Dental Infections Oral and Systemic

(Volume I)
(Part I)

BEING A CONTRIBUTION TO THE PATHOLOGY OF DENTAL INFECTIONS FOCAL INFECTIONS, AND THE DEGENERATIVE DISEASES

BY

WESTON A. PRICE, DDS, MS, FACD

THIS IS THE EXPERIMENTAL BASIS FOR VOLUME II

"DENTAL INFECTIONS AND THE DEGENERATIVE DISEASES"

VOLUME I
PARTS 1 AND II
PRESENTS

RESEARCHES ON FUNDAMENTALS OF ORAL AND SYSTEMIC EXPRESSIONS OF DENTAL INFECTIONS

VOLUME II
PARTS I AND II
PRESENTS
RESEARCHES ON CLINICAL EXPRESSIONS OF DENTAL INFECTIONS

WESTON A. PRICE, D. D. S., M. S., F. A. C. D.

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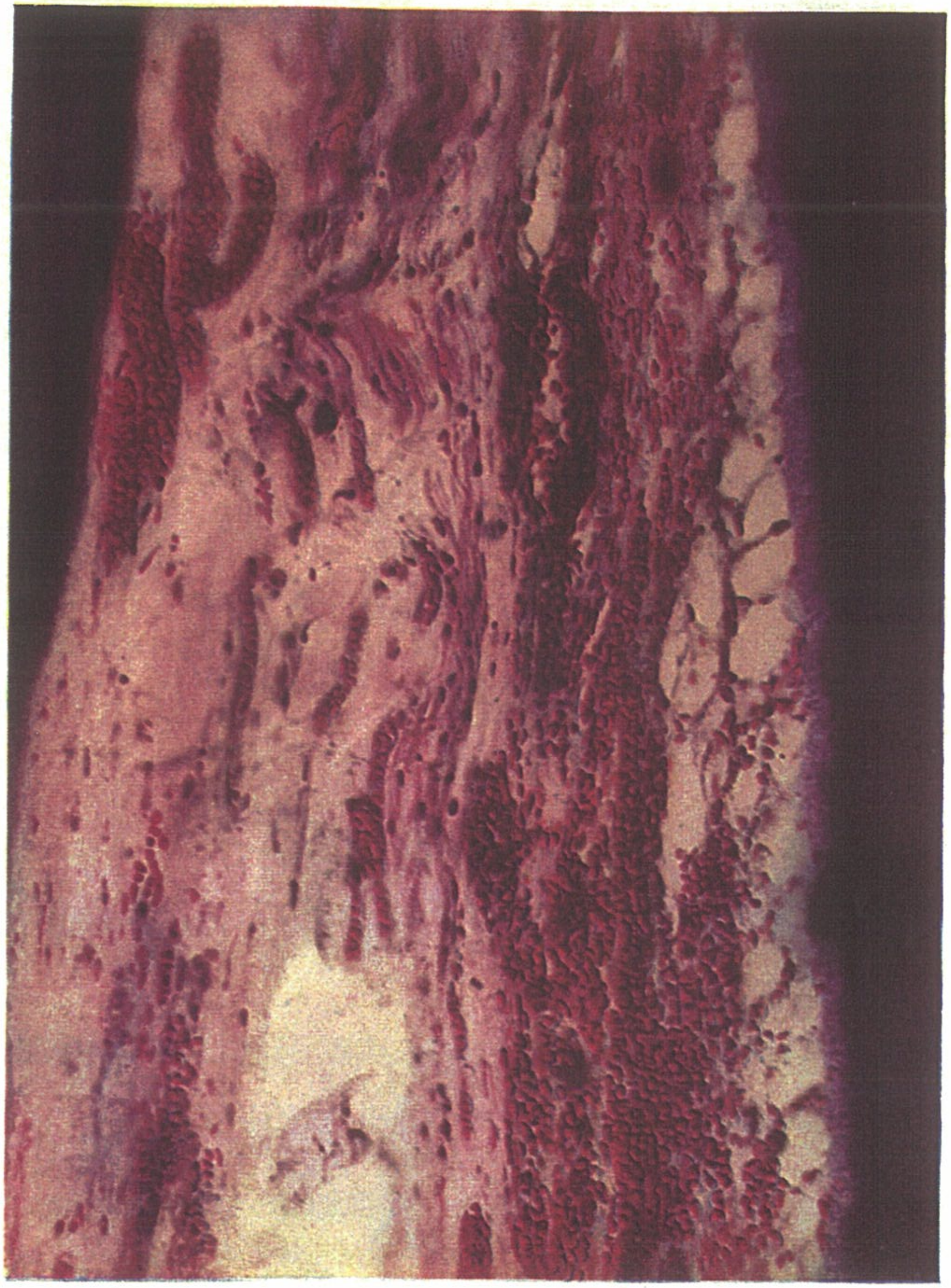
VOLUME I

RESEARCHES ON ORAL AND SYSTEMIC EXPRESSIONS OF DENTAL INFECTIONS



ACUTE PULPITIS.

SYMPTOMS—HYPERSENSITIVENESS TO THERMAL CHANGE. HISTOPATHOLOGY—INTERSTITIAL HEMORRHAGE INTO DENTAL PULP FROM INFECTED CARIES.

FRONTISPIECE VOL. I.

To

MR. AND MRS. FRANCIS E. DRURY
MY ESTEEMED FRIENDS

IN APPRECIATION OF
THEIR KINDLY ENCOURAGEMENT AND HELPFUL INTEREST
THIS VOLUME IS AFFECTIONATELY DEDICATED

A New Truth a New Sense

"THE acquisition of a new truth is like the acquisition of a new sense, which renders a man capable of perceiving and recognizing a large number of phenomena that are invisible and hidden from another, as they were from him originally." LIEBIG. *Chemische Briefe*

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RHEUMATIC GROUP LESIONS OF INDIVIDUALS AND THEIR RELATIVES

Susceptibility Studies by Alphabetical Groups

Alphabetical Group	Total Number	Average Age	Male Number	Average Age	Female Number	Average Age
A	18	41.6	5	44.6	13	40.4
B	28	42.0	6	37.1	22	43.3
C	41	42.0	15	41.7	26	42.2
D	3	36.0	0		3	36.0
B2	46	43.9	14	47.6	32	42.3
D	27	43.0	8	47.6	19	41.0
E	11	42.6	3	38.7	8	44.1
F	11	43.0	7	39.6	4	49.0
FG	59	43.4	18	48.3	41	41.2
H	44	44.0	6	51.5	38	42.8
I	4	43.2	0		4	43.2
J	17	37.4	4	39.0	13	36.8
K	33	37.2	10	36.2	23	37.7
L	29	38.0	8	39.6	21	37.4
M1	12	44.2	3	37.7	9	46.3
M2	56	37.4	12	36.1	44	37.7
P	35	42.2	11	49.0	24	39.1
NO	18	35.3	5	39.4	13	33.7
R	17	44.9	7	46.6	10	43.7
S1	36	43.2	13	47.4	23	40.9
S2	20	41.4	8	44.2	12	39.5
T	25	44.1	8	40.6	17	45.7
V	9	44.4	5	39.2	4	51.0
W	55	39.7	15	40.5	40	39.4
YZ	9	34.5	2	31.0	7	35.6
S3	18	41.7	6	43.2	12	40.9
Totals	681	1070.3	199	1006.4	482	1070.9
		41.2	29%	41.9	71%	41.2

Alphabetical Group	Number of Lesions of Individuals: Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues
A	13	13	5	12	16	12	14
B	16	17	10	17	25	18	23
C	22	22	12	21	29	23	24
D	1	3	2	3	3	2	3
B2	16	29	12	28	39	33	26
D	13	18	6	18	22	13	19
E	8	7	2	6	11	10	7
F	8	5	4	7	9	8	9
FG	28	33	17	38	48	31	43
H	19	32	8	28	33	26	26
I	3	3	2	2	3	3	3
J	7	8	4	12	14	8	14
K	21	20	3	18	29	20	22
L	16	18	9	21	26	20	22
M1	4	8	2	8	11	10	11
M2	34	36	13	29	45	31	41
P	13	23	6	18	24	22	19
NO	9	13	10	11	14	8	12
R	7	11	2	7	12	7	10
S1	23	23	6	21	30	24	21
S2	8	13	6	11	16	10	12
T	16	18	8	20	24	17	21
V	2	3	2	6	8	5	8
W	35	35	15	34	43	29	38
YZ	7	5	5	6	7	5	6
S3	11	12	5	14	14	11	14
Totals	360	428	176	416	555	406	468
Average Number of Breaks per 100 Individuals in	53	63	26	61	81	60	70
	Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues

Alphabetical Group	Number of Relatives Affected: Brothers	Sisters	Father	Father's Relatives	Mother	Mother's Relatives
A	13	13	15	9	16	11
B	12	20	24	14	21	14
C	19	23	30	16	25	21
D	2	2	2	1	3	3
B2	23	27	34	17	32	22
D	16	10	22	9	21	9
E	7	8	7	4	5	7
F	8	5	9	5	8	6
FG	25	22	35	16	47	28
H	20	21	33	14	37	24
I	3	2	3	1	3	1
J	9	8	12	4	11	5
K	19	21	27	14	27	16
L	15	19	21	15	23	19
M1	7	7	11	2	10	7
M2	27	29	41	22	48	33
P	12	12	22	12	27	19
NO	9	9	15	8	14	10
R	10	6	12	4	12	8
S1	14	14	26	18	26	15
S2	10	11	16	6	15	8
T	15	10	17	12	21	14
V	6	4	3	3	8	2
W	30	29	41	24	46	29
YZ	6	3	5	4	8	3
S3	9	13	15	9	15	12
Totals	346	348	498	263	529	346
Percentage of Relatives Affected	51	51	73	39	78	51
	Brothers	Sisters	Father	Father's Relatives	Mother	Mother's Relaives

Alphabetical Group	Number of Lesions in Individuals and their Families: Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues	Total Severe	Per cent Severe	Total Severe and Mild	Per cent Severe and Mild
A	28	65	35	23	131	50	37	278	15.4	369	20.5
B	37	69	39	29	164	96	57	310	11.1	491	17.5
C	47	98	48	35	163	139	66	417	10.2	596	14.5
D	8	20	14	9	29	10	24	53	17.6	114	38.0
B2	29	93	50	38	239	157	58	495	10.8	664	14.4
D	27	76	34	33	156	71	70	302	11.2	467	17.3
E	9	24	8	9	42	38	16	106	9.6	146	13.3
F	18	30	18	12	33	42	27	97	8.8	180	16.4
FG	56	108	73	51	281	137	111	543	9.2	817	13.8
H	37	110	49	45	208	148	69	486	11.0	666	15.1
I	3	19	7	3	46	21	13	85	21.2	112	28.0
J	15	28	16	14	62	37	22	128	7.5	194	11.4
K	58	120	42	57	294	158	115	616	18.7	844	25.6
L	31	72	36	38	194	86	64	360	12.4	521	17.9
M1	9	32	9	14	73	41	20	155	12.9	198	16.5
M2	71	152	79	46	320	179	114	657	11.7	961	17.2
P	35	71	47	29	147	97	65	300	8.6	491	14.0
NO	29	71	43	34	128	78	54	317	17.6	437	24.3
R	12	35	10	9	68	46	20	147	8.6	200	11.8
S1	32	72	47	31	160	121	39	370	10.3	502	13.9
S2	16	42	26	16	89	50	35	183	9.1	274	13.7
T	28	62	45	27	155	70	41	320	12.8	428	17.1
V	2	15	3	9	48	13	16	70	7.7	106	11.7
W	65	131	96	51	289	164	102	628	11.4	898	16.3
YZ	12	21	13	7	41	24	20	91	10.1	138	15.3
S3	24	54	29	21	99	53	43	208	11.6	323	17.9
Totals	738	1690	916	690	3659	2126	1318	7722	307.1	11137	453.4
									11.8		17.4
Average Number of Breaks per Family in	1.08	2.48	1.35	1.01	5.37	3.12	1.94	11.34	11.34	16.35	16.35
	Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues	Average Severe		Average Severe and Mild	

Alphabetical Group	Inherited: No. Pat's. in Group	Severe	Average Severe	Severe and Mild	Average Severe and Mild	Acquired: No. Pat's. in Group	Severe	Average Severe	Severe and Mild	Average Severe and Mild	Absent: No. Pat's. in Group	Severe	Average Severe	Severe and Mild	Average Severe and Mild
A	11	220	20.0	286	26.0	6	53	8.8	77	12.8	1	5	5.0	6	6.0
B	14	219	15.6	301	21.5	8	60	7.5	113	14.1	6	31	5.2	77	12.8
C	20	307	15.3	431	21.5	10	62	6.2	85	8.5	11	48	4.4	80	7.3
D	3	53	17.7	114	38.0										
B2	25	365	14.6	473	18.9	15	113	7.5	160	10.7	6	17	2.8	31	5.2
D	13	201	15.5	326	25.1	8	50	6.2	77	9.6	6	51	8.5	64	10.7
E	5	67	13.4	86	17.2	4	30	7.5	45	11.2	2	9	4.5	15	7.5
F	4	48	12.0	83	20.7	3	26	8.7	50	16.7	4	23	5.7	47	11.7
FG	32	388	12.1	583	18.2	19	130	6.8	198	10.4	8	25	3.1	36	4.5
H	30	401	13.4	533	17.8	7	57	8.1	81	11.6	7	28	4.0	52	7.4
I	3	84	28.0	111	37.0						1	1	1.0	1	1.0
J	8	91	11.4	125	15.6	6	31	5.2	51	8.5	3	6	2.0	18	6.0
K	17	505	29.7	663	39.0	10	89	8.9	142	14.2	6	22	3.7	39	6.5
L	21	311	14.8	441	21.0	4	27	6.7	48	12.0	4	22	5.5	32	8.0
M1	8	120	15.0	151	18.9	2	26	13.0	31	15.5	2	9	4.5	16	8.0
M2	39	568	14.6	804	20.6	14	77	5.5	140	10.0	3	12	4.0	17	5.7
P	17	215	12.6	358	21.1	11	51	4.6	85	7.7	7	34	4.9	48	6.9
NO	14	301	21.5	412	29.4	3	13	4.3	20	6.7	1	3	3.0	5	5.0
R	9	90	10.0	122	13.6	7	57	8.1	78	11.1	1	0	0.0	0	0.0
S1	24	300	12.5	404	16.8	4	31	7.7	47	11.7	8	39	4.9	51	6.4
S2	13	148	11.4	207	15.9	5	30	6.0	54	10.8	2	5	2.5	13	6.5
T	19	282	14.8	374	19.7	3	26	8.7	37	12.3	3	12	4.0	17	5.7
V	4	41	10.2	63	15.7	5	29	5.8	43	8.6					
W	34	524	15.4	730	21.5	12	80	6.7	117	9.7	9	24	2.7	51	5.6
YZ	6	78	13.0	108	18.0	3	13	4.3	30	10.0					
S3	12	168	14.0	261	21.7	5	36	7.2	54	10.8	1	4	4.0	8	8.0
Totals	405	6095	398.5	8550	570.4	174	1197	170.0	1863	265.2	102	430	89.9	724	152.4
Average Number of Lesions Severe, and Severe and Mild, per Susceptibility Group	Susceptibility Inherited					Susceptibility Acquired					Susceptibility Absent				
	59	15.05	15.3	21.11	21.9	26	6.88	7.1	10.71	11.0	15	4.22	4.1	7.098	6.9
		Average Severe		Average Severe and Mild			Average Severe		Average Severe and Mild			Average Severe		Average Severe and Mild	

(Note: This chart is a consolidation of 140,000 determinations some of which took minutes, and some hours of time.)

The summary at the right (bold face) is an analysis of the susceptibilities of the patients and their families to the rheumatic group lesions

SUMMARY

Susceptibility *Inherited*		Susceptibility *Acquired*		Susceptibility *Absent*	
Average Severe	15.05	Average Severe	6.88	Average Severe	4.22
Average Severe & Mild	21.11	Average Severe & Mild	10.71	Average Severe & Mild	7.089

FIGURE 41.

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PREFACE

THE PURPOSE of these two volumes is to present new data and important new interpretations suggested by them. I wish to assure the readers of these volumes that I am not unmindful of the tremendous responsibility that is involved in my presuming to furnish to the medical and dental professions a new interpretation of the pathology of oral and systemic expressions of dental infections and of their role in the production of the degenerative diseases. Notwithstanding this great responsibility, however, I have a sense of deep confidence that the new interpretations, I am herewith presenting, more adequately harmonize the available evidence and clinical findings than do any that we have had heretofore.

This title presumes that dental infections have been demonstrated to be an important contributing factor in the production of the degenerative diseases. I have no hesitancy in leaving to the evidence herewith submitted, whether I am justified in using this title; and now that we see it in the new light, we understand with a kindly sympathy the misapprehensions and the causes for the confusions of the past. It is probable that there seldom has been and seldom will be in the history of humanity, so universal a misapprehension based upon misconceptions, if we may judge importance on the merit of factors involved. Briefly stated, this misapprehension has been this. We have mistaken effect for cause. Since everything is relative, infection has had to have a quantity factor, and that quantity factor has had to be measured. The measure has been the structural change at the point of the focus. This has presupposed that quantity and virulence of organism, on the one hand, determined in large part the danger to the host. With this as the fundamental, it has been practically universally conceded that comfort and serviceability were dependable symptoms of safety and efficiency. But this being the fundamental conception, it has been most natural that exceptions to this rule would challenge the presumption that a large enough quantity of dental infections would do harm, whereas a small quantity would not. There has, accordingly, been a paradox that heretofore has been unanswered, and which probably has been the basis for nearly all of the opposition to the proposition that dental *infections could do systemic harm. Such a one has been the following:*

In the various out-clinics of hospitals and in such groups where large numbers of individuals could be observed, it has been continually noted that those individuals with apparently the largest quantity of pus exuding from infected roots, and particularly from apical fistulæ, had no rheumatism, heart, or kidney involvement; and in those cases where these lesions did exist, there was no such evidence of discharge. If, then, these individuals with so much infection were not involved, why strain the point so far as to assume that less infection was the cause in these other individuals? The fundamental conception has been wrong, for the individual with the large quantity of pus, as evidenced by the flowing fistulæ, did not necessarily have more infection that those of the other group who, with similar conditions, had no such physical expressions. The difference is in the individuals of the two groups, and this is one of the important new truths that these volumes will bring, and is a difference in the capacity for reaction. The teachings of the past have assumed that there was a distinct difference in the attacking power and virulence as well as the quantity factor involved in different dental infections. These reports will show that our problem is not one, primarily, of the morphology and biological characteristics of the strain involved, but, on the contrary, any strain of the streptococcus group, which may chance to get into that environment, will tend to produce the same unit characteristics, and that these characteristics will, because of the very great adaptability of these organisms, be the resultant of the pabulum furnished by the host as the culture medium for the strains involved.

Further, I have shown that an individual's defense for the streptococcal group of infections is primarily a matter of a special defensive factor or mechanism for the streptococcus group, which factor he inherits from his ancestry just as he inherits all his other unit characters, that this quality has a unit basis in relation to individual organs quite independent of the entire body. This normal defensive factor is a relative one and is subject to modification through a wide range as a result of overloads. The individual, therefore, with a high defense, expresses that defensive capacity immediately about the tooth, for he efficiently resists the invader immediately about the point of entrance. This warfare will be shown to be a matter of the establishment and maintenance of a local and systemic quarantine, the mechanisms for the development and maintenance of which are brought out in succeeding chapters. I have shown that these very defensive factors are all measurable and can be expressed *quantitatively by chemical analyses of various factors of the blood and by determining the bactericidal properties of the blood. It is a most significant and lamentable fact, that there has been practically no important progress in our understanding of the etiology of periodontoclasia, or pyorrhea alveolaris, of the etiology of dental caries, or of the true relation of dental infections to systemic disease except in the most general terms, in a whole century, and this, fundamentally, because the accepted doctrines had no basis in experimental pathology, but were a matter of inheritance from preceding generations and were the logical assumptions. These new interpretations, which I furnish herewith, as well as the data from which I have drawn them, adequately explain the dental paradoxes as being precisely what we should have expected, and as being in complete harmony with this newer view.*

This work must stand or fall absolutely on its merits in this regard, and I have no hesitancy in sending this bark out into the storm which I know must follow, and I have no desire that it shall weather the storm if its cargo is not entirely that of truth. The very large scope of the presentation precludes the possibility of the detailed argument and presentation of data that will be desirable simply in a critical review. I have, accordingly, undertaken to make this serve the double purpose of being directly applicable to clinical practice (for it has grown out of the most exacting and intimate study of clinical relations) and to furnish an adequate amount of experimental data to establish and justify the new interpretations. The data, that I am furnishing, are only a small fraction of what I have available.

With regard to the timeliness of such a message, there is no question or doubt.

