mentioned. For instance, the ten members of the task group which in 1984 wrote the latest WHO book on this subject: Environmental Health Criteria 36. Fluorine and Fluorides, referred to the importance of “lifelong consumption” of fluoridated water.
GLOSSARY

APPROXIMAL SURFACE
Adjacent surfaces of teeth in the same jaw (upper or lower)

“BLIND”
See Examinations “Blind”

CALIBRATIONS
Readings or assessments made in appropriate units

CARIES
Progressive decay of teeth (or bones). See Dental Caries

CARIES EXPERIENCE
The extent and severity of dental caries within a population - usually measured with indexes such as DMFS or DMFT etc.

CARIGENIC SUBSTANCE
One which produces decay (within a tooth).

CARIOSTATIC
Decay retarding

DECIDUOUS TEETH
(Primary or “milk” teeth). These start to erupt around 6 months and are shed around 12 years when the permanent teeth start to appear.

DEMINERALIZATION
Reduction of the mineral content (principally, calcium and phosphorous) of a (issue, notably the enamel, dentine or cementum of teeth.)
DENTAL CARIES
Disease of the teeth resulting in the demineralization, cavitation and breakdown of calcified dental tissues (enamel, dentine or cementum) by microbial activity.

DENTAL DECAY
See Dental Caries

DENTAL FLUOROSIS
A disturbance of tooth formation caused by fluoride being present in the tissue fluids over a prolonged period during tooth development. The disturbance results in the development of porous enamel which has an altered appearance ranging from the most mild forms, in which small flecks of white discolouration can be observed on the tooth surface, to the most severe forms, in which the enamel develops pitting and brown staining. Dental fluorosis must be distinguished from other disorders of enamel including: enamel opacities (see below) of non-fluoride origin; early `white spot’ caries lesion; enamel hypoplasia; amelogenesis imperfecta; dentinogenesis imperfecta; and tetracycline stains.

DENTAL PROPHYLAXIS
The prevention of dental disease, especially dental caries.

DENTRIFICE ("TOOTHPASTE")
A pharmaceutical compound used in conjunction with the toothbrush to clean and polish teeth. It contains a mild abrasive, a detergent, flavouring agent, binder, and occasionally deodorants and various medicaments designed as caries preventives, for example, fluoride and antiseptics.

DENTITION
Natural teeth in the jaws

DEF, DMFT, DMFS, DIMFT, def, dft, dfs, dmft
Indexes describing the dental caries experience of individuals or populations. The DMFT index is computed by summing the number of permanent teeth which are Decayed, Missing or Filled. For any person, the index can range in value from zero to 32, the maximum number of teeth. The DMFS index is a count of the number of permanent tooth surfaces which are decayed, missing or filled. Teeth may have either four surfaces (incisors and canines) or five surface (premolars and molars) for the purposes of the DMFS index.

The DIMFT index includes, in addition to a count of decayed, missing and filled teeth, the number of teeth which are Indicated for (in need of) extraction. (DEF decayed, extracted, filled)

Lower case lettering refers to the deciduous dentition. Hence the dft index is count of the number of deciduous teeth which are decayed or filled, while the dfs index refers to the number of surfaces affected. Missing deciduous may also be included in the index, hence constituting the dmft index.

DISCRETIONARY FLUORIDE
Any form of fluoride which is used actively and preferentially by an individual in the prevention of decay. This includes fluoride tablets or drops, fluoridated toothpastes or mouthrinses, and professional topical applications. It does not include fluoridated drinking water.

DOSE (Fluoride)
The amount of fluoridated water drunk, consumed, ingested, by each person. This “dose” differs enormously depending on age, weight, size, state of health, temperature of day, different types of work and sport played etc.

EFFICACY
The extent to which a specific intervention, procedure, regimen, or service produces a beneficial result under ideal conditions. Ideally, the determination of efficacy is based on the results of a randomized controlled trial.

ENAMEL DISTROPHY
Defective formation of tooth enamel (or deterioration)
ENAMEL OPACITY
An opaque area on the normally transparent (translucent) enamel (often referred to as “mottled” teeth)

ENDEMIC AREA
Present within a localized area or peculiar to persons within such an area.

ERROR
BETWEEN-EXAMINER ERROR - variations between the assessments of two different examiners when examining the same person.
WITHIN-EXAMINER ERROR - an examiner may record different opinions when examining the same mouth on different occasions.

EXAMINATIONS “BLIND”
Are carried out to avoid bias. The examiner should be unaware of the background or treatment of his patient.

EPIDEMIOLOGY
The study of the distribution and determinants of disease in human populations and the identification and evaluation of methods of preventing or alleviating illness.

EPIDEMIOLOGY - EXPERIMENTAL
A study in which a population is selected for a planned trial of a regimen whose effects are measured by comparing the outcome of the regimen in the experimental group with the outcome of another regimen in a control group. In some experiments, for example, fluoridation of drinking water, whole communities have been allocated (usually non-randomly) to experimental and control groups.

EVALUATION
A process that attempts to determine as systematically and objectively as possible the relevance, effectiveness, and impact of activities in the light
of their objectives. Several varieties of evaluation can be distinguished, for example evaluation of structure, process and outcome.

FISSURE
A minute crack in the surfaces of a tooth (caused by the imperfect joining of enamel during development).

FISSURE SEALANT
An adhesive, plastic film applied to those surfaces of teeth which have pits or fissures to assist in the prevention of caries - however this film does not tend to last as long as prophylatic fillings.

FLUORIDATION
Is achieved by the mechanical addition of fluorides to a public water supply to attain a concentration of approx. 1 part fluoride to one million parts of water (lppm) (see also RATE) FLUORIDE COMPOUNDS Used to artificially fluoridate water supplies. They are derived from industrial processes and dissolve readily in water.