This can perhaps best be summarized by suggesting the present stage of advancement of general medical and dental knowledge. This was splendidly done at the recent meeting of the American Association for the Advancement of Science (Boston, December 26-30, 1922) when a general session was addressed by Dr. Livingston Farrand, president of Cornell University, on the subject, "The nation and its health." Science, in abstracting his address, stated:

"Dr. Farrand reviewed the progress of public health work in this country and pointed out that since 1870 the average length of life has been increased by fifteen years, that marked reduction has occurred during this period in infant mortality and in mortality due to tuberculosis, typhoid, smallpox and many other diseases. The efforts of health workers and organizations have, however, been unable thus far

to prevent increases in certain unconquered diseases, such as cancer and diseases of the heart and kidneys. The most outstanding problem at present concerns the control of the degenerative diseases of later life, an increase in mortality from these being an inevitable consequence of improvements in the control of diseases of infancy and youth."

If, as I interpret these researches to demonstrate, the degenerative diseases to which he refers, particularly of the heart and kidneys, are very markedly increased in their severity and in many instances actually caused by dental focal infections, there probably is no more important problem for our modernly civilized communities than the study of means for the prevention, in every way possible, of these degenerative processes.

Few, if any, of the contributors to medical science have shown a greater appreciation of this need and deserve greater credit for the danger signals given to the profession and humanity, than Sir William Hunter. In a recent discussion by him before the Medical Society of London (December 11, 1922) he called attention to the fact that the present discussion in which he was taking part was the first which had taken place on oral sepsis before that society since the subject originated in its newer phase in the paper presented by him before that society twenty years ago. In the paper which he was discussing Sir William Willcox had given a general resumé of the literature without new experimental data and had stated that he agreed with Dr. Beddard who had expressed the opinion that 90 per cent of the non-specific infective arthritis cases were due to infection arising from the teeth. Lord Dawson in closing the discussion stated that the subject afforded a very good example of a necessity for teamwork, that what was wanted was some really connected work upon the subject to which dentists, radiographers, and bacteriologists would all contribute.

In conducting the researches herewith reported, I have undertaken to secure the closest coöperation possible by engaging men for my staff, whose exclusive attention has thereby been concentrated on the particular phase for which they were engaged. There has, therefore, been the closest possible coöperation without the possibility of distraction or conflicting purpose; and I am profoundly indebted to these collaborators, who have been many, during these two and one-half decades, in working on these problems.

No work on this subject can be presented at this time, if at any time in the future, without recognizing in a very important way the *exceptional pioneer work that has been done by Dr. E. C. Rosenow, first while working in Chicago at the Presbyterian Hospital in association with Dr. Frank Billings, and latterly in the Mayo Institute at Rochester, Minnesota. Probably to Dr. Billings more than to any other American internist is due the credit for the early recognition of the importance of streptococcal focal infections in systemic involvements, for his work practically paralleled that of Sir William Hunter in England.*

I wish to express my deep indebtedness to all these pioneers in this field; and if my work shall have removed some of the confusions which have been largely responsible for the lack of appreciation of, and opposition to, the efforts of these great pioneers, I should be doubly glad because of my esteem for their courage in the midst of the bitterest of opposition, and also for the larger helpfulness that may come to humanity by a more universal medical and dental appreciation of this need. There could not possibly be a stronger tribute to the sincerity of these men than that they should so persistently follow the line of their conviction in the midst of the unprecedented antagonism, for theirs was the vision of a great new truth.

It is my judgment that the most important phase of this contribution will not be simply the correcting of a misconception of fundamental dental pathology, but the making of an important new contribution to the pathology of focal infections and the dengerative diseases. In the light of the succeeding chapters there is strong evidence that the degenerative processes which we have thought of as various diseases, such as Bright's disease, heart disease, nervous system involvement, digestive tract disfunction, etc., etc., are primarily the end products of disturbed processes of metabolism and catabolism, and that an important contributing factor to these disfunctions will be found to be focal infection, whether of dental or other origin. Since, however, they develop most largely in adult life, more than 95 per cent of the members of the human race will be found to have a source in the form of an infected non-vital tooth for the disturbers of the hormones which control organ and tissue functions. The evidence in these chapters will take the form of the measurement of these factors, the reproduction of the various cycles of animal experimentation, and numerous evidences of the elimination or betterment of the human physical disfunction and organ degeneration following the removal of the dental infection.

While the preparation of this text has involved a series of researches extending over more than twenty-five years, it has not seemed to me *wise to publish a less complete statement for the following reasons:*

The earlier researches involved seemed to establish that the current fundamentals, as universally accepted, were not based on truth; and I soon learned from the presentation of papers and illustrated lectures that the bringing to the profession of a negative statement, simply challenging the old fundamentals without putting something in their place, was a very unwelcome message. The role of an iconoclast is seldom, if ever, a happy one.

Second, while it was a relatively simple matter to demonstrate that the accepted fundamentals were in error, it has been a tremendously difficult matter to develop new working hypotheses that would stand the most critical test that I have been able to put to them.

Third, a new interpretation must, by the very nature of things, and it is well that it is so, run a gauntlet of intensive criticism, which not only is right, but becomes a purifying fire; for only by the most exacting tests should new truths presume to supplant old ones.

It is a matter of deep regret to me that so much of my energy has had to be expended in the business side of dentistry in order that the means might be available for conducting these investigations, which has not left an adequate amount of time and strength to perfect these volumes to a greater degree.

It has been impossible because of the voluminous presentation, for me to include an historical review and bibliography for each chapter, which, in themselves, would add many hundred pages.

These researches have required the use of approximately five hundred rabbits a year, for several years; and, for those who would criticize their use, I wish to state that many of these rabbits have in my judgment made a far greater individual contribution and service to the welfare of humanity than hosts of human beings. Rabbits that run wild and are chased by their enemies have not been as well fed and as happily housed, or been privileged to die under chloroform. I have had many patients express their gratitude and confidence by offering themselves for any experiments that I would care to try upon them, if, by so doing, they too could help humanity. The greatest tragedy that I see in the whole development of this subject in the past, has been that humans alone have been used as the experimental material and the experiments have not been properly checked; for it has been considered that comfort and serviceability were a sure proof of the success of the experiment, entirely misapprehending that a lack of reaction about the tooth, and the consequent comfort, only meant that the quarantine was not in operation and the toxin and bacterial invasion *were passing to other parts of the body, there to break down tissue and shorten life.*

I am deeply indebted to many persons for assistance in the development of this work. First of all, I want to pay the highest tribute possible to the patients who have given me every possible coöperation. While I have no doubt they felt a gratification for relief given them from distressing symptoms, and distinct improvement in health and comfort of living, the spirit in which they have coöperated not only by paying liberally for the services as they were charged to them, but by material contributions made in many instances to the work, without which it could not have been so efficiently conducted. For those who are unfamiliar with the unusual expense of this type of work, it will be of interest to note that the research work involved in these two volumes has cost in excess of $250,000, which has been provided almost entirely by the fees from the patients.

I wish especially to thank all the members of my staff averaging sixteen in the last three years, and seldom less than five during the preceding twenty-five years in which I have been doing this work. It is true that we have had a common joy in this service to humanity, but I cannot pay too high a tribute to the earnestness and completeness with which they have joined in the search for these new truths. I cannot imagine a greater joy in any enterprise than that which they have given me by the earnestness and completeness of their coöperation. This work could not have been done without this superior assistance and coöperation. The length of the list, and the extent of the time over which the work has been in progress, preclude their individual mention.

I am also deeply indebted to several friends who have given me encouragement that has been exceedingly helpful, and who, by their deep interest and constant encouragement, have helped me to overcome obstacles that seemed very forbidding.

Volumes One and Two, herewith presented, are so interrelated and inseparable in context, being but different phases of the same problem, that they are treated as a unit and neither volume should be considered apart from its cross references to the other. Those who are familiar with the expense of illustrations, and particularly of four-color process engravings, will appreciate that no trouble and expense have been spared. The color separation negatives for the four-color histopathological plates were made by us directly from the tissue sections (not Lumière or Paget), which is, we believe, a distinct advantage over hand-colored photographs or drawings, all of which *introduce the personality of the artists.*

I wish to thank the printer and the various engravers for their splendid coöperation and assistance in presenting this message.

Weston A. Price.

8926 Euclid Avenue
Cleveland, Ohio
August, 1923

INTRODUCTION.

THE GENERAL STATUS OF HUMANITY AND THE HEALING PROFESSIONS.

Since a new truth is a new sense, because with it an individual can see things that he could not see before he had that new truth, and things that persons who have not that truth cannot see, it must follow that the blindness of ignorance is not only the greatest inconvenience but the greatest affliction to humanity. The tragic position in which our modern civilization finds itself with regard to dental infections and their local and systemic effects, is emphasized by comparison with the absence of similar suffering among many less civilized peoples, and it is entirely probable that the coming of many of the new comforts which have amounted to reformations in our methods of living, has exaggerated to a great extent our present conditions. There exists today, in the minds of the members of the laity as well as in those of the members of both the medical and dental professions, a confusion of ideas regarding dental infections that amounts to a group of misapprehensions and contradictions leading everywhere to uncertainty. We feel sure there is no other major affection about which there is this maze of uncertainty and apparent contradiction. The purpose of this volume and of the extended researches which it represents, has been pursued and inspired with the hope that it will aid by bringing some fundamental new truths which will furnish us all with an additional or new sense which we may term a *dental infection sense.*

In approaching this subject we should have in mind that dental disease is one of the most universal of all the afflictions of humanity. As has been emphasized, only a few isolated tribes or limited civilizations have been or are free from its curse. According to our statistics, over ninety per cent of the children in the United States have dental infection in some form. The statistics for the cause of absence and tardiness in school work show dental infections to be the chief factor, and these largely because of the immediate discomfort from dental caries. This, however, does not constitute the most serious phase of dental infections:

namely, their systemic expressions. I am more and more impressed and convinced after more than twenty-five years of intensive study of this problem, that the members of the healing professions have generally a quite incorrect conception of the variety and extent of the systemic expressions of dental infections, and this because of the absence of this new sense which is just developing with the coming of new, though meager truths.

Surely, the lay humanity is helpless to save itself, and, as with all the other scourges, so many of which have been removed by the coming of new senses through new truths, it is dependent upon the healing professions for relief and ultimate rescue. Unfortunately, many difficulties have combined to aggravate and complicate the already obscure problems. In the first place, the lack of coöperation between the medical and dental professions has been an incalculable hindrance and disadvantage to both the progress of healing science and the well-being of society. Much of the research that has been done on this problem has been by those who, by the very nature of things, have been handicapped by an opportunity to check every detail and check up against the clinical. The clinicians have had neither training nor time available to interpret their clinical findings in terms of laboratory procedure. The whole problem has been clouded by a group of contradictions of symptoms which, probably more than all other matters combined, has delayed progress. What could be more convincing to the medical and dental practitioners that dental infections do not have systemic expressions than to find that the majority of people examined with extensive dental infections, and some exceedingly so, have as yet no systemic disturbance; and the converse, that the people suffering severely from obscure disturbances, have relatively slight evidence of dental infection? If some of the new truths we are presenting in this volume are fundamental and correct, as we believe they are, they not only harmonize this apparent contradiction but suggest that these are just what we should expect.

The growing tendency for coöperation between the medical and dental professions, so long withheld, is a first requisite in humanity's behalf. However, until our dental colleges teach more of general medicine and clinical pathology, and the medical colleges teach more of dental pathology, both local and systemic, humanity must wait and suffer. The slow rate of progress of the past can only be accelerated by the perfection of organization and equipment for research in this field, a first requisite of which is the closely coöperating group of specialists working with such complete harmony that every step is interpreted in terms of all those factors which are held in common, as well as those which relate chiefly to the field of each specialist. To be more specific, the solving of these problems requires the skill and experience of each of the following: a well trained clinical dentist who knows every detail of the steps in the various operations and of the dental structures, physical, chemical, anatomical, and surgical, the histopathologist, biological chemist, general pathologist, serologist, and internist.

We cannot conceive of anything more monumental and personal than the responsibility of each and every member of the healing professions for the solution in humanity's behalf of this problem. The outstanding feature of the present moment is the utter meagerness of the effort that is being made to solve this problem in proportion to humanity's need. If we would compare this effort with that, say of farm stock or timber land, it is so insignificant as to be a cause for deep chagrin and humiliation, if not a consciousness of a misplaced trust on the part of humanity. This has been looked upon as a problem of dental research and, as such, has been left largely to the dental profession. Endowments have not been made available and the few workers in the dental profession who have had a vision of the responsibility and opportunity, have been completely handicapped by lack of moral and financial support. Their feeble strength has gone out in battering against a great wall of impenetrable difficulties ahead, while they have been flanked on each side by indifference and lack of equipment. This is one of humanity's greatest problems today and, as such, is worthy of the most splendid support, moral and financial, that can be given to any of its interests.

ANALYSIS OF PRESENT CONDITIONS.

THE GENERALLY ACCEPTED BASES FOR THE INTERPRETATION OF DENTAL INFECTIONS.

Each dental caries, dental abscess, gingival and alveolar inflammation and necrosis, has been interpreted as essentially infective processes, and hence their extent is essentially a measure of the infection. It now seems strange that we should have stumbled so long and interpreted the effect, as expressed in extent, as a measure of the cause. It has generally been accepted that the infection is produced by organisms presenting specific attacking powers for the dental and supporting tissues involved, and particularly in the case of gingival infections it has been looked upon as being largely a bacterial invasion, contracted by contamination with the involved organism. It has been anticipated, that the first step in the eradication or prevention of these diseases would, of necessity, be the identification of the specific causative variety or strain of organism, which would be followed by a suitable warfare against it. We have looked upon various individuals as comparable, and hence the effects produced by organisms as comparable. On these premises we have accepted a tentative set of rules of interpretation, formulated for, and applied to, all and various members of the human family. These rules, as generally accepted and applied, are about as follows:

1. Human beings are similar, and therefore comparable in their reactions to dental infections.

2. That dental infections, when they occur at the apices of roots, are produced by the invasion of that area by organisms from the mouth which enter that tissue through the open pulp canal; and that the question of danger from such an infection is dependent upon the invasive qualities of that organism.

3. That roentgenograms of teeth will reveal the presence of infection.

4. That infection will express itself as absorption.

5. That the apparent extent of the absorption is the extent of the infection.

6. That a given dental infection will express itself in the local tissues of the mouth approximately the same in all people.

7. That a tooth without visible absorption at its apex is not infected.

8. That a tooth with visible absorption at its apex is infected.

9. That pulps of teeth not exposed by caries are not infected.

10. That pulps of teeth with periodontoclasia pockets not involving the apex are not infected.

11. That an area of absorption, if present, can be disclosed by the roentgenogram.

12. That the quantity or extent of the absorption is a measure of the danger.

13. That flowing pus from a fistula is, necessarily, very dangerous to the patient.

14. That infected teeth can be sterilized readily by medication.

15. That usual medications do not injure the supporting structures.

16. That root fillings fill pulp canals and continue to do so.
17. That even a poor root filling is better than none.
18. That so called modern dentistry is a great and unmixed blessing.
19. That the field of the dentist is the oral cavity.
20. That a dentist or physician, and especially experts, can look at roentgenograms of the teeth and diagnose what should be done for the patient.
21. That local comfort and efficiency of treated teeth are an evidence and measure of the success of an operation.
22. That when infected teeth produce disturbances in other parts of the body, it is primarily because the patient is overwhelmed by a large quantity of infection.
23. That the quantity of infection in a tooth with a good root filling could not be sufficient to produce serious systemic disturbance, both because there is no place for bacteria and the quantity would have to be large, even teacupfuls, since the germs in dental infections are understood to have exceedingly low virulence or disease-producing power.
24. That there is adequate information available to practice dentistry properly and safely.

ARE CONDITIONS SATISFIED BY THESE OLD AND CURRENT INTERPRETATIONS?

According to the accepted fundamentals as presented in the preceding paragraph, the individuals showing the most extensive areas of tissue absorption about dental infections should be considered to be the persons having the most serious ones; and, consequently, we should expect that our worst cases of systemic involvement would be found in this group. Notwithstanding the almost universal belief that this is so, as a matter of fact, when we tabulate after careful clinical examination, we do not find that the evidence substantiates this, for we find that the individuals with these extensive areas of absorption resulting from a given dental infection, not only frequently but generally, are the persons but slightly affected with systemic expressions during long periods of their lifetime. This seems like a paradox and has constituted one of the chief closed doors to progress in the interpretation of dental infections. Not only does the extent of bone change vary greatly in different individuals with a given amount of infection, but the type of bone change also distinctly varies with the same cause, so far as we can judge regarding kind and amount of dental infection. Again, the effect of treatment and the extent and permanency of improvement are not at all in harmony with the above accepted fundamentals; contrary to expectation, there is a wide variation in the morphology and biologic characteristics of the organisms producing each of the certain types of local tissue change and the distinct kinds of systemic disturbance. There are also group characteristics that are not satisfied by the preceding statement of fundamentals. These group characteristics are such that they cannot be harmonized or accounted for on the basis of environment, diet, race, etc.

GENERAL LINES OF INVESTIGATIONS SUGGESTED BY THE OLD AND ACCEPTED FUNDAMENTALS AND BY THE CLINICAL CONDITIONS FOR DETERMINING THE ROLE OF DENTAL INFECTIONS.

A careful review of medical and dental literature for the purpose of finding the origin of the accepted fundamentals and to ascertain whether they have been founded upon dependable observations, has demonstrated that they seem to have grown up much as Topsy did. They have crept into the literature and practice of the sciences and have been quite generally accepted, apparently without question. One of our first undertakings has been, therefore, to check over, as carefully as we might, to determine to what extent they were based upon fact. Consequently, an enormous amount of really constructive research has consisted in proving that certain accepted beliefs were not true. None, but those who have been so placed, can know the misfortune of being placed in the position of tearing down the foundations and being termed an iconoclast, about which hang some tragic chapters of dental history. It has, however, been necessary to build new foundations deep down into the substrata of physics, biology, and chemistry. We will not review in detail, for lack of space, a large volume of research work that has really been negative in result, in that it has only proved that the accepted theories were incorrect. In general, we have undertaken to determine to what extent teeth are infected and what the nature of that infection is; to what extent routine procedures have been efficient in eliminating dental infection; the efficiency of root-filling methods as applied from a physical and mechanical standpoint; the general biological laws underlying susceptibility to dental caries and to gingival infections; the local expressions of dental infections; the tendency to development of systemic expressions of dental infections; the biologic properties of the organisms involved; the acquired factors which modify these susceptibilities; the influence of each diet, environment, habits, altitude, physical and mental states, and age. This work has covered a period of over twenty-five years, during which we have used many hundreds of animals and have engaged the assistance of skilled bacteriologists, physicists, chemists, histopathologists, and serologists; and the deductions we are here making should be received as a preliminary report since we have not sufficient information, as yet, to make us certain that we are able to interpret correctly, and will expect to make additions and modifications as further information becomes available.

Some of humanity's unsatisfied conditions are the following:—

Why do some individuals tend so readily to have periodontoclasia, while others have practically no tendency to it?

Why does dental caries become a constant menace for some individuals, while others have practically no tendency to it?

Why is it that when teeth are extracted for some individuals the conditions found are so unlike those anticipated, as judged by symptoms and roentgenographic appearance?

Why do some individuals always have much trouble from the healing of the sockets after extraction, while others have none?

Why are tissues about some teeth so easy to infiltrate with anesthesia and others so extremely hard?

What is the so-called dry socket?

Why do some individuals react with so much depression following even minor surgical operations and react alarmingly from extensive operations, and how can they be anticipated?

Why are some cases of periodontoclasia and suppurative gingivitis and alveolitis so resistant to treatment and others so amenable?

Since some teeth have, because of conditions, an intrinsic value to the patient that is very great and since similar teeth may in some cases do harm that is very great, how can we determine with relative safety when they should be retained and when removed; or otherwise stated, how can we establish the factor of safety of a given patient in relation to a given tooth?

When do given dental operations constitute a potential harm to the patient and when a potential good?

How can dental diseases be prevented?

To what extent are degenerative diseases the direct effect and result of dental disease?

Why do organs and tissues cease to properly function and ultimately degenerate, thereby wrecking the whole life either by premature death or what is often worse, a long protracted and agonizing death?

Why is it that in about ninety per cent, as estimated by high authorities, the final blow is struck by an organism, usually the streptococcus, even in patients with such involvements as pneumonia, and where do the streptococci come from?

These are some of the questions that we have undertaken to answer in these volumes.

CHAPTER I.

CAPABILITIES AND LIMITATIONS OF THE ROENTGEN-RAYS.

PROBLEM: To what extent are the Roentgen-rays capable of disclosing dental infections?

INTRODUCTION.

Since it is not my intention in this volume to furnish an historical review and criticism of the literature on the many topics that will be presented, both because it would be beyond the scope of the presentation and because it would entail such a voluminous text that it would defeat the very purpose by its energy consuming detail and unwieldiness, I will, accordingly, make important references throughout the text without purporting to make an historical review.

I have selected for presenting as the first problem a study of the efficiency of the aid in dental diagnosis which is more depended upon by the members of both the medical and dental professions than any other—namely, the Roentgen-rays—and which, because of the almost implicit confidence of the members of the medical and dental professions, the members of the laity have come to look upon as being infallible and practically limitless in application. I have divided this first problem into three sections: (a) An analysis of the fundamental that *roentgenograms are capable of revealing the presence of infection;* (b) *That the apparent extent of the absorption is the extent and volume of the infection or focal area;* and (c) *That an area of absorption, if present, can be disclosed by roentgenograms.*

EXPERIMENTAL AND DISCUSSION.

A review of the literature of the past and present regarding the applications of the Roentgen-ray for the disclosing of dental infections, together with the accepted general practice of the medical and dental professions and the clearly defined attitude of the public, which has been trained by the medical and dental professions, establishes that it is so nearly universal that it must be

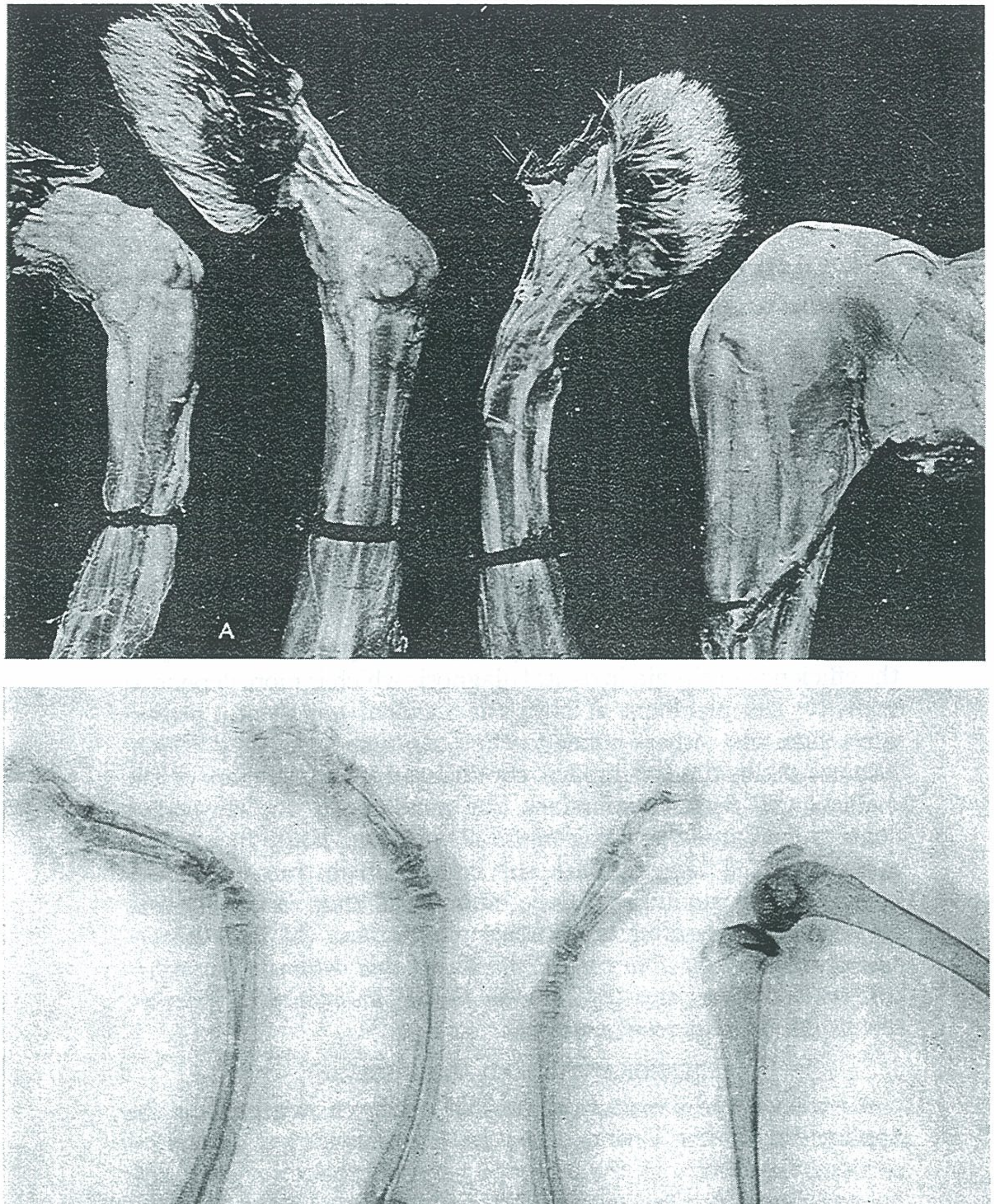


FIGURE 1. ABOVE: RHEUMATIC NODULES IN RABBITS' FEET. BELOW: ROENTGENOGRAPHIC VIEW OF SAME.

considered current belief, that the Roentgen-rays will reveal the presence of infection; particularly if present in bone, that the apparent extent is the actual extent of bone change; and that a chamber or area of lesser density, if present in soft or hard tissues, will, of necessity, be disclosed by the Roentgen-rays. No surer evidence would seem to be possible, establishing that this is the attitude of mind of members of the medical and dental professions and laity, than that the members of each continually bring roentgenograms of the teeth with the full confidence that the reading that will be made from the roentgenograms will answer the question as to what should or should not be done for that patient with regard to the teeth. An illustration of this attitude of mind is found in the current expression that the roentgenogram has made possible a living postmortem.

Our studies have included both the testing of the general principles on material that could be cut up and verified, and a careful comparison of the actual conditions as revealed at operation, with the apparent conditions as disclosed by the Roentgen-ray. It is, for example, common practice to have joints that are very badly swollen and with extensive involvements roentgenographed, in order that a proper diagnosis may be made for the patient. Figure 1 shows a photograph of three feet and one knee joint of a rabbit, each one containing extensive areas of infection. The three feet were much swollen, and on removing the skin little sacs filled with pus were present, in size and shape corresponding to garden peas. The knee, shown in A, was enlarged and dense with a pulsating mass of pus which involved the synovial membranes and joint capsule. B shows roentgenograms of these limbs; and the evidences of infection, as disclosed in the photograph, are not such as would be suggested by the roentgenograms.

Not succeeding in revealing the presence of pus in these cases, we selected two very extreme cases, as shown in Figure 2, where the quantity of pus present was so large that it was breaking out of the tissue. It will be seen that the Roentgen-ray does not reveal the presence or extent of these infections.

It is very true that we are dealing here with relatively soft tissue, the muscle, which, compared with the bone of the face,

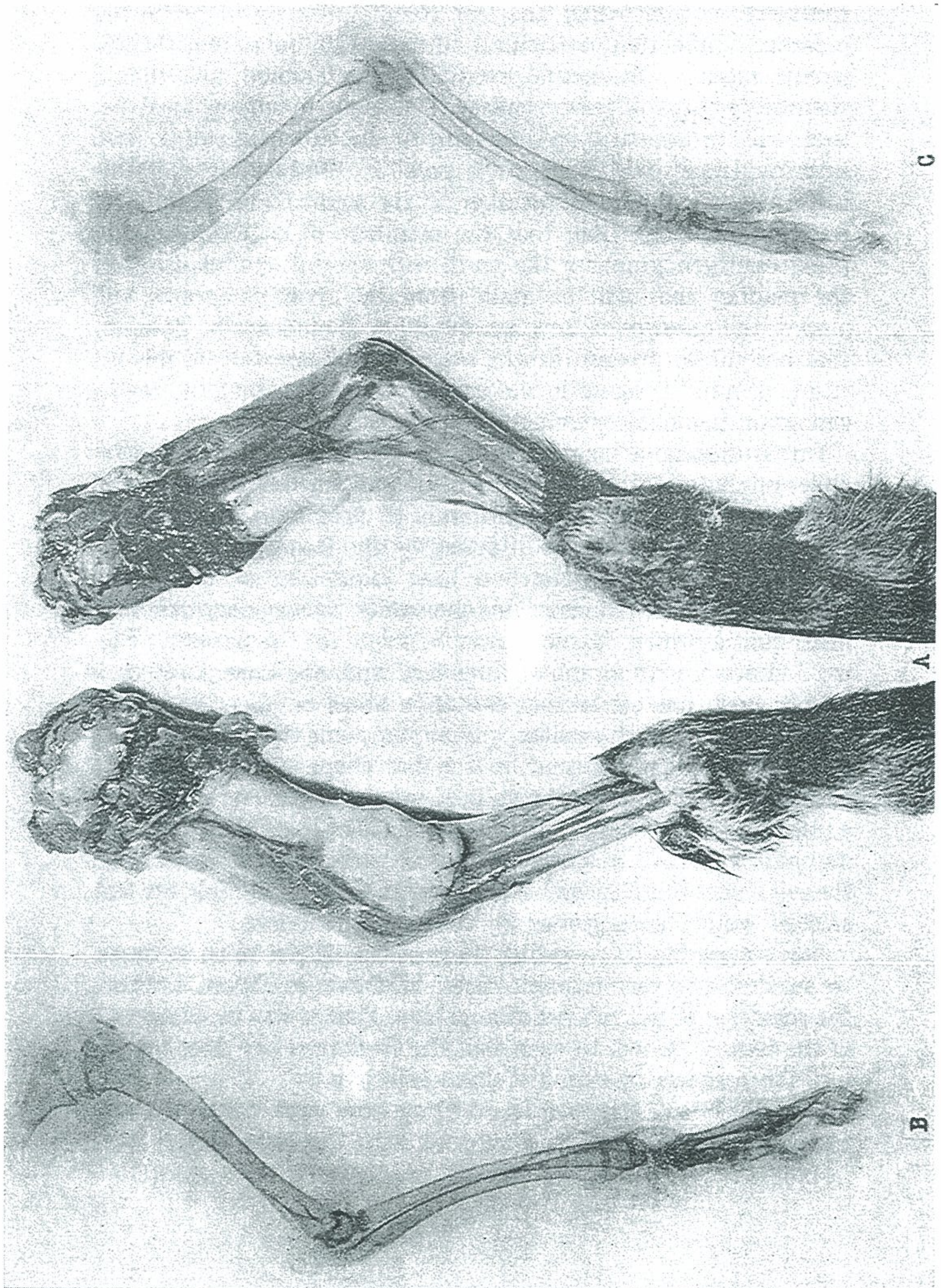


FIGURE 2. VERY EXTENSIVE PURULENT ARTHRITIS, WHICH HAS EXTENDED BEYOND THE JOINT CAPSULES AND BETWEEN THE MUSCLES. NOTE: THE ROENTGENOGRAM FAILS TO DISCLOSE THE INFECTION.

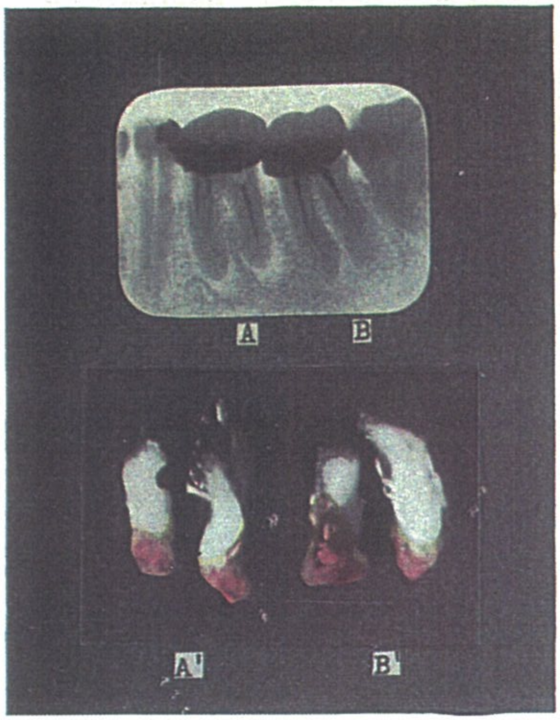


Figure 3. Comparison of the apparent with the actual. A and B show, roentgenographically, two maxillary molars. Note their difference. A′ shows the periapical granulomata attached to the roots of A, and B′ the periapical granulomata attached to the roots of B. The zones of rarefaction in A and B are obscured by zones of condensing osteitis.

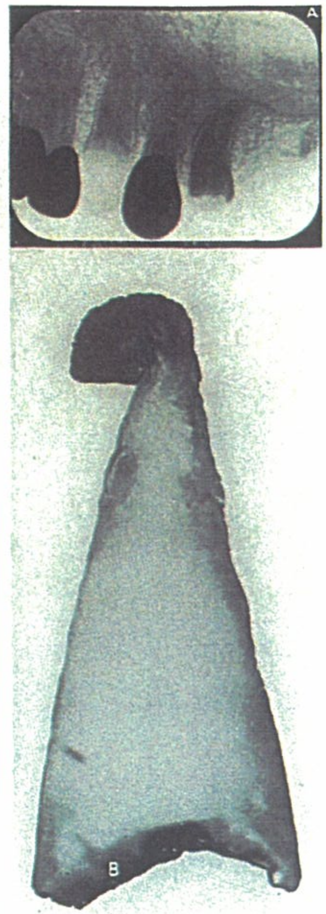


Figure 4. A, roentgenographic view of the root and apical area of a cuspid; B, an enlargement of the root and granuloma removed from this area.

may be considered to be very much less capable of differentiation, since the difference in density of pus and muscle tissue may be said to be less different than pus and bone. We have, accordingly, made careful comparisons of the apparent with the actual conditions as they obtain in the mouth. Figure 3 is a good illustration. In A and B, we have a sample of the usual roentgenographic disclosures. It is probable that not only the majority, but almost the complete personnel of the medical and dental professions and laity, would see in the first and second molars two radically different conditions of pathology. There is definite evidence of bone absorption about the apices of both roots of the first permanent molar, exceedingly little about the mesial root of the second molar, and practically none disclosed about the apex of the distal root of the second molar. A′ and B′ show the conditions of these roots when extracted, each having extensive adherent granulomata, which granulomata were larger on the second molar than the first, though they were not revealed by the roentgenogram; and the granulomata seen on the first molar were very much larger than the areas suggested by the roentgenogram. Our large accumulation of evidence of this type has crystallized our convictions into quite definite form, as expressed in succeeding chapters.

Figure 4 is another illustration. The root shown between the two gold crowns in A does not appear to have a granuloma; and yet, in B, it is demonstrated that this root when extracted had, notwithstanding its appearance in the roentgenogram, a very large granuloma. It may be argued by some that, if roentgenograms were taken from other directions or with rays having other degrees of penetration, these would be disclosed. Figure 5 shows an effort to disclose the presence or extent of pockets of periodontoclasia about a tooth. A and B show two different angles; yet neither the presence nor extent of the gingival and periodontal infection is definitely established. In C, some flexible gutta-percha points were placed in the gingival pockets and roentgenographed, which quite readily disclose much additional information. D is a roentgenogram of the extracted tooth to show the difference in the density of the root surrounded by pus and the adjoining roots. It should be noted that not even the roentgenogram shown in C discloses the actual condition; for, at the time of removal of the tooth, it was revealed that there was a continuation of the pocket through into the antrum.

It is common practice to determine the efficiency of root fillings as they exist in the mouth by means of the Roentgen-ray, on the

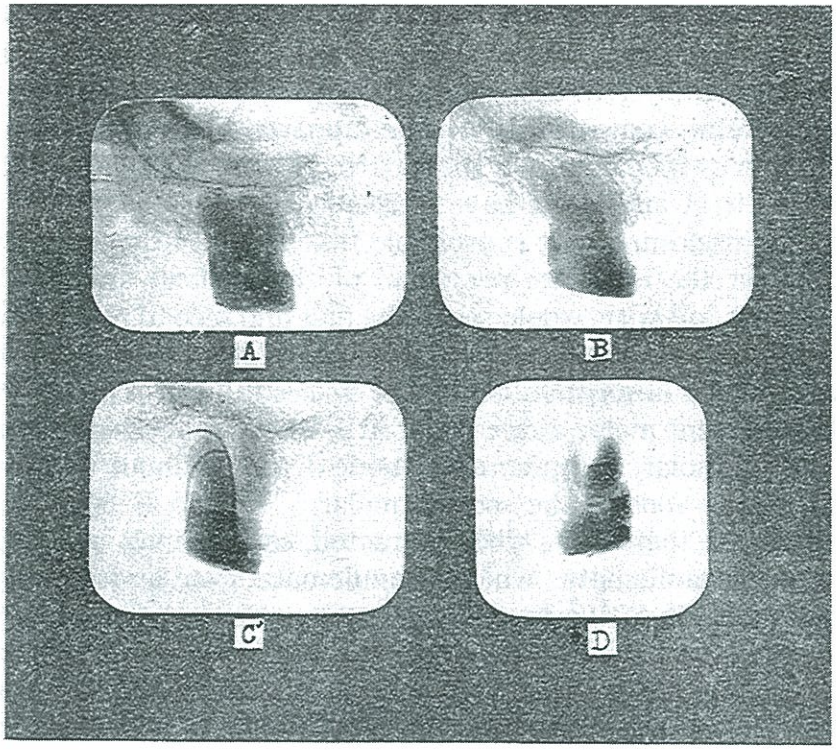


FIGURE 5. ROENTGENOGRAPHIC STUDIES OF PERIODONTOCLASIA: A AND B, DIFFERENT ANGLES; C, WITH FLEXIBLE GUTTA-PERCHA POINTS; D, EXTRACTED TOOTH.

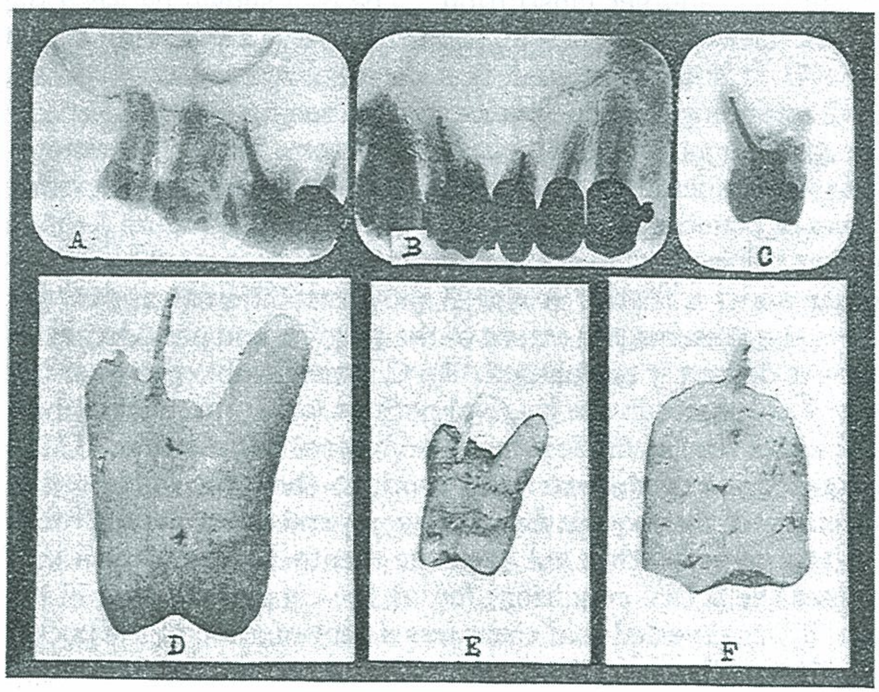


FIGURE 6. COMPARISON OF ROENTGENOGRAPHIC VIEW OF TEETH WITH THE PHOTOGRAPHS OF SAME WHEN EXTRACTED.

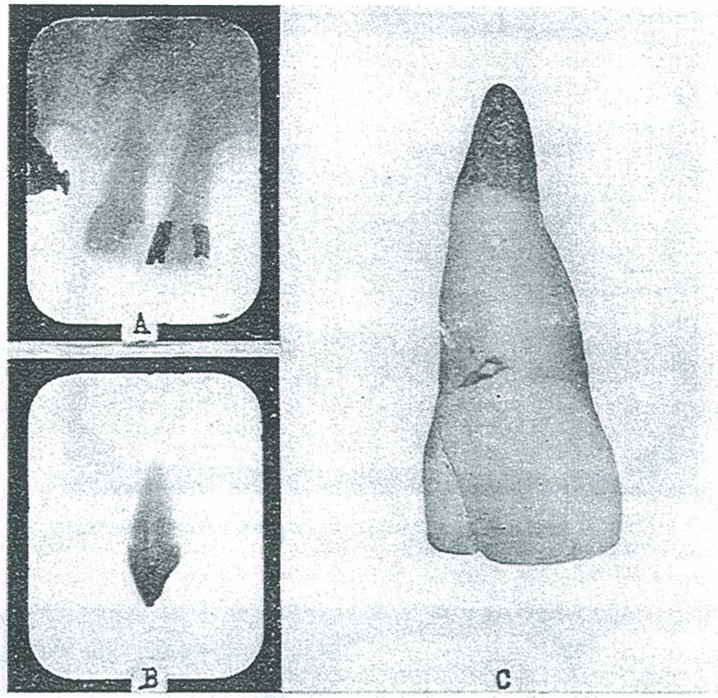


FIGURE 7. COMPARISON OF ROENTGENOGRAPHIC AND PHOTOGRAPHIC VIEWS OF AN INCISOR.