FLUORIDES
Are substances containing fluoride ions which are constituents of the element fluorine.

FLUORIDES (NATURAL)
Natural fluorides found in nature are those found in which the fluoride ions are bound to calcium and are very insoluble.

FLUORIDE VEHICLE
The means by which supplementary fluoride is provide for the prevention of dental caries. The principal fluoride vehicles are: water fluoridation, fluoride tablets or drops, fluoride toothpastes or mouthrinses, and professional dental applications.
FLUORINE
Is a rare and toxic gas (yellow, pungent), however the term fluorine in most cases can be substituted for fluoride (mainly European use).

FLUOROSIS
Fluoride poisoning due to ingesting or drinking too much fluoride in drinking water over a long period of time (or to ingestion of pesticides containing fluoride salts). Chronic fluorosis results in the “mottling” of (children’s) teeth.

FLUOROSIS Dental - See Dental Fluorosis

FLUOROSIS Skeletal - See Skeletal Fluorosis

GEL (Fluoride gel)
Contains 1.23% Fluoride for direct application by dentists to teeth during prophylactic treatment.

GINGIVAL SURFACE
That part of the tooth surface which is adjacent to or immediately above the gum (gingiva).

HYPOPLASIA
Incomplete development of an organ or part.

INCIDENCE
The number of new cases of a given disease or other condition in a given population at risk of the disease during a designated time. The word is often used to mean incidence rate.

MFP
Monofluorophosphate - one formulation commonly used for the addition of fluoride to toothpaste.
MOTTLED TEETH
See Enamel Opacity and Dental Fluorosis

MOUTH RINSE, MOUTH WASH
A mouth rinse possessing cleaning, germicidal, and / or palliative properties.

OCCLUSAL SURFACE
The “biting” surface of the tooth which makes contact with that of the opposing jaw.

OSTEOPOROSIS
Porosity and brittleness of bones due to loss of protein from the bone matrix.

PERMANENT DENTITION
The set of natural, permanent teeth in the dental arches.

PPM
Parts per million - a measurement of the concentration of a substance. A concentration of one part per million is equivalent to one milligram per kilogram. For example, fluoridated water at 1 ppm contains one milligram of fluoride ion per litre of water.

PREVALENCE
The number of individual cases of a given disease or other condition in a given population at a designated time. The word is often used to mean prevalence rate.

PRIMARY DENTITION
The set of natural, primary (deciduous) teeth in the jaws.
PROPHYLATIC FILLINGS
Dental fillings which, in the past, were placed in the pits or fissures of sound (non-decayed) teeth with a view to preventing the development of advanced decay.

PROTOPLASMIC POISONS
Fluorides are generally protoplasmic poisons because of their capacity to modify the metabolism of cells by changing the permeability of the cell membrane by inhibiting certain enzyme systems.

PUBLIC HEALTH
Encompasses the problems affecting the health of a population, the collective status of health of the people, environmental health and health services, and the administration of health care services.

RANDOM VARIATION
This term is used to describe the differences which may occur constantly from year to year in treated or untreated areas due to the influence of many random factors which affect caries rates, some of which are unknown.

RANDOMIZED CONTROLLED TRIAL
An epidemiological experiment in which subjects in a population are randomly allocated into groups, usually called “study” and “control” groups, to receive or not to receive an experimental preventative or therapeutic procedure or intervention. The results are assessed by comparison of rates of the disease, death, recovery, or other appropriate outcome in the study and control groups, respectively. Randomized controlled trials are generally regarded as the most, scientifically rigorous method of hypothesis testing available in epidemiology.

RATE
The amount of fluoride (F) in the water either naturally or added by a water authority expressed as p.p.m. (parts per million) or mg/litre (milligrams per litre)
REMINERALIZATION
The process whereby a demineralized or hypomineralized tissue takes up minerals again (used here in the sense of ionic exchange in enamel).

SKELETAL FLUOROSIS
This is caused by excessive intakes of fluoride from many sources including drinking water supplies. Bone fractures caused by fluoride changing the structure and decreasing the tensile strength (although increasing bone mass). Advanced skeletal fluorosis is a crippling process causing stiffness of joints and limiting joint movement.

SMOOTH SURFACE CARIES
Dental caries occurring on those gingival and approximal surfaces of the teeth which do not have a natural pattern of fissures, pits or grooves.

SUPPLEMENTARY FLUORIDE
Any form for fluoride which is used by humans in addition to the amounts to which they would be exposed through the environment, foods and background levels in drinking water (typically at a concentration of 0.3 ppm fluoride or less). Sources of supplementary fluoride include fluoridated drinking water, fluoride tablets or drops, fluoridated toothpastes or mouthrinses, and professional dental applications. Hence, the term “supplementary fluoride” here is broader in scope than that used by some others to refer only to tablets or drops.

SYSTEMIC
Affecting the whole body.

TABLETS
See Discretionary Fluoride, Supplementary Fluoride and Fluoride Vehicle.

TOPICAL
Pertaining to or acting upon a particular surface area.
TOPICAL APPLICATION
Application locally (in the mouth onto the tooth surface).

TOPICAL FLUORIDE
The application of fluoride which is intended primarily to act locally on the teeth.

TOXICITY
The fluoride ion is toxic to all life when not “bound” (as when it occurs naturally). “Free” fluoride ions may exert toxic effects.

This glossary has been compiled from the work of Dr. P. R. N. Sutton, Glen S. R. Walker, The Collins Dictionary and portions of information from the N.H. and M.R.C. (Australia).