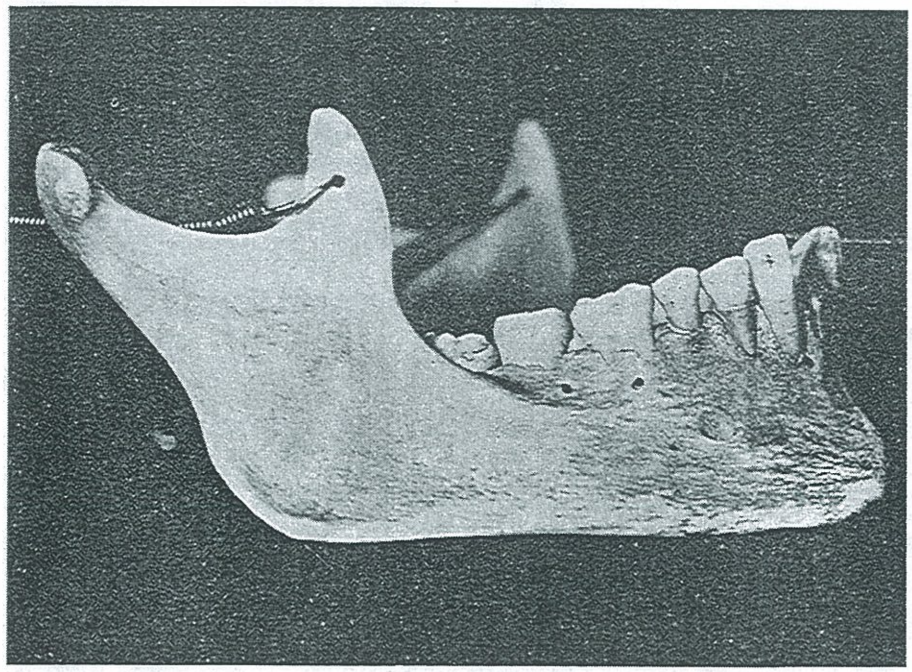


FIGURE 8. PHOTOGRAPHIC VIEW OF EXTERNAL OBLIQUE RIDGE OVER SECOND AND THIRD MOLARS, WHICH CAST THE SHADOW SHOWN IN FIGURE 9.

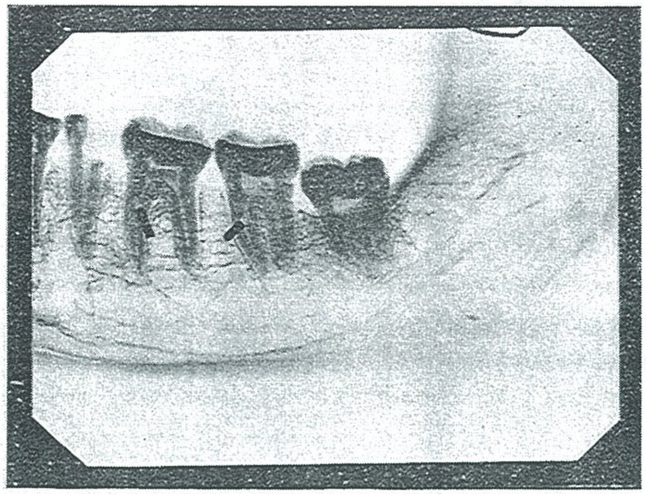


FIGURE 9 SHOWS A RADIOPAQUE AREA OVER ROOTS OF SECOND AND THIRD MOLARS. (SEE FIGURE 8.)

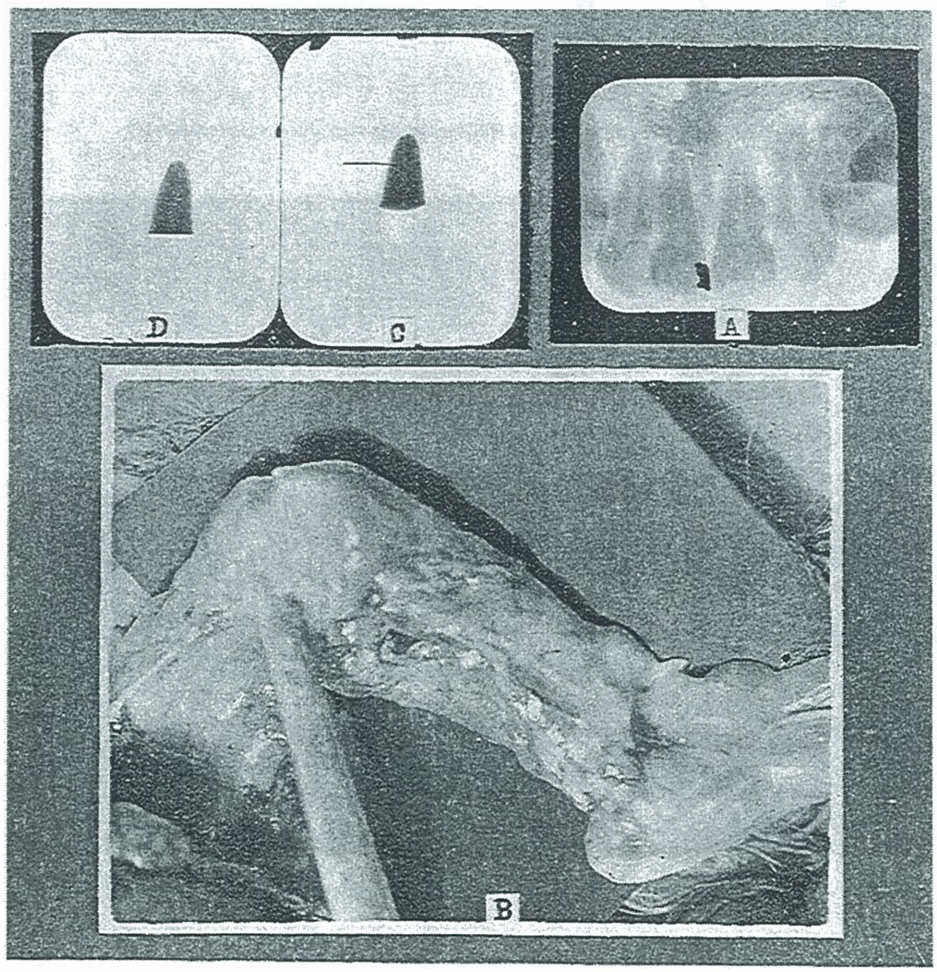


FIGURE 10. A ROENTGENOGRAPHICALLY INNOCENT TOOTH, BUT ACTUALLY A VERY DANGEROUS ONE.

presumption that the root filling is more opaque than the tooth structure, and being within the tooth structure, its true position and extent can be revealed by the Roentgen-ray. We have made many studies to determine this. A typical illustration will be seen in Figure 6, in which A and B show two different angles of the molars and bicuspids in a case under study. You will note that there is little evidence of the actual condition revealed in this case. There was an extensive cyst over the bicuspid and molar, only slightly outlined in the roentgenogram. Its presence and nature were established by microscopic examination of aspirated material. D and E show different photographic views of the molar from which both buccal roots had been absorbed, and one of the root fillings is seen projecting and exhibits a condition which is not revealed by the Roentgen-ray. F shows a photographic view of the second bicuspid from which the apical third was absorbed and its root filling extending considerable distance beyond the tooth; and yet, in the roentgenograms there is no evidence of this condition. It would appear in both A and B that the root filling is short of the apex. This case is referred to again in the chapter on Dental Cysts.

Figure 7 shows in A, roentgenograms of two central incisors, neither of which was tender to percussion or had given evidence of sensitiveness, one of which did not respond to temperature change when a search was made for a cause for rheumatism. It had a putrescent pulp and a very serious periapical involvement, without evidence of same in the roentgenogram. C shows the apical third of this root from which the pericementum had been destroyed, apparently for years, so that it was greatly discolored; and yet, the lamina dura and bone adjacent to the alveolus were found to be in almost normal position though not in normal condition, notwithstanding it is not a condition that is revealed by the Roentgen-ray. This is discussed in the next chapter.

Figures 8 and 9 show a photograph and roentgenogram of a mandible to illustrate the influence of the external oblique ridge in superimposing a dense bone, (varying greatly in different individuals), over the apices of molar roots, which modifies in large measure the roentgenogram as shown but only in part accounts for the condition as seen in Figure 3. While Figures 8 and 9 illustrate the effect of an interposing dense bone, our studies show that frequently the interposing hard substance may be the

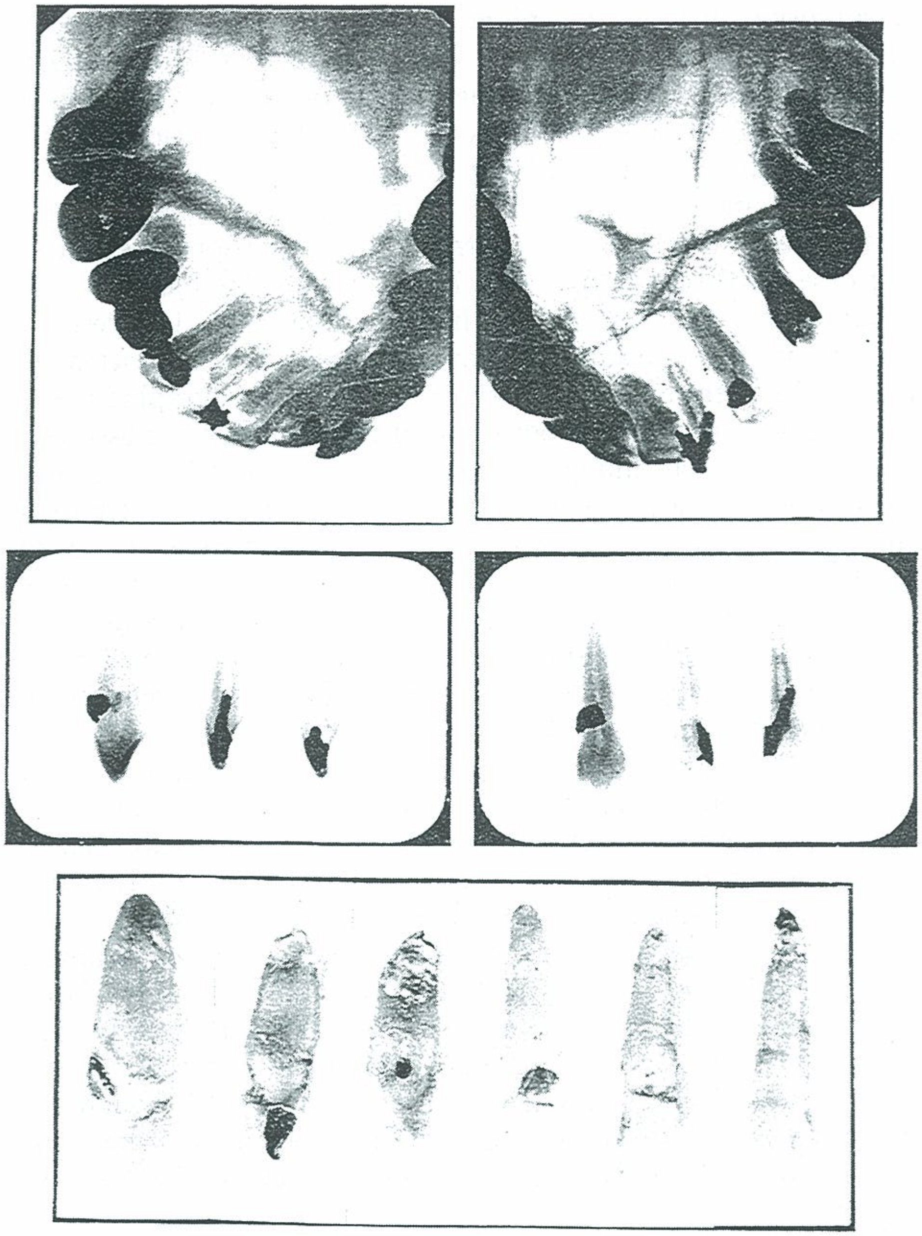


FIGURE 11. A COMPARISON OF THE ROENTGENOGRAPHIC AND PHOTOGRAPHIC STUDIES OF TEETH. NOTE THE ABSENCE OF EVIDENCE OF ACRETIONS IN THE ROENTGENOGRAMS OF THE TEETH *in situ* ABOVE, AND AFTER EXTRACTION IN THE CENTER, WHICH ARE VERY CLEARLY REVEALED IN THE PHOTOGRAPHS OF THE SAME AFTER EXTRACTION, SHOWN BELOW.

tooth itself, or another root of the same tooth, as illustrated in Figure 10, in which the roentgenogram of the central incisors is shown. There is exceedingly little difference in the condition disclosed about the apices of these two centrals; yet one is vital and the other non-vital, as disclosed in the search for a cause for a very acute and severe attack of rheumatism which had been recurring with increasing frequency and severity; and at this time, the patient could scarcely walk and was compelled to shuffle the feet along with almost no use of the feet and ankles. Thermal and electrical tests revealed the fact that the pulp was apparently non-vital in the central incisors showing the small filling. The color of both teeth was practically identical. The patient protested against losing the tooth. The reason for extraction and not root filling will develop in later chapters. The proof of local infection, secured for diagnosis, was established by aspirating from over the root and comparing the blood count with the patient's general circulation, which disclosed a higher leucocytic invasion than normal. The extraction of the tooth revealed a lateral canal with a small area of absorption at about the junction of the middle and apical thirds, which, being situated on the external surface of the root, was in line with the tooth and therefore not disclosed by the Roentgen-rays. The opaque tooth hid the area of absorption which was very slight. This is shown in C with a metal point placed in the lateral foramen. B of this figure shows a knee and ankle very profusely bathed in pus coming from the synovial sacs of the knee and ankle joints of a rabbit which was inoculated with the culture from this supposedly normal and harmless tooth.

It is quite remarkable how little difference the Roentgen-rays will reveal in the structure of a vital and non-vital tooth. Nor do we look for a change in the tooth structure as a rule. The change is expected to be in the supporting alveolar bone. Many teeth, however, carry depositions and incrustations, or zones of absorption, without any evidence of the same in the roentgenogram. An illustration of this will be found in Figure 11 which shows different views of a cuspid and two laterals by each method, roentgenographing and photographing, the latter both before and after extraction. The corresponding photographic views of the extracted teeth are shown in both photographs and roentgenograms. There is little suggestion in the roentgenograms of the extensive structural changes which are disclosed in the photographs.

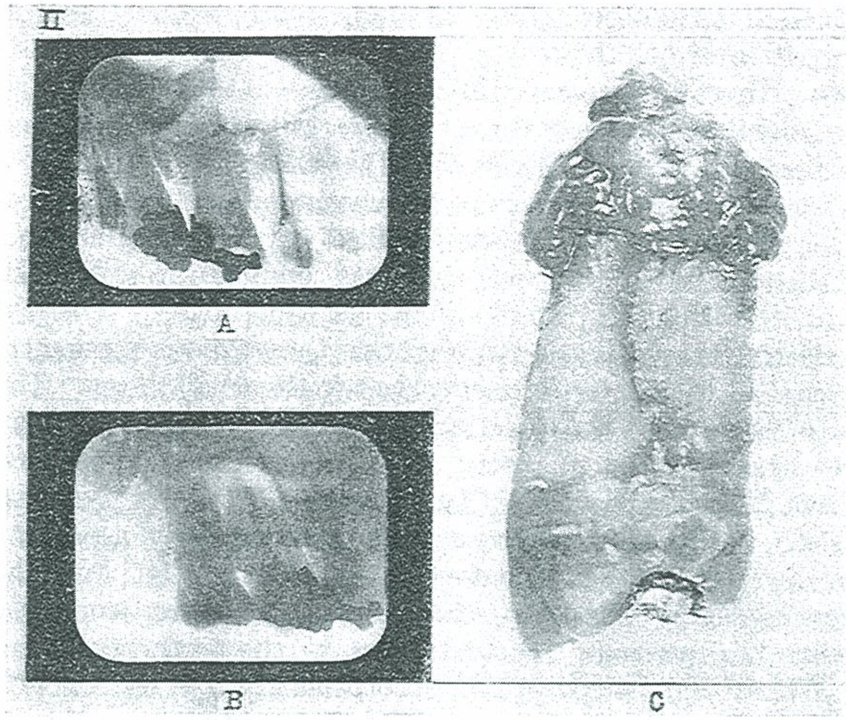


FIGURE 12. A, A HIGH ANGLE VIEW OF THE UPPER MOLARS. B, A LOW ANGLE. C, THE APPEARANCE OF THE EXTRACTED TOOTH.

One of the most common defects met with is the securing of roentgenograms of the upper molars because of the anatomical complications. The hard palate is often too low to permit the film to be placed laterally to the tooth, thereby requiring it to be placed at an angle if the apices of the roots are to be secured. While this condition can be partly overcome by retaining the lateral parallel position, there is a distinct limitation which requires the angle of incidence of the rays to be raised, which condition brings the malar bone into the field. This frequently casts a shadow of opacity, partially or entirely masking the details of the bony structure about the apices. Such a case is shown in Figure 12. In this instance A shows a roentgenogram brought by the patient from an excellent radiographer who, working with a group who had been looking for a cause for a neuritis in this patient, advised her that there was no contributing involvement of the teeth. B shows a different angle of these molars, disclosing a distinct area of radiolucency about the apices of the second molar. C shows an enlarged view of this tooth with its very extensive granulomatous mass, which evidences the possibility of

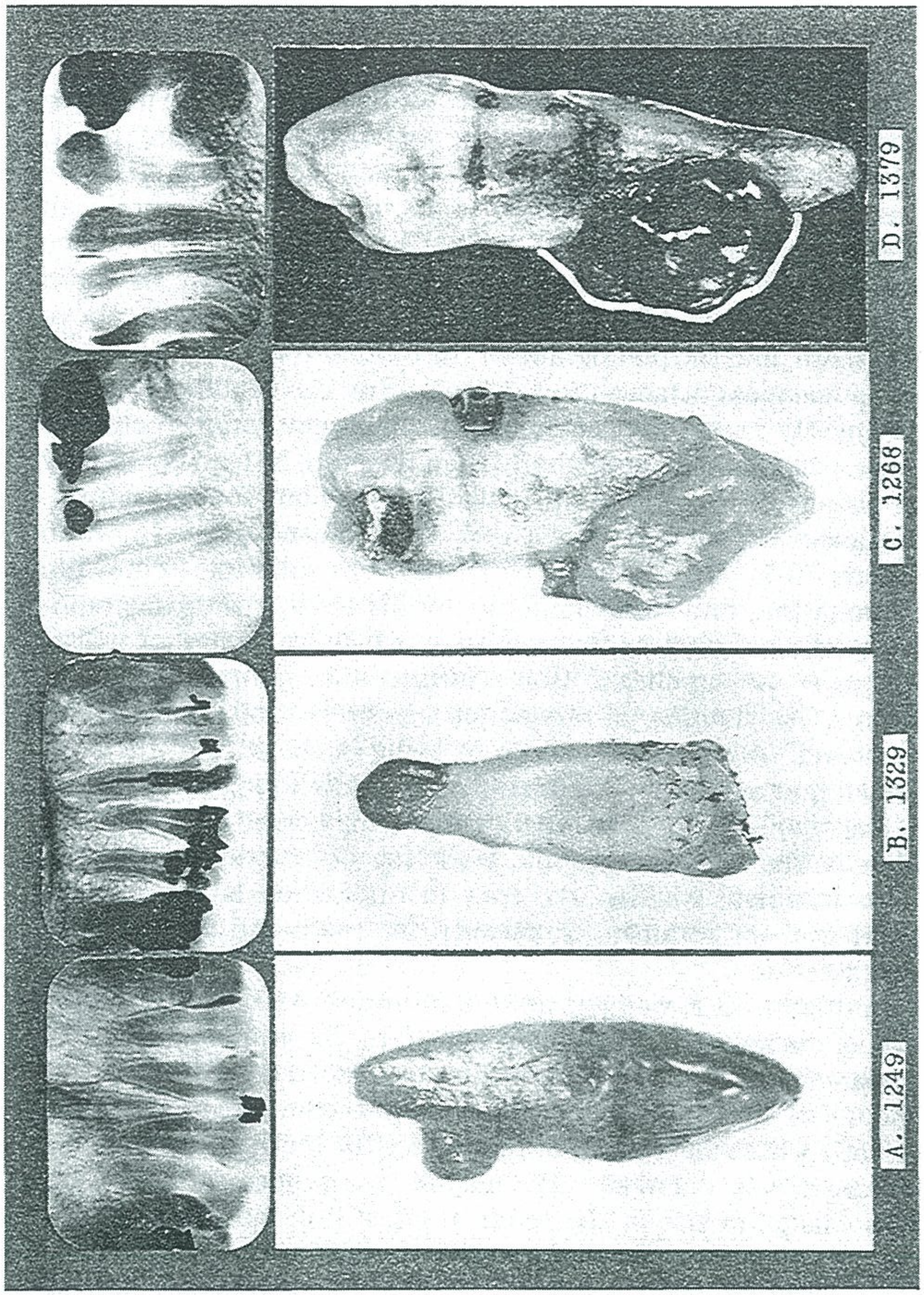


FIGURE 13. COMPARISON OF THE ROENTGENOGRAPHIC AND PHOTOGRAPHIC APPEARANCES OF EXTRACTED TEETH.

there being an extensive involvement of periapical tissue without its being disclosed by the Roentgen-ray.

It is not an uncommon condition for the largest of the multiple foramina of the tooth to be on the side of the root at any point, often about half way between the apex and the gingiva. In these cases there may be an extensive zone of rarefaction laterally to the root, which, because of the physical conditions, cannot be disclosed by the Roentgen-ray. Two of such cases are shown in Figure 13 in A and D. In D, the roentgenogram is shown above the tooth, and the photograph of the tooth shows the large granulomatous mass attached to the root. But this condition is more frequently found on incisors; and if the lateral foramen chances to be either labially or lingually from the pulp, instead of mesially or distally, neither the foramen nor the granulomatous mass forming about its exit will be revealed. Such a condition is shown in Figure 13-A. In this case the patient was suffering from acute rheumatism, and the examination of his teeth, roentgenographically, did not disclose this condition, which we have shown elsewhere to be significant from cultural and animal inoculation tests. The chamber of rarefaction was present but could not be disclosed, owing to its position, it being in the path of the root, which prevented the Roentgen-rays from disclosing it. Much as a dog would not be seen when it was lying behind a tree, except that in the roentgenographic work the obstruction of the tree is as complete whether the zone of rarefaction is between the tooth and the foramen, or between the tooth and the source of Roentgen-ray.

In Figure 13-B we have an illustration of the difference in the size of the area of rarefaction occupied by the granuloma, and its apparent size in the roentgenogram. I do not mean that this picture of the granuloma corresponds with the picture as shown in the roentgenogram, for in the former the tooth and granuloma are purposely enlarged. By making the proper correction for this change in size of the tooth, these still do not have the expected evidence in the roentgenogram. This is the more striking since the granuloma, as shown, is in relatively correct size, having shrunken considerably, due to exposure to the air, between the time of its extraction and its being photographed. This condition is a very common one which we will discuss later in the relationships between condensing osteitis and rarefying osteitis.

In Figure 13-C we have a condition somewhat similar to that in D; and while it is from a case of extensive periodontoclasia, its structural relationship to the tooth to which it is so closely adherent with a distinct separation from the surrounding connective tissues, except through the peridental membrane, only a small portion of which has remained vital, strongly suggests that this granulomatous mass is directly related to the tooth and products coming from it.

In Figure 14, I have undertaken to show a group of such conditions, with the teeth shown approximately normal size for comparison with the roentgenograms; and it will be noted that there is very little evidence of the existence of the extensive periapical absorptions such as are necessary to accommodate the large granulomata present. Indeed, it will in many of them be difficult without experience to determine which tooth in the roentgenogram is the one involved. In A, it is the first biscuspid; in B, it is the first molar; in C, it is the lateral; in D, it is the third molar; and in E, it is the second molar.

Since the roentgenogram is only a shadow, the angles of incidence of the rays to both the tooth and the photographic plate receiving the impression, have all significance. We are all familiar with the increasing length of our statures as we walk away from the sun in the evening, or the shortening of our shadow toward noonday; and, indeed, the length of this shadow has constituted one of the most important means for reckoning time, since the height of the object causing the sun's shadow was known, or could be determined. But in this field we find ourselves reversing the process and undertaking to determine the length of the object causing the shadow, all of which can, of course, be accurately done only when we know these angles of incidence. This is illustrated in Figure 15, in which I have shown three photographic views of the same tooth, which is an upper biscuspid and has a protruding root filling. In the first view from the left, the relation of the root filling to the apex is shown laterally, which is the mesiodistal position. When the same tooth is viewed from the buccal position, some of the root filling is covered because of the fact that it does not leave the root at the most dependent point; and since it is between the extension of the root and the source of ray, the shadow of part of the exposed root filling is obstructed by the lingual aspect of this root. But this view is taken at nearly right angles to the long axis of the root, a position that is impossible in this patient's mouth because of the low palate; and when the tube is raised sufficiently to have the shadow of the tooth fall upon a film that could be placed in the mouth, so much of the

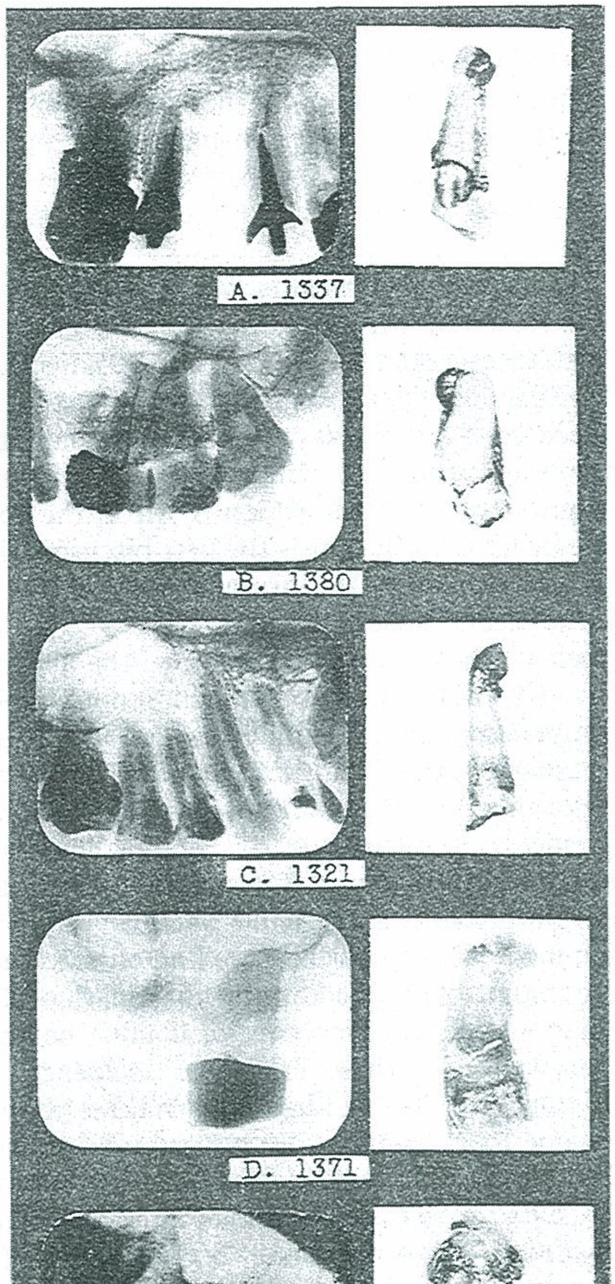


FIGURE 14. COMPARISON OF THE ROENTGENOGRAPHIC AND PHOTOGRAPHIC APPEARANCES OF EXTRACTED TEETH.

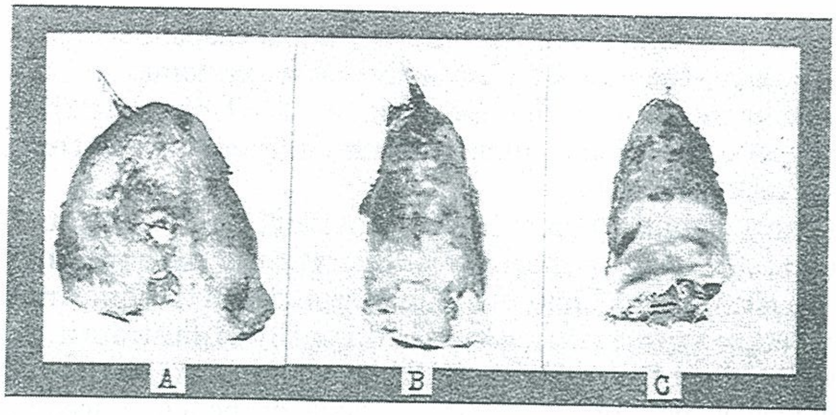


FIGURE 15. DIFFERENT VIEWS OF THE BICUSPID ROOT SHOWN IN FIGURE 6. A, SIDE VIEW, RIGHT ANGLE; B, LATERAL VIEW, RIGHT ANGLE; C, LATERAL VIEW, FORTY-FIVE DEGREES.

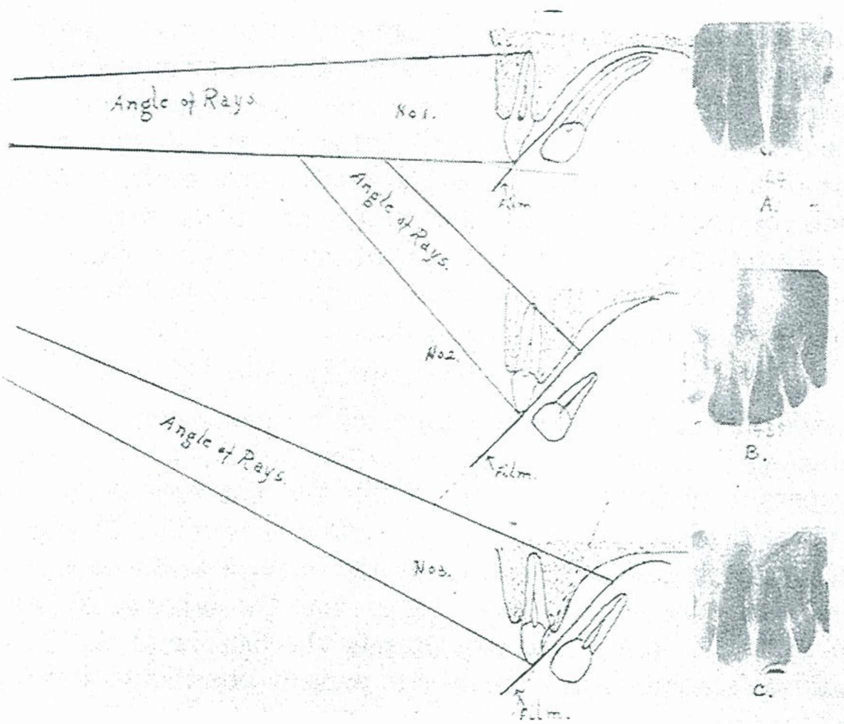


FIGURE 16. RELATION OF ANGLE OF INCIDENCE OF RAYS TO TOOTH AND FILM PLANES: NO. 1 AT RIGHT ANGLE TO PLANE OF TOOTH; NO. 2 AT RIGHT ANGLE TO PLANE OF FILM; NO. 3 AT RIGHT ANGLE TO A PLANE HALF WAY BETWEEN PLANE OF FILM AND PLANE OF TOOTH, THE CORRECT POSITION TO MAKE TOOTH APPEAR THE PROPER LENGTH.

exposed root filling is in line with the lingual aspect of this root, that it gives the appearance of only a short perforation. This is shown in the view to the right, Figure 15. Unfortunately, this is not an uncommon or unusual condition, for it is one that obtains very frequently.

I have published, previously,[1] an article on the relationship between the angle of incidence of the rays and the apparent size of the shadow. In Figure 16, I have reproduced this drawing and the roentgenograms that were used originally to illustrate it. In A it will be seen that a low angle of the ray at right angles to the long axis of the tooth, but with the plane of the film placed at an oblique angle to the plane of the axis of the tooth, makes an elongated shadow of the root of the tooth, illustrated in the roentgenogram also taken at this angle. B shows the same case photographed at an angle high enough to be at right angles to the plane of the film, in which case the tooth is foreshortened, as shown in the roentgenogram of the same case taken at this angle. C shows the correct position for obtaining the shadow of the tooth of the same total length as the length of the tooth, to obtain which the rays must fall at right angles to a plane which is half way between the plane of the tooth and root, and the plane of the film, as shown. C shows the roentgenographic appearance of the same case as A and B when so rayed.

SUMMARY AND CONCLUSIONS

From these few illustrations selected to demonstrate different conditions, it is apparent that the problem is not so simple as it is generally understood to be. While the analyses of the new problems disclosed by this study are made in detail in subsequent chapters, in general, it is demonstrated in these and a very large number of cases not included here, that the original premises a, b, and c, as stated, do not present the facts; and that these should be stated in the light of our present knowledge about as follows:

> **(a) That Roentgen-rays will not necessarily reveal the presence of infection, either in soft tissue or in hard; (b) That the apparent extent of the absorption is not necessarily the actual extent of the absorption; and hence, even assuming that the extent of the infection is the extent of the absorption, is not the extent of the infection; and (c) That an area of absorption may be present and not be disclosed by the roentgenogram, nor are conditions and relations necessarily as they appear to be.**

[1] See bibliography.

CHAPTER II.

THE NATURE OF THE ORGANISMS INVOLVED.

PROBLEM: Is the danger from a dental infection primarily dependent upon the invasive quality of the organisms involved; or, otherwise stated, is it true that dental infections, when they occur at the apices of roots, are produced by the invasion of that area by organisms from the mouth which enter that tissue through the open pulp canal; and that the question of danger from such an infection is dependent upon the invasive qualities of that organism?

EXPERIMENTAL AND DISCUSSION

While many workers, besides ourselves, in the field of the bacteriology of dental infections, have called attention to the fact that streptococci are usually present in root end infections, it is generally understood by the members of the dental and medical professions that, whatever the classification of the organism, the injury it will produce has to do with the particular kind or strain of organism involved. Those most familiar with the literature are familiar with the fact that the organism involved is now generally understood to be a streptococcus.

The research herewith reported, which has covered many years, has centered about the following phases of the problem:

(a) What are the morphological characteristics of the organism?

(b) What are the biological characteristics of the organism?

(c) What is the relationship between the morphological and biological characteristics and their local and systemic tissue expressions?

(d) To what extent are the organisms influenced by variations in the pabulum, or culture medium, in which they grow?

(e) Are the organisms capable of producing specific toxic substances and, if so, under what conditions?

(A) WHAT ARE THE MORPHOLOGICAL CHARACTERISTICS OF THE ORGANISMS?

To determine these factors, we have made a study of the organisms secured from the various dental tissues involved, and have compared them with the strains recovered after their injection into various types of experimental animals. We have found, in general, that in approximately 98 per cent of instances, the dominating organism present appears as a coccus, growing in diploic and chain form, chiefly short chain; and that where the organism is taken from dental lesions, and inoculated into experimental animals and recovered from their tissues, in one series taken from forty different sources and inoculated into about one hundred animals, the organisms as recovered from two hundred lesions in those animals had the same morphology except that there was an increased tendency to grow in short chains and in diploic form until regrown in artificial media. In the histological sections of the tissues from these lesions, the organisms were seen almost entirely in diploic form. In later chapters I have discussed the teeth as carriers of infections for contagious and infective fevers.

In a large number of cases the culture injected, as grown from the original focus, contained in addition to this coccus other bacterial forms, chiefly a short bacillus and staphylococcus, also in spiral forms. In a few instances we recovered from tissues by cultural methods the staphylococcus as well as the diplococcus, and in less than 2 per cent staphylococcus only. As we now view this work, we do not feel sure that the diplococcus was not present in those cases where we did not identify it, having come to recognize that it is quite exacting in some instances regarding oxygen tension and hydrogen ion concentration; and where both organisms grew out, the diplococcus may have been overgrown. We do not question the presence of the staphylococcus, but we are not certain of the absence of the diplococcus. It is significant that animal passage generally destroyed all organisms except the diplococcus. Our recent methods of culturing are showing the presence of streptococci or diplo-steptococci as one or the only organism in infection of the tooth structure.

(B) What are the biological qualities of the organisms involved?

These have been determined by their reactions on culture media and animal tissues. We have found many varieties of the streptococcus. Figure 17 is made from sixty-seven successive cases and shows that many types of the streptococcus may be found in dental lesions, chiefly the following: fæcalis, ignavius, salivarius, infrequens, mitis, non-hæmolyticus I, non-hæmolyticus III, hæmolyticus I, subacidus, and pyogenes. The column marked "Per cent" shows the percentage of these various varieties occurring in the total number included in the study. The ratio or percentage of these to each other is expressed graphically by the solid lines to the left under "Graphic Expressions." This shows strikingly the very large percentage of fæcalis.

RELATIVE PREVALENCE OF DIFFERENT STRAINS

*Type of Streptococcus	%	Graphic Expressions
Fecalis	65½	
Ignavius	1½	
Salivarius	1½	
Infrequens	9	
Mitis	7½	
Non-Hemolyticus I	3	
Non-Hemolyticus III	3	
Hemolyticus I	3	
Subacidus	1½	
Pyogenes	4½	

* (Note—Streptococcus viridans is the group name of non-hemolyzing streptococci producing a narrow green zone when grown on blood agar)

FIGURE 17.

BACTERIAL CLASSIFICATION IN RELATION TO TISSUE AFFECTED.

Type of Lesion in Patient	S. Ignavius	S. Salivarius	S. Infrequens	S. Mitis	S. Non-Hemolyticus I	S. Non-Hemolyticus III	S. Hemolyticus I	S. Subacidus	S. Pyogenes	S. Fecalis	Percentage S. Fecalis	Ratio of Chance
Rheumatism			2		1	1				**7**	11	**7.1**
Heart										**2**	3	**1.3**
Nerves		1	3	4	2	1	2		3	**21**	33	**24.05**
Lassitude			2	1						**11**	17	**9.1**
Internal Organs		1	1	1						**8**	13	**7.1**
Special Tissues	1		3	1		3				**7**	11	**9 7**
No Lesions								1		**7**	11	**5.2**

FIGURE 18.

We have failed completely to establish a relationship between the various strains of streptococci and particular types of tissue lesions in the patient, and the results of animal inoculations have been similar, in that the particular types or strains of streptococci that were introduced, did not of necessity produce a particular type of lesion in animals, although various strains were found to reproduce in animals particular lesions possessed by the patient from whom they were taken. There was also no constant relationship between the biological properties, as expressed in sugar fermentations and the expressions in animal tissues.

(c) What is the relationship between the morphological and biological characteristics and their local and systemic tissue expressions?

If, as has been so generally supposed, the particular type or strain of invading organism determined the tissue reaction and elective localization, we should expect that a careful study of the biological properties, such as sugar fermentation, would have a direct relationship to the type of tissue involved in a particular individual. To determine this we have made a careful study of the percentage of instances in which the various strains of the streptococcus were present, and have noted the particular type of tissue that was involved in that patient's body. The result of this study is strikingly shown in the chart in Figure No. 18. It will be seen, not only that the localization in the various structures is not limited to a particular type, but, on the contrary, the various tissues have been invaded indiscriminately by the different varieties of streptococcus. For example, the nerve tissues were invaded by all but two of the ten principal varieties of streptococci.

By expressing this relationship in percentage, we have an opportunity to compare the appearance of each of these varieties with its appearance on the basis of chance, assuming there was no localizing quality characteristic of each variety. This is shown for S. Fæcalis under the column, the second last to the right, entitled "Percentage S. Fæcalis." For this study a group of one hundred localizations was used; and hence, for any one tissue the total number of appearances of a particular variety, say Fæcalis, in proportion to the total number of appearances will give us the percentage. For example, in rheumatism there were 7 instances of Fæcalis out of a total of 63 appearances of Fæcalis. The percentage of appearances as rheumatism of Fæcalis was 11 per cent. The ratio of chance, were there no elective localization, would give on this basis 7.1; whereas, the actual appearance, as shown, was 7. For heart, the ratio of chance will give Fæcalis 1.3; the actual appearance was 2; for nerves, 24.05 and 21; for lassitude, 9.1 and 11; internal organs 7.1 and 8; special tissues 9.7 and 7; no lesions, 5.2 and 7. It will therefore be seen, that there is so close an adherence of the actual appearance to that which should be expected on the basis of chance, that we must conclude that the elective localization qualities are not dependent upon inherent biologic properties of the variety of the streptococcus involved. This is a matter of exceeding great importance since it shifts the burden of fundamental responsibility from the bacterium to the host, as will be clearly demonstrated later.

(D) To what extent are the organisms influenced by variations in the pabulum, or culture medium, in which they grow?

The next problem—namely, to what extent are the organisms influenced by the nature of the pabulum or culture medium in which they grow—must throw direct light upon this quality of accommodation of the organism. To determine this, several series of studies were made to ascertain the ability of streptococci to adapt themselves to their environment. These are shown graphically in Figures 19, 20, 21 and 22. For this study, strains were planted in a culture medium in which they were known to

FORMALIN

	1-160	1-320	1-640	1-1280	1-2560	1-5120	1-10,240	1-20,480	1-40,960	1-81,920	1-163,840	1-327,680
1-3	—	—	—	—	—	—	1+	4+	4+	4+	4+	4+
2-5	—	—	—	—	—	3+	4+	4+	4+	4+	4+	4+
2-6	—	—	—	—	—	4+	4+	4+	4+	4+	4+	4+
2-7	—	—	—	—	4+	4+	4+	4+	4+	4+	4+	4+
2-8	—	—	—	—	4+	4+	4+	+	+	+	+	+
2-9	—	—	—	—	4+	4+	4+	+	+	+	+	+
2-10	—	—	—	4+	4+	4+	4+	+	+	+	+	+
2-11	—	—	—	4+	+	+	+	+	+	+	+	+
2-13	—	—	2+	4+	+	+	+	+	+	+	+	+
2-17	—	—	4+	+	+	+	+	+	+	+	+	+
2-20	—	—	+	+	+	+	+	+	+	+	+	+
2-24	—	—	+	+	+	+	+	+	+	+	+	+
2-28	—	—	+	+	+	+	+	+	+	+	+	+
3-22	—	+	+	+	+	+	+					

+ = weak growth.
4+ = full growth.
— = negative growth.

FIGURE 19.

grow well, and to this medium, toxic substances were added to determine the point at which the toxicity would prevent the growth of the organisms. It was found that by taking the dilution just greater than would entirely inhibit the growth of the organism and maintaining the organisms in that concentration, they presently acquired a quality of growing quite readily in the presence of that poison; and by replanting, it was found that they would soon permit a new increase without seriously handicapping them. In this way, by slowly but continually increasing the concentration of the poison, it was found possible to grow streptococci taken from dental infections in one to three hundred twenty of formalin; whereas growth of this strain was inhibited by one to five thousand in its original strength. Similarly, it was found that these varieties would accommodate themselves to almost any irritant, even growing luxuriantly in twenty per cent alcohol (See Figure 21), one to ten iodoform saturated in alcohol (Figure 20), one to eight hundred phenol (Figure 22), one to two hundred thalium sulphate (Figure 22).

This has a very wide bearing upon our whole problem and suggests the answers to many questions. For example, we have often asked why it is that teeth that had been root-filled with iodoform, and in which the odor of iodoform was still persistent after years of time, would show an abundant growth of streptococci in the midst of the root dressing highly fragrant with iodoform. The organisms had very clearly accommodated themselves; for in our tests of feeding this chemical to streptococci, we found that they would ultimately grow in saturated aqueous solutions of iodoform.

Under another chapter we will discuss the reasons for the efficiency of iodoform in the treatment of wounds.

In our studies of the characteristics of the organisms growing in infected teeth, extending over several years, we have found that with an increased knowledge of the nature of the organisms and their power of accommodation and adaptation to their environment, they come to take on quite relatively stable, or well defined, characteristics, with regard to the kind of culture medium in which they will grow. With our increasing knowledge we are finding that the data accumulating show a quite definite per cent of teeth which are proven by positive culture to be infected, which means that whereas all positive cultures, of course, are positive, the absence of growth is not necessarily an evidence of the absence of an infection in the material

IODOFORM SATURATED IN ALCOHOL

	1-5	1-10	1-20	1-40	1-80	1-160	1-320	1-640	1-1280	1-2560	1-5120	1-10,240	1-20,480	1-40,960	1-81,920	1-163,840	1-327,680
2-28	—	—	—	—	+	+	+	+	+	+	+	+	+	+	+	+	+
3-22	—	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

FIGURE 20.

ALCOHOL

	1-10	1-20	1-40	1-80	1-100	1-200	1-400	1-800	1-1600	1-3200	1-6400	1-12,800	1-25,600	1-51,200
48 hrs. 2-11 to 13	—	—	—	—	—	—	1+	4+	4+	4+	4+	4+	4+	4+
	—	—	—	—	—	—	4+	4+	4+	4+	4+	4+	4+	4+
2-24	—	—	—	—	—	4+	4+	+	+	+	+	+	+	+
2-28	—	—	—	—	4+	4+	4+	4+	4+					
3-1	—	—	+	+										
3-22	—	+	+	+										

— = negative growth
\+ = weak growth
4+ = full growth

FIGURE 21.

PHENOL

	1-100	1-200	1-400	1-800	1-1600	1-3200	1-6400	1-12,800	1-25,600	1-51,200	1-102,400	1-204,800	1-409,600	1-819,200
2-1 48 hrs.	—	—	—	—	—	—	—	—	—	+	+	+	+	+
2-15	—	—	—	—	—	—	+	+	+	+	+	+	+	+
2-17	—	—	—	—	+	+	+	+	+	+	+	+	+	+
2-24	—	—	—	2+	4+	4+	4+	4+						
2-27	—	—	—	4+	4+	4+	4+	4+						
THALIUM SULPHATE														
2-17	—	—	—	—	—	+	+	+	+	+	+	+	+	+
2-24	—	—	—	+	+	+	+	+						
2-27	—	—	—	+	+	+	+	+	+	+	+	+	+	+
3-22	—	+	+	+	+	+	+	+	+	+	+	+	+	+

— = negative growth.
\+ = weak growth.
4+ = full growth.

FIGURE 22.

being cultured. When organisms grow in a root filled tooth for a long period of time, they tend to become anaerobic, and will frequently not start to grow in aerobic culture in less than from twenty to thirty days but will grow promptly under anaerobic or partial tension conditions. This has a very great bearing upon the procedures that have been advocated for the testing of teeth to determine when they are ready for root filling. Figure 23 illustrates this condition. Two tubes are shown which were inoculated at the same time from the old root filling material of a suspected tooth. The culture made aerobically remained sterile, whereas the culture made anaerobically grew out profusely in 48 hours, showing a good growth in 24 hours. In the illustration, the two tubes are supported in front of a white background, through the center of which passes a black strip. The clear transparent media of the aerobic culture shows the black of the strip through with nearly the same clearness that is seen on each side of the culture media, whereas the tube with the anaerobic culture, or rather partial tension in this case, for the organisms are growing in a medium covered with oil, which medium did not have its oxygen removed from it before inoculation, is sufficiently toxic to hide quite completely from view the black strip behind the tube. In subsequent chapters I have discussed the significance and importance of this in its various phases. When, however, these anaerobes are grown in a constantly increasing oxygen tension, they soon come to be aerobes, again illustrating this most important quality: namely, their capacity for accommodation to their environment.

(E) ARE THE ORGANISMS CAPABLE OF PRODUCING SPECIFIC TOXIC SUBSTANCES?

There has been practically nothing in the dental literature to throw light upon the complicated problem of the relative importance of bacterial invasion and the passage of the toxins produced by the organisms, to the tissues of the host. To determine this we have inoculated large numbers of animals with washed organisms and bacterial filtrates, using cultures from infected teeth of involved cases. The clinical symptoms will be discussed in succeeding chapters under special headings.

A typical illustration of the effect of the organisms upon the filtrate of the culture media in some cases, for all strains do not produce comparable toxins, is the following: A rabbit which was inoculated with 1 cc. of the filtrate from a culture grown

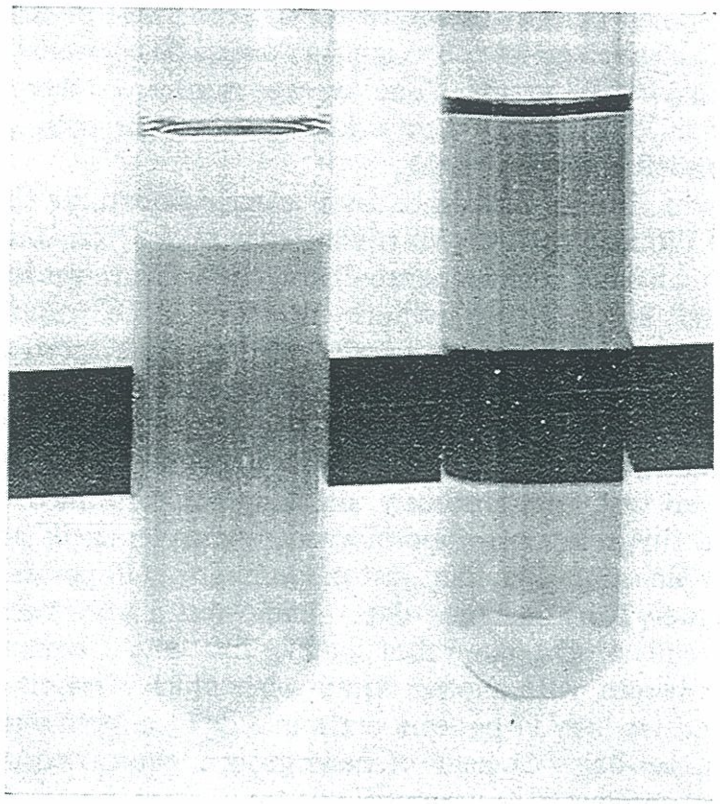


FIGURE 23. A COMPARISON OF THE AEROBIC AND ANAEROBIC GROWTHS OF INOCULATIONS WITH A STRAIN TAKEN FROM THE INTERIOR OF AN INFECTED TOOTH. AEROBIC, TO RIGHT, COMPLETELY TRANSPARENT, NO GROWTH; ANAEROBIC, TO LEFT, HEAVY GROWTH HIDING BLACK STRIP BEHIND TEST-TUBE.

from an infected tooth, died in sixteen days, having lost 35 per cent of its weight. On postmortem, it showed acute myocarditis, acute passive hyperemia of the liver and kidneys, and acute atrophy of muscles. The anatomical diagnosis for the cause of death was recorded as toxemia. We will review many instances later of animals dying from the effects of the toxin present in infected teeth, which teeth have been crushed and washed, and the washings filtered, the clear filtrate being inoculated into the animals, and producing quite as striking results as the above.

Similarly, the inoculation of animals with the washed organisms has demonstrated that very small quantities of these germs will be adequate in the case of many strains, to produce very serious structural changes with or without termination in death. Such a condition is illustrated in the following: Of 8 rabbits so inoculated, 2 died within 24 hours, 3 others within 9 days, and the other 3 within 13 days. The average loss of weight was 2.6 per cent per animal per day, as expressed in percentage of the total weight. The average loss of weight per animal before death was 18 per cent, or 203 grams. The average number of days lived was 7. Four of the 8 had a total loss of over 28 per cent, or an average of 319 grams. (*See Fig. 24.*)

There is a distinct difference, as is clearly shown, in the behavior of different strains which we have taken from infected teeth and cultured, then separated from the culture media and washed and suspended in sodium chloride. Of 15 rabbits so studied, none of which were included in the former group of 8, 4 gained in weight, an average of 336 grams, or an average of 26 per cent, nearly 1 per cent per day. The former group of 8 all died spontaneously as a result of their injections. None of this latter group died spontaneously, all being chloroformed. The other 11 of this group were chloroformed on an average of 10 days each, at which time they had lost on an average 10 per cent, or approximately 1 per cent per day. There was a wide variation in the amount of loss, one rabbit having lost only 1 per cent in 13 days, whereas, with another strain with which 3 rabbits were inoculated, they lost 17 per cent, each in 4 days, or an average of 4 per cent per day. In each of these groups, typical structural lesions were produced in various tissues and organs.

When we compare these effects of both the filtrates and the washed organisms on similar dosages of organisms plus filtrates, or rather the organisms in the culture medium in which they have grown without either filtering or washing, we have found that ½ to 1 cc. doses, which were usually given, produced quite different results in accordance with the particular strain being used. For example, in a group of 19 rabbits which had been so inoculated, and all of which were chloroformed, 7 proceeded to gain in weight, and 12 inoculated with other strains lost in weight. The average gain per rabbit in the 7 of this group was 88 grams in an average of 22 days, or an average of 4 grams per day. The other 12 inoculated with approximately the same quantities of other strains, lost an average of 11 per cent, or an average of 112 grams, in an average of 11 days, or 1 per cent per day. When we compare this part of the group with those of this group which died spontaneously, we find that on account of their more severe infections these 16 rabbits lost on an average, 18 per cent, or 209 grams in an average of 6 days, or 3 per cent per day, which it will be noted, is a much more rapid rate than in the preceding, and indicates a marked difference in pathogenicity of the strains.

In a larger series of a similar study, in which 108 rabbits were used, 55 died spontaneously in an average of 7 days, with an average loss of weight of 226 grams, or 20 per cent. This, it will be noted, amounted to a loss per day of 2.9 per cent per rabbit. Of this group of 108, 53 were chloroformed. Of these, 34 were

COMPARISON OF WASHED ORGANISMS AND WHOLE CULTURES.

No. in Group	Death	Days Lived	Loss			Gain		
			Actual	%	% per day	Actual	%	% per day
A. Organisms Washed and Suspended in Sodium Chloride.								
8	*Spontaneous	7	203	18	**2.6**			
4	Chloroformed	31				336	26	.8
11	Chloroformed	10	119	10	1			
B. Whole Culture								
7	Chloroformed	22				88	9	4
12	Chloroformed	11	112	11	1			
16	*Spontaneous	6	209	18	3			
55	*Spontaneous	7	226	20	**2.9**			
34	Chloroformed	23	150	11	.5			
19	Chloroformed	46				167	13	.3

* Spontaneous deaths in Group A—35%
* Spontaneous deaths in Group B—50%

FIGURE 24.

chloroformed in an average of 23 days, having lost an average amount of 150 grams, or 11 per cent, or ½ of 1 per cent per day per rabbit. Nineteen of those chloroformed, in 46 days had gained an average of 167 grams, or 13 per cent, and $^1/_3$ of 1 per cent gain per day per rabbit.

In all of these groups there were lesions in various organs and tissues of the body. We will not here discuss their nature and their relation to the disturbances from which the patients were suffering, as these will be taken up in subsequent chapters.

A typical illustration of the evidence that the host may build up and maintain a defensive mechanism by which the toxins are neutralized is found in the following experiment. A virulent strain which grew from an extracted tooth killed a number of rabbits in succession by the planting of the tooth beneath the skin, being thoroughly washed each time before replanting. The exudate aspirated from the vicinity of the tooth during the period while the animal was showing a vigorous defense, when injected through the skull of a rabbit into the brain, produced very much less disturbance than the same material aspirated when the rabbit's defense was rapidly breaking and it was nearly overcome by the infection. In fact, the filtrate from the material aspirated at the time the rabbit had a good defense, a fine Berkefeld filter being used, when injected into the brain of a rabbit, produced so little disturbance that both it and the control injected similarly with normal salt solution were as frisky as normals for weeks afterwards. And, similarly, an old culture that had become toxic by large quantities of dead organisms sufficient to inhibit its own growth, when injected into the brain of a rabbit, produced death in a few hours; whereas, the same quantity and approximately the the same bacterial density of a freshly grown culture similarly inoculated produced no evidence of effect for many hours. In the chapter on the mechanisms of defense I have discussed this question from another angle, and also in the chapter on sensitizations.

SUMMARY AND CONCLUSIONS

(a) With regard to the matter of morphology, we have found that in approximately 98 per cent of cases, the organism involved in root canal and root apex infection, a coccus growing in diploid and chain form, is present, and that this organism is nearly always the only one recovered from animal passage; and in some teeth this organism, though present, grows with very great difficulty; and we conclude that it is present in practically 100 per cent of cases, even where other organisms grow out in artificial medium and it does not.

(b) The biological qualities of the organisms growing in root end infections, as determined by their sugar fermentations, show ten varieties of diplococci present in one hundred cases.

(c) The elective localization qualities were found to be related neither to the morphology nor sugar fermentation qualities; that various tissues were invaded in the order of chance so far as the different varieties were concerned. Note: *This does not imply that the particular tissue breaking down is a matter of chance.*

(d) The organisms found in dental infections were found to have a very great power of accommodation to the influence of the culture medium, coming finally to grow readily in concentrations of poisons by which, originally, the strain was completely destroyed.

(e) The organisms growing in dental infections are found to produce toxic substances which have very far-reaching and disastrous effects on experimental animals.

We would, accordingly, summarize the role of the bacterial invasion in dental infection and express the fundamental involved therein as follows:

Dental infections involving root canals and their apices and supporting structures practically always contain streptococci, of which, biologically, there are many types or strains, any one of which may be the important causative factor for any of the various types of rheumatic group lesions, regardless of biological classification. The elective localization and attacking qualities are developed by the environment and are, consequently, a factor of the soil or host.

CHAPTER III.

LOCAL ORAL STRUCTURAL CHANGES PRODUCED BY DENTAL INFECTIONS.

PROBLEM: Is there a constancy in the local oral expressions of similar dental infections?

INTRODUCTION.

It is practically universally expected and accepted that (a) *dental infection in bone will express itself as bone absorption,* and (b) *that similar dental infections will express themselves in the local tissues of the mouth approximately the same in all people.* This group of researches has been made to determine the correctness of these assumptions.

EXPERIMENTAL AND DISCUSSION.

We are dealing here with a matter that is so fundamental that to suggest even a possibility that it may not be true will be considered by many a gross presumption. This study has not been made to disprove but to verify, in order that a justification for our faith might be found, with the expectation that we would have to look elsewhere for the explanation for the gross confusion that exists. We have again two fundamental problems and two fundamental sources of information: Clinical material provided by the patients presenting different types of lesions and conditions, and researches on experimental animals.

To determine the various effects of infections on humans with regard to tissue reactions and their constancy in various people, we have only to study more intensively the clinical material that is available. That infected teeth have an area of absorption about them in proportion to the quantity and severity of the infection, will seem to be established by the examination of the available diagnostic reports made upon almost any set of dental roentgenograms.

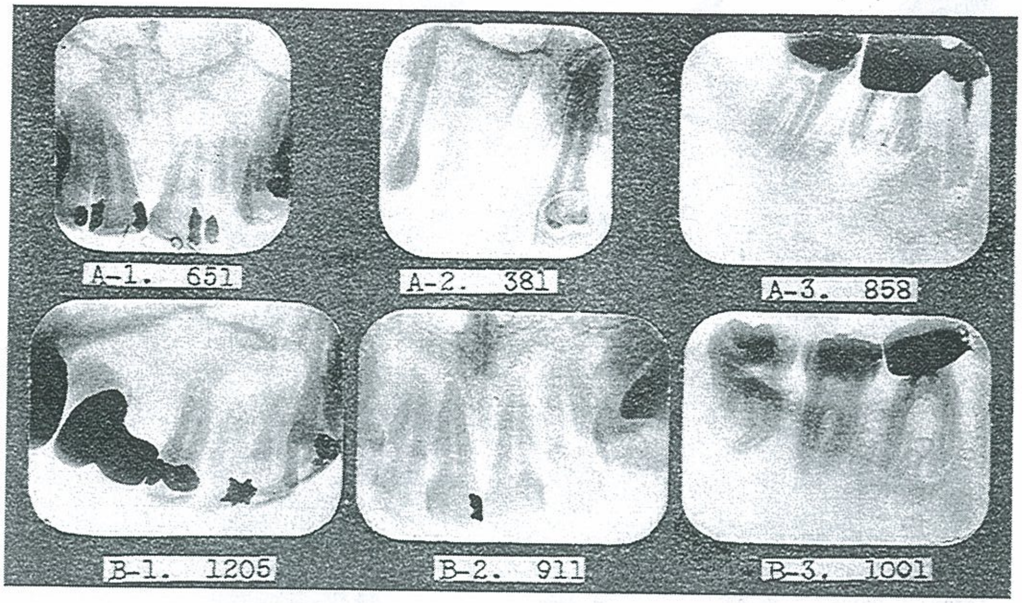


FIGURE 25. ILLUSTRATIONS OF DIFFERENT TYPES OF REACTIONS INVOLVING APICES OF ROOTS AND THEIR SUPPORTING STRUCTURES.

When we compare the roentgenograms made from similar conditions in a large number of individuals, we find, contrary to the presumption just made, very marked variations in the reactions from similar dental infections. Assuming that the amount of infection involved in the entire quantity of a putrescent pulp in either a central or lateral would be a similar and therefore comparable quantity to compare in another case, we have compared typical conditions as roentgenographically expressed and find such a condition as is illustrated in Figure 25. This shows in A-1 a right central for one patient and a right lateral for another in B-1, each showing very extensive areas of absorption. In A-2 and B-2 we show similar teeth from two other patients, which have comparable quantities of infection but which show almost no absorption about the apices. These are all of long standing and are typical of large groups of individuals.

It may be argued that the pulps of teeth may represent other factors and forces which we do not understand, and that therefore these conditions are not comparable as to quantity of infection, etc. We have, accordingly, added as A-3 a molar root and the

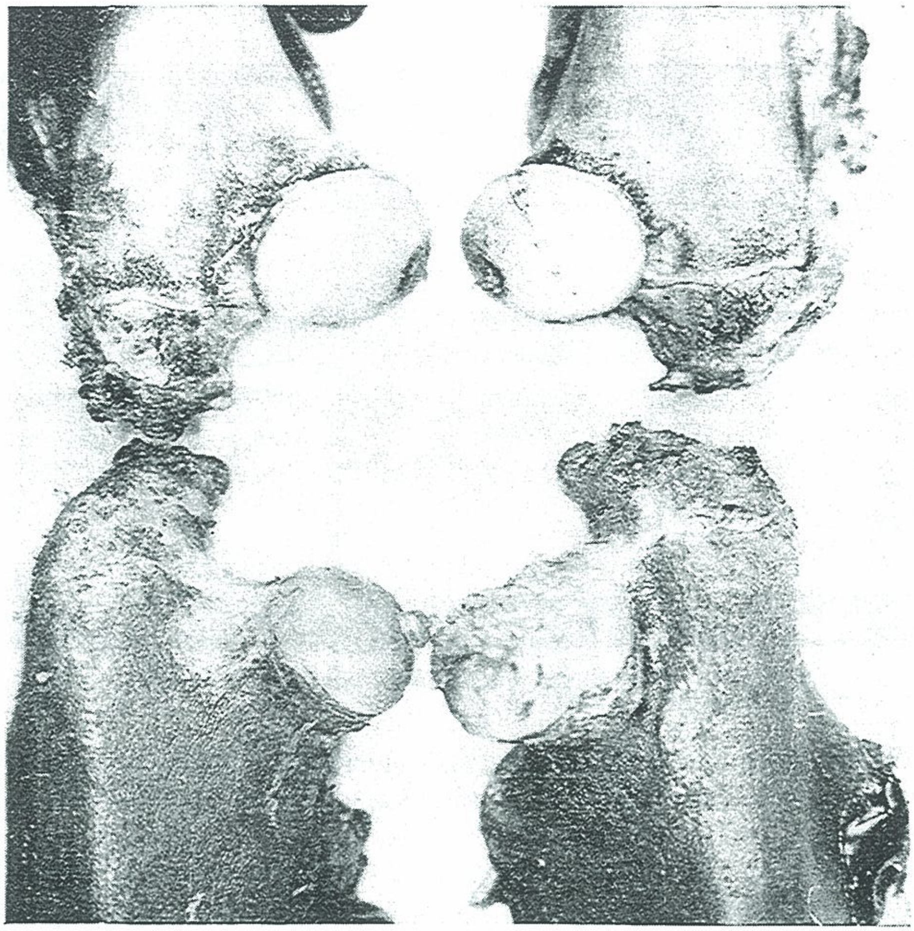


FIGURE 26. A DEGENERATIVE ARTHRITIS OF A RABBIT'S FEMUR, WITH NORMAL ABOVE.

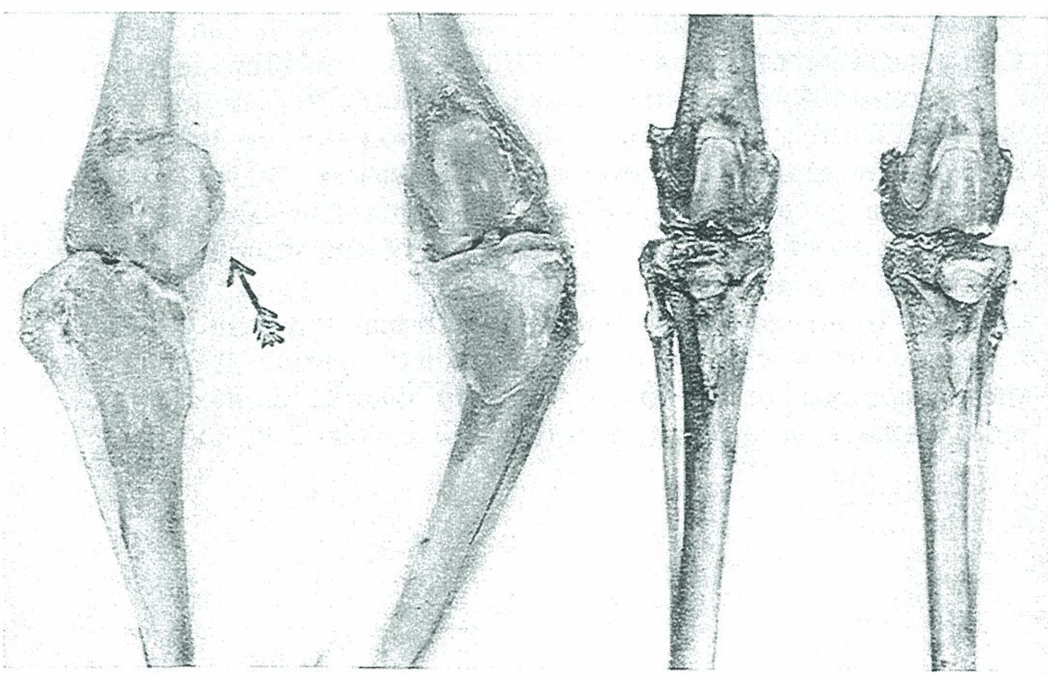


FIGURE 27. PROLIFERATIVE ARTHRITIS OF A RABBIT'S KNEES WITH NORMALS TO RIGHT.

structures beyond it showing very extensive absorption, in which one root is apparently nearly filled, the other partially so. In comparison with this we have shown in B-3 a molar tooth with apparently little or no root filling and a very little absorption about the mesial root and practically none about the distal root.

Culturally, practically all these pulp canals have been demonstrated to be infected by the same type of germ:—chiefly a diplococcus in fresh smear taken from the pulp chamber, but growing also in streptococcal form in liquid media. If, as has been quite universally accepted, all individuals react locally approximately the same, then the conditions found in A and B of Figure 25 will, of necessity, be related to the type of invading organism. The chart in Figure 18 of Chapter 2, Problem 2, "The Nature of the Organisms Involved," illustrated that no constancy has been apparent in the type of streptococcus found in different types of lesions. In the consecutive culturing of several thousand teeth from over a thousand individuals, which had been diagnosed as being infected, practically all were found to be so; and the type of tissue reaction found seemed clearly to be related to some factors other than the type of organism. The chart in Figure 17 of Chapter 2 shows graphically the relative number of different types of lesions produced by the different strains of streptococcus, and shows the relationship to be approximately that of chance and not dependent upon sugar fermentation qualities. If the difference in general structural change in Figure 25, A and B, is not due to the lesions being produced by different biological strains of streptococci, it immediately suggests that either various strains may take on the ability to produce these two different types of reactions, or these reactions may be related directly to differing qualities in the host.

Figures 26 and 27 show similarly two distinctly different types of reactions in the bones of rabbits. In Figure 26, we have extensive absorptions of the head of a femur, which is shown in comparison with its own mate of the opposite side, and two normals shown just above it. In Figure 27, the organism has produced an entirely different type of reaction; in this case, a deposition of lime salts instead of a rarefaction. Greatly enlarged knee joints are shown to the left and, for comparison, two normals shown beside them to the right. Since different rabbits inoculated with this culture produced the same type of reactions in bone tissue, we assume that the characteristic resided in the organism at this

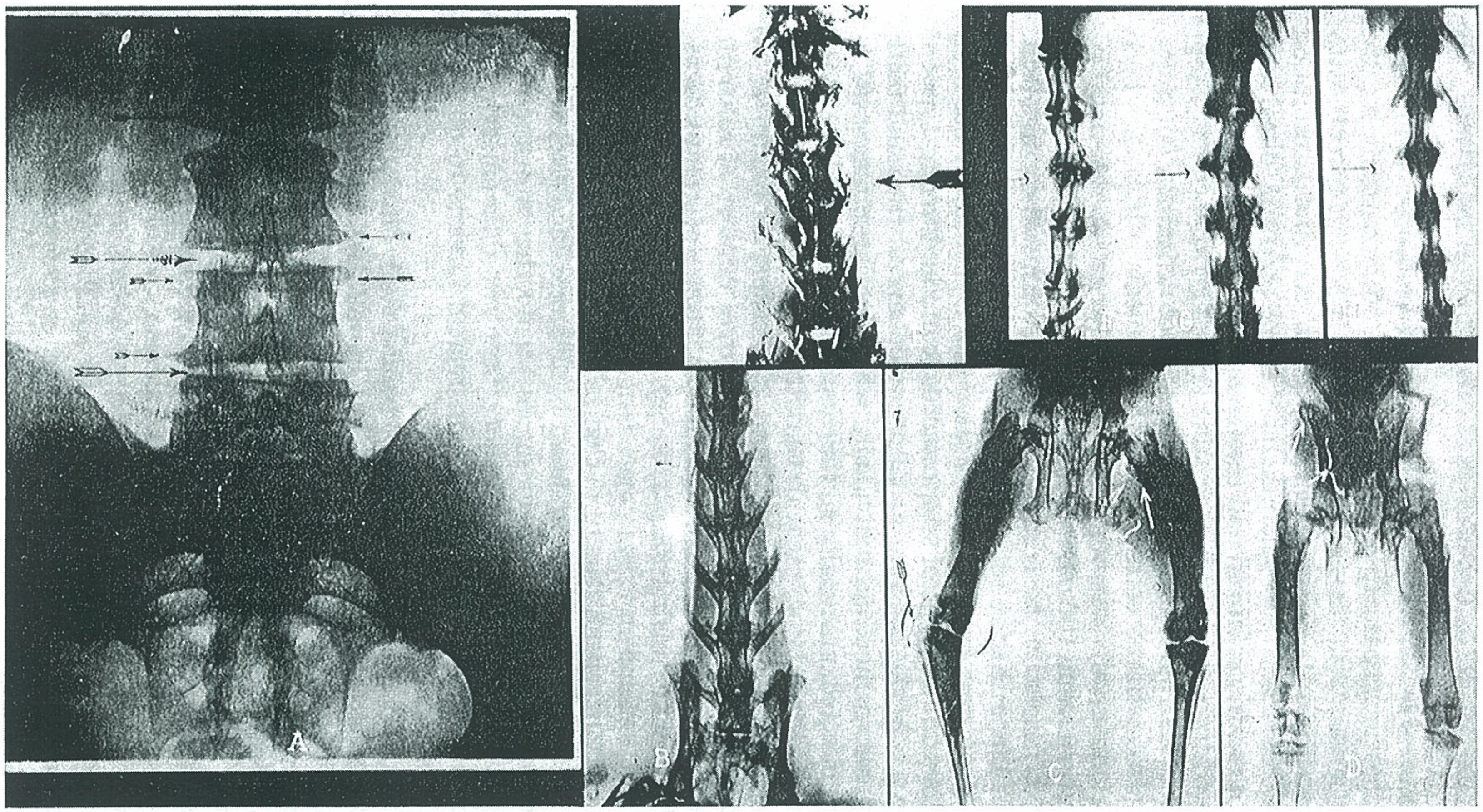


FIGURE 28. PROLIFERATIVE DEFORMING ARTHRITIS OF PATIENT'S SPINE TO THE LEFT. PROLIFERATIVE DEFORMING ARTHRITIS OF RABBIT'S PELVIC BONES, KNEES, AND SPINE, TO THE RIGHT.

time and not in the host. When we compare the reaction in the rabbit with the reaction in the patient from whom the organism was taken, we find that in the latter case it was from a man suffering from deforming arthritis, with such extensive depositions in his spine that he could scarcely bend or rotate from his head to his hips; and the roentgenogram of his spine, shown in Figure 28, and discussed in detail in Chapter 64 shows marked spinous production as do also the roentgenograms of the spines, bones, hips, etc., of the rabbits inoculated with that culture, shown also in the same figure. The source of the culture from which the inoculations were made producing the lesions shown in Figure 26, was a patient having marked nervous system disturbances without arthritis.

In Chapter 2, Problem 2, "The Nature of the Organisms," we have found that the qualities of the organisms, particularly that of growth, will be determined by the qualities of the culture medium, that they have the capacity to adapt themselves to their environment through a very wide range. This is also illustrated by the following: A strain which produced heart lesions in 93 per cent of a group of thirty rabbits inoculated with a twenty-four hour growth, produced heart lesions in only 10 per cent of rabbits when the organism was grown on artificial media for seventeen days.

Many of the researches reported in various chapters illustrate that, the organisms taken from different teeth of the same patient, produced when in the patient, similar local tissue changes, and when inoculated into rabbits produced similar types of bone change systemically to those suffered by the patient, as illustrated in Figures 27 and 28. These many studies suggest that the host determines in large measure the characteristics but not the biological type or classification of organism; and the nature of the tissue change which accompanies the presence of the organism and also determines that there is some difference, in most if not all patients, in the local tissue reactions related to the quantity of local infection, seems evident; but that the former is a direct measure of the latter, regardless of the characteristics of the host, is studied in the following experiments.

Results of these studies should be compared with the data developed in the researches presented in Chapter 2, "The Nature of the Organisms Involved."

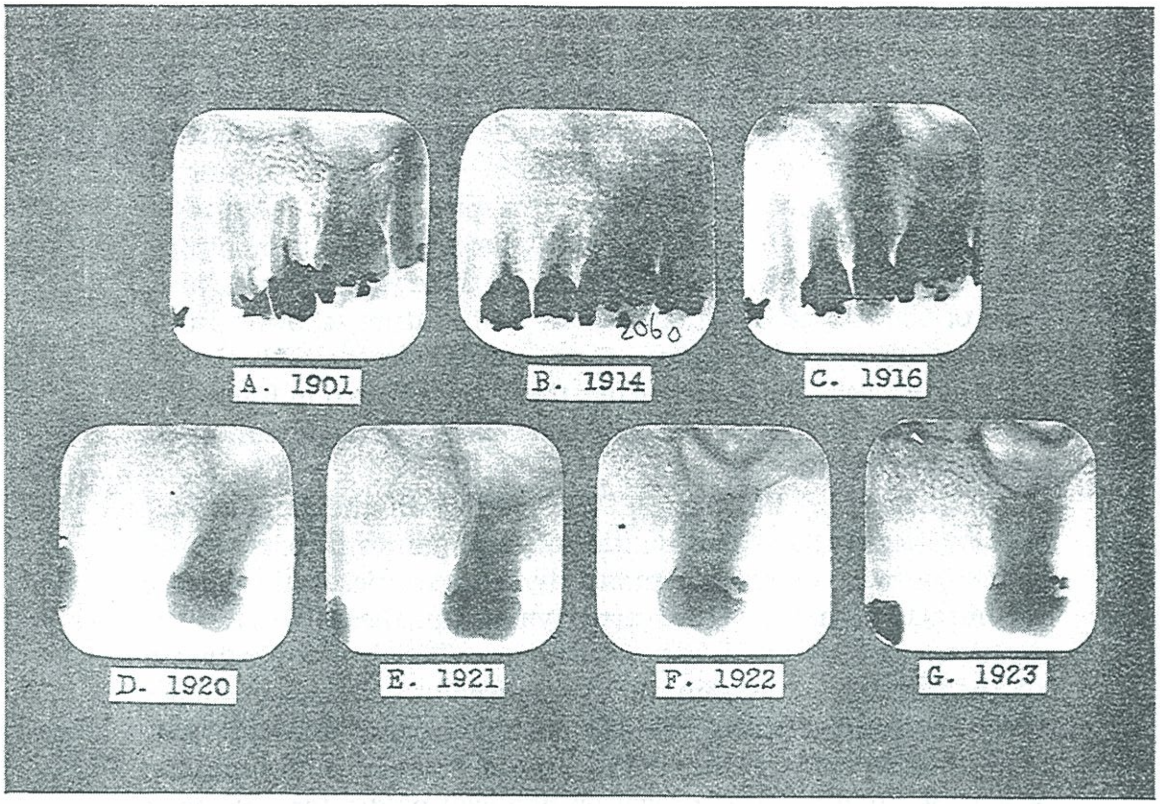


FIGURE 29. PROGRESSIVE VIEWS OF THE BONE ABOUT THE SECOND BICUSPID OF A PATIENT WITH DEFORMING ARTHRITIS OVER A PERIOD OF TWENTY-TWO YEARS. NOTE EXTENSIVE CONDENSING OSTEITIS IN C, DISPLACING RAREFYING OSTEITIS IN A, FOLLOWING TREATMENT AND REFILLING OF ROOT, WHICH CONDENSATION DISAPPEARED AFTER EXTRACTION. TOOTH AND BONE BOTH INFECTED.

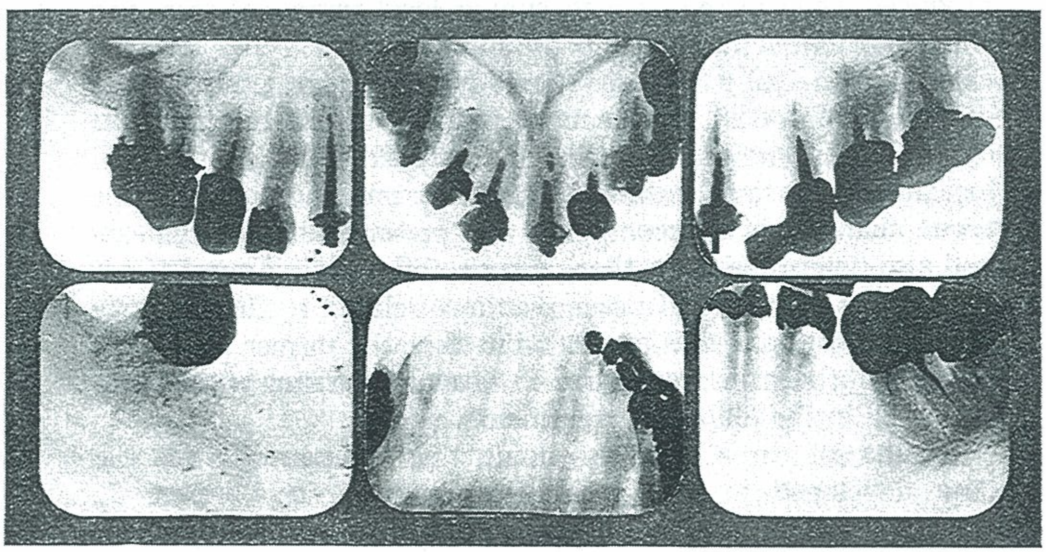


FIGURE 30. SHOWING SIMILAR LACK OF REACTION ABOUT SEVERAL INVOLVED TEETH OF SAME PATIENT.

Note: It will seem like a paradox or contradiction when I have said that the culture medium rather than the organism determines the type of tissue involved in the elective localization processes and then state that the lesions produced in the rabbits by the organisms tend under certain conditions to produce the same type of bone change in the rabbit as that organism would in the patient. In another chapter (Chapter No. 22) we show that the organisms rapidly lose their elective localization qualities when transferred to another type of media. This makes it necessary that the organism shall be transferred with as much dispatch as possible from the patient to the animal, and with as little modification by artificial growth as is possible.

Figure 29 shows the progressive history of a certain tooth: the upper right biscuspid. In 1901, twenty-two years ago, I made the roentgenogram shown as A, which was one of the early cases studied by this means. It shows clearly a tendency to a lessening of the density about the root apex. At this time I removed a poor and incomplete root filling; and after sterilizing the root with the regulation methods of the time, placed a new root filling with the assumption and confidence that all organisms had been destroyed and the tooth would remain in safe condition, for such was the confident teaching of the time. From time to time I made roentgenograms to determine that the bone was filling in about the apex. Six years ago I sent for the patient, having become convinced that it was not safe for her to have that tooth, notwithstanding the fact that it was apparently getting better, as proven according to the accepted fundamentals by the reconstruction of bone about the apex. In the meantime the patient had become progressively more seriously involved with rheumatic arthritis with so much rigidity that she had to be carried to the office. B of Figure 29 shows the dense bone forming about the apex. The tooth was not in the least tender. The tooth was extracted after using the actual cautery in the anesthetized gum at the neck of the tooth to destroy infection at the gingival border. It was cultured; animals were inoculated. The culture of the tooth showed a very profuse streptococcal infection. Cultures were taken from the bone surrounding the root apex to a distance of one-fourth inch from the apex, which grew out the same type of organism notwithstanding its density. About four months later, the same bone was cultured by sterilizing the external alveolar tissue with a cautery, after anesthetizing, and going through into the alveo-

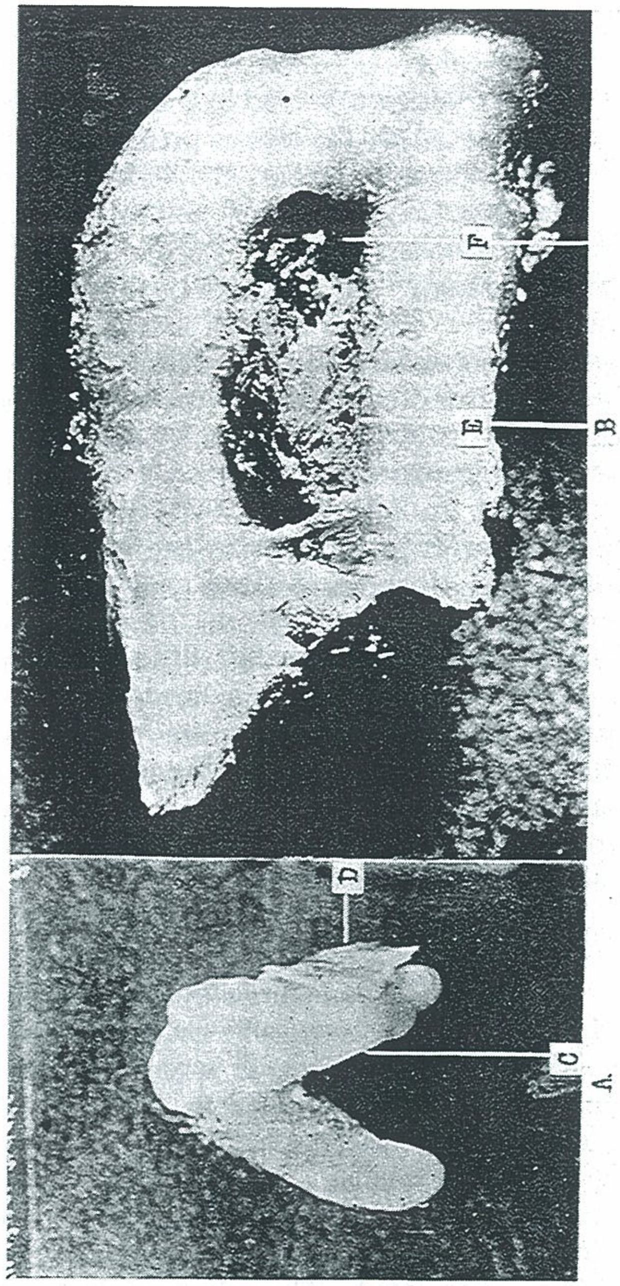


FIGURE 31. A AND B SHOW TWO VIEWS OF AN EXTRACTED MOLAR ROOT OF THE CASE SHOWN IN FIGURE 30, WITH MARKED EXCEMENTOSIS FUSING THE MESIAL AND BUCCAL ROOTS, AND AT D, THE FIRMLY ATTACHED PIECE OF ALVEOLAR BONE.

lar bone, and the same organism was found. C of Figure 29 shows this alveolar bone two years later, at which time the dense bone was becoming less dense. Incidentally, the patient improved after the extraction of these and other similar teeth so that she was able to get about on crutches, whereas formerly she had to be carried. Note that, in this case even the large quantity of infection in a half filled root canal of long standing, produced only a radiolucency without the development of granuloma. A lesser quantity of infection produced no radiolucency but a radiopacity. We are, in this patient, apparently dealing with forces which resemble in their effect those expressed in Figure 25, A-2 and B-3, and quite unlike those in Figure 25, A-1, B-1, and A-3.

A careful study of the various types of local reaction found resulting from dental infections was made as a part of this research by comparing different teeth in the same individual. This was done with a large number of individuals. Figure 30 is a typical illustration. Here it will be noted that the roentgenographic evidence of the condition would indicate a similar type of condition in the bone surrounding the roots of the many involved teeth. None had extensive absorption, notwithstanding the fact that we have the quantity of irritant that would be available from a putrescent pulp, as seen in the upper left first biscuspid and in the unfilled roots of the mesial root of the upper left first molar, all upper incisors, the mesial root of the lower right second molar, and the upper right first and second molars. The condition in this mouth, as revealed at the time of operation, showed that there was a very unusually dense bone about the roots of all the infected teeth, as also a marked excementosis of the roots of the upper left first molar which is shown in photograph in Figure 31. It will be noted that the mesiobuccal and distobuccal roots are fused together so completely as to be a continuous mass. When this root was extracted, the alveolar bone was so adherent that a piece of it came away with these roots, shown in Figure 31-B.

In striking contrast to this last case, we find many patients with very extensive areas of absorption about similarly involved teeth. Such a one is shown in Figure 32. In this case it will be noted, the upper central with very little evidence of unfilled root space has a very extensive zone of absorption, as does also the upper left first biscuspid and lower right first biscuspid; and this is the more important because the former was a young person and this patient is fifty-six years of age. The matter of the compari-

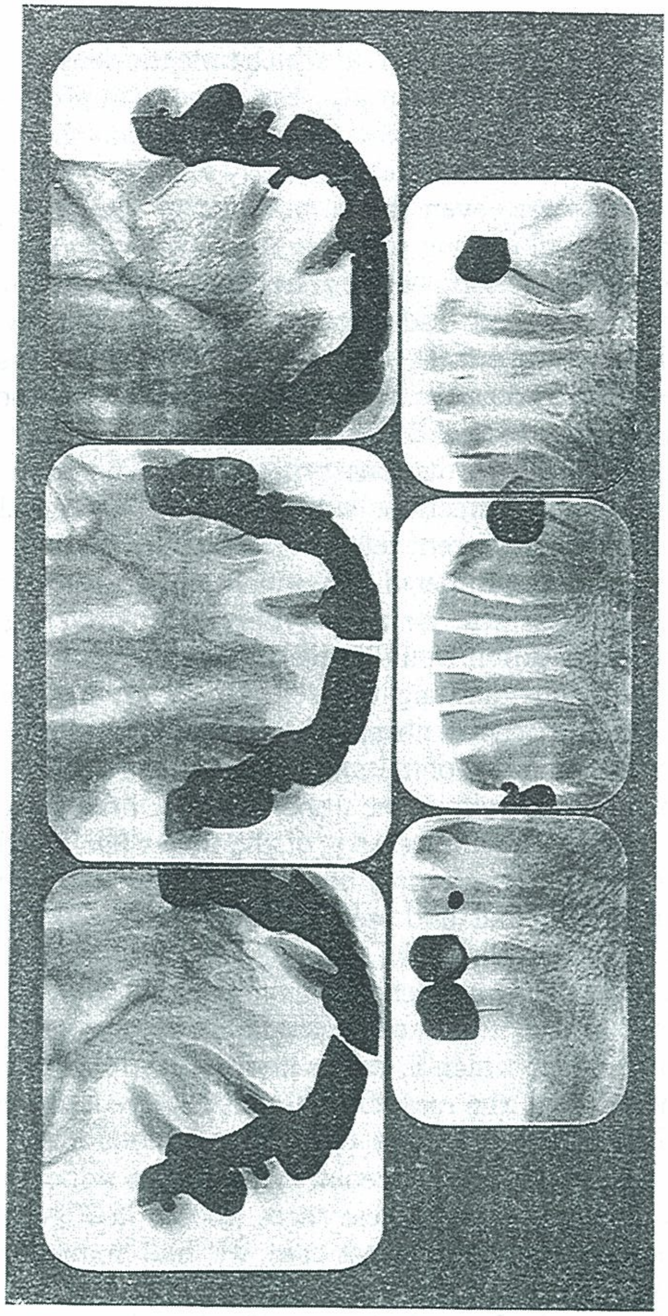


FIGURE 32 SHOWS EXTENSIVE ABSORPTION OF ALVEOLAR BONE ABOUT INVOLVED TEETH OF THIS INDIVIDUAL, IN CONTRAST WITH FIGURE 31.

son of the health of these two individuals is one which we will take up in subsequent chapters, for it has very direct and important bearing on the type of pathology.

A study was made of the different members of the family in a large series of families to ascertain whether the same general conditions tend to prevail; that is, whether a dental infection tends to produce the same type of tissue reaction about the teeth in the different members of a given family. Figure 33 shows a typical illustration of this study. In it we have the roentgenographic record of the reactions to dental infections in three sisters and a brother of the patient reported in Figure 31. It will be noted by comparison, for example, of the two roentgenograms shown in each A, B, C, and D, that there is a very marked similarity in the type of tissue change, as was also demonstrated at the time of surgical removal of these teeth in the clinical condition found. Note particularly the mesial root of the lower molar in A and part of the distal root of the same, give the same evidence of incomplete root filling, which condition also exists in the upper right second molar. B shows a similar condition of a lower and an upper molar; C does of a lower molar; and D of an upper molar and incisors. If quantity had been the all-determining factor, as expressed by the capacity of the space left in the unfilled root, these might all be expected to have large apical areas of rarefaction. In all the teeth of these various members of this family there was found not only the similar condensing osteitis but a very great tardiness in the healing process. The sockets tended to become infected and painful, requiring relatively long periods for the filling in of the alveolus. It should be noted that in addition to the similarity of these members of this family from the standpoint of the local structural reaction to dental infections, there was also a marked similarity in systemic involvements, all of which were breaking at from twenty to thirty years of age. This we have discussed in further detail in Chapter 59.

Similarly, in Figure 34, we have the roentgenograms of the teeth of the brother and sister of another family showing an entirely different type of reaction to dental infection. In this case there are areas of very extensive rarefaction about all involved teeth. At the time of surgical removal of these teeth it was found that, whereas in the last case they were all exceedingly difficult to extract and all surrounded by very dense bone, in this family they not only have roentgenographic evidence of exten-

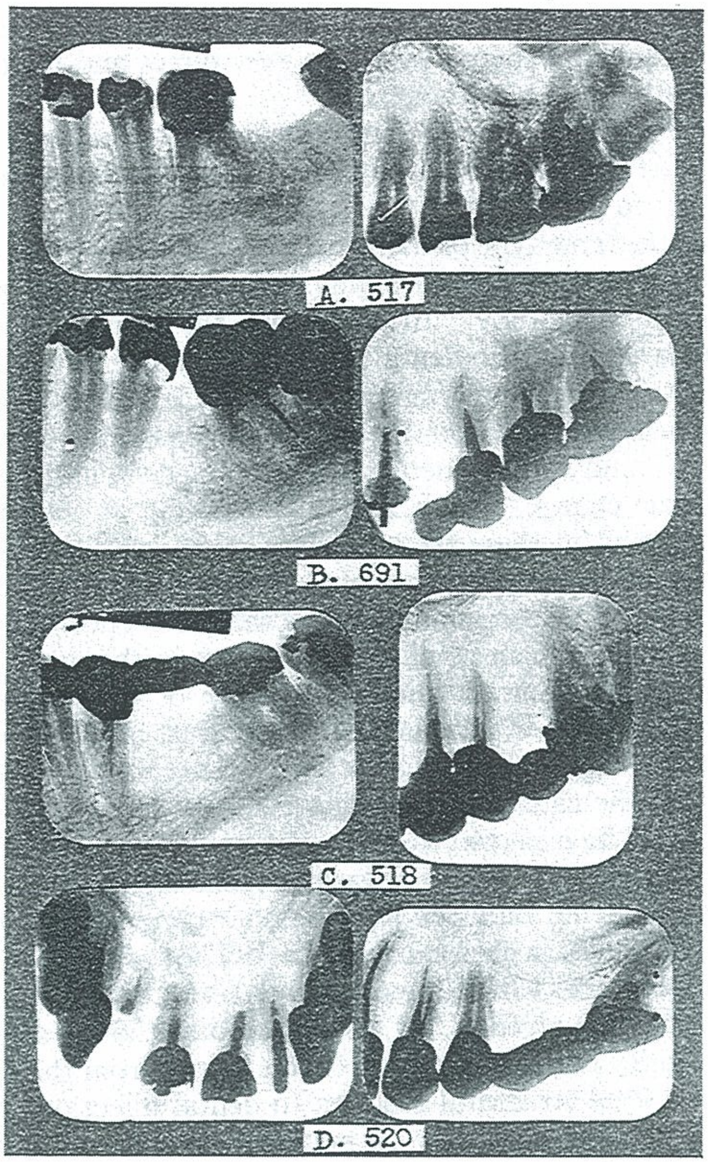
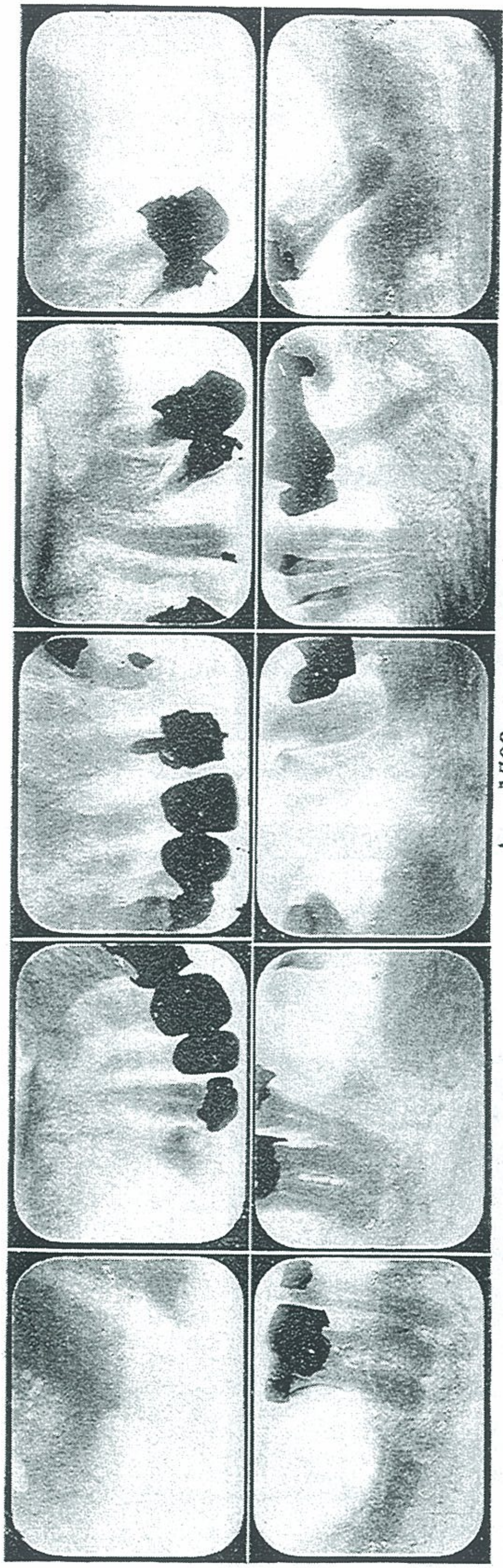


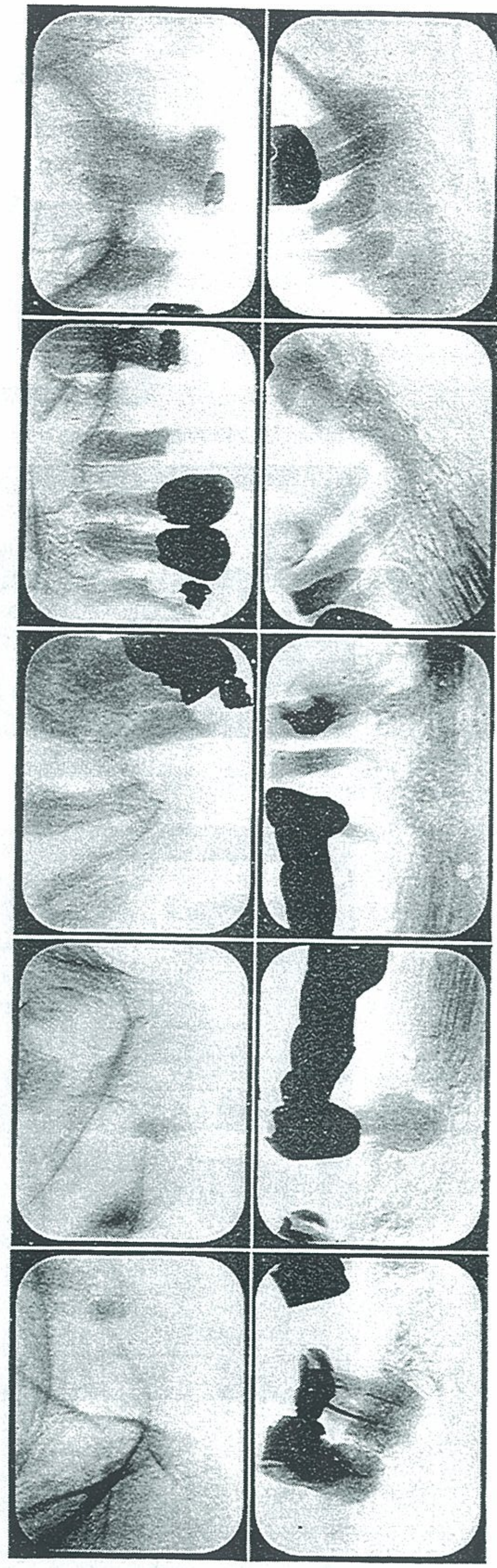
FIGURE 33. A COMPARISON OF THE TYPE OF REACTION IN DIFFERENT MEMBERS OF THE SAME FAMILY. NOTE THE ABSENCE OF APICAL REACTION.

sive areas of absorption in all members of the family where dental infections exist, but at the time of surgical removal it was also found that there was a marked similarity in the structural conditions. The teeth were easily extracted; the bone was not dense about them; and the sockets healed with great rapidity and without discomfort or secondary infection. Nature in these instances did not require any assistance.

A study was then made to see in what different groups individuals could be placed, with regard to the type of reaction de-



A. 1399



B. 1398

Figure 34. A comparison of the type of reaction in different members of the same family. Note the presence of extensive apical reaction.

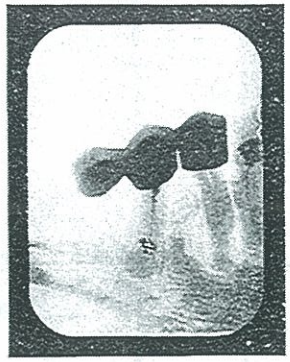


FIGURE 35. DIFFERENT TYPES OF REACTION ABOUT ADJOINING TEETH.

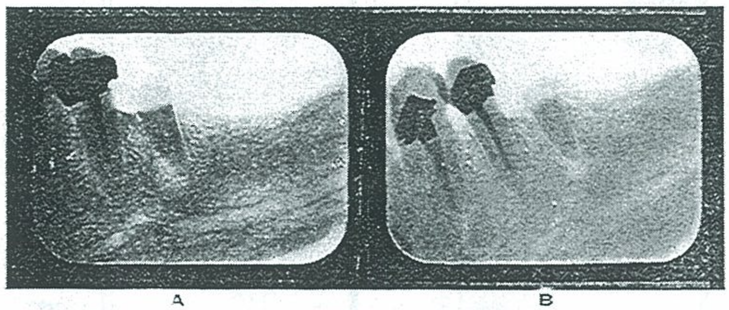


FIGURE 36. AN APPARENT IMPROVEMENT IN DENTAL CONDITION FOLLOWING BREAK IN HEALTH.

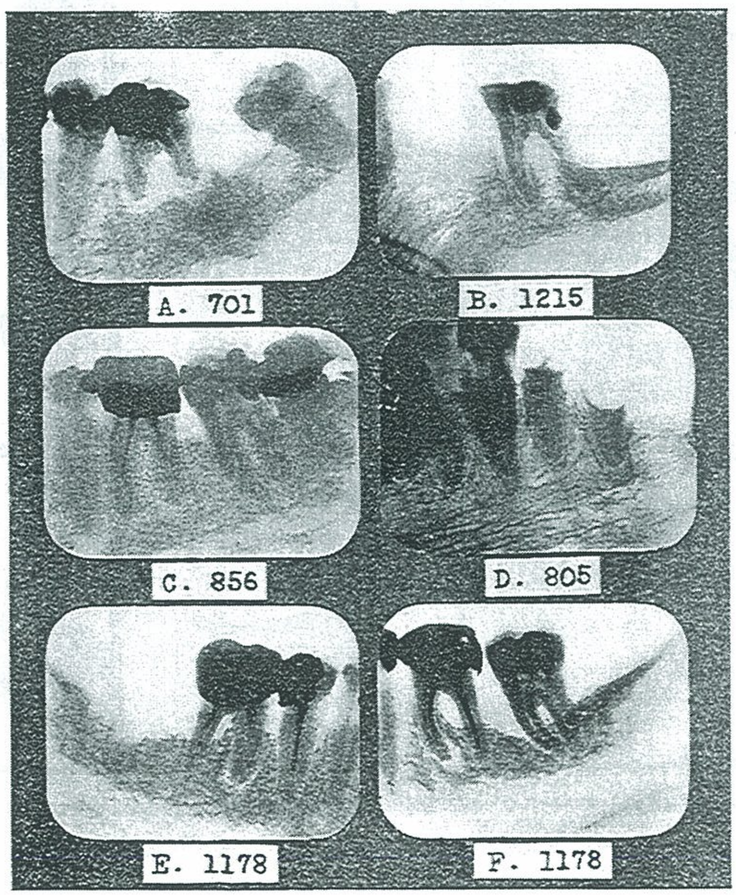


FIGURE 37. ROENTGENOGRAPHIC APPEARANCE OF CONDENSING OSTEITIS ABOUT INFECTED TEETH.

veloping in tissue, as a result of various types of dental infections, and it was found that in addition to the two types illustrated above, there was frequently found a type which was similar in many respects to that in Figure 34, except that the area of rarefaction seemed bounded by a zone of more dense bone, as it were, a zone of condensing osteitis around one of rarefying osteitis. This type is illustrated in Figure 37. It was found that the various patients having this type of condition were comparable in that the teeth were frequently difficult to extract, often had a history of a fistula having existed previously, and closing sockets did not heal as readily as the type in Figure 34. In many respects individuals of this class presented sockets that were similar to those in Figure 33 in the condition found clinically and the difficulties attending the healing process, while the conditions roentgenographically resembled in a general way in that there were frequently large areas of absorption, the type of reaction found in the group as shown in Figure 34.

While extensive rarefaction as a result of infection, and condensation or absence of rarefaction, may be typical expressions of different types of systemic reaction, therefore appearing in different individuals with different physical characteristics, it is possible to find both these conditions in the same individual, as shown in Figure 35 which shows a very marked area of alveolar absorption around the root of the first biscuspid with no absorption around the second biscuspid, which latter has an excementosis. This will appear to some like a contradiction. It probably is due to the following conditions: It will be noted that the first biscuspid with its large area of absorption has a zone of condensed bone surrounding that zone of rarefaction; also that it has evidence of an absorption process on the root; and this tooth had all the infection of the unfilled pulp canal in addition to the infected dentin as a source of irritation. The second bicuspid has a root filling and probably has very much less total infection and capacity for toxic material than the first bicuspid. It is also probable that in addition to this great difference in quantity, the condensing osteitis of the first bicuspid has formed during a period succeeding the period during which the absorption developed; and it will be shown later that the condition of excementosis about the second bicuspid has probably taken place during the period in which the condensing osteitis surrounding the rarefying osteitis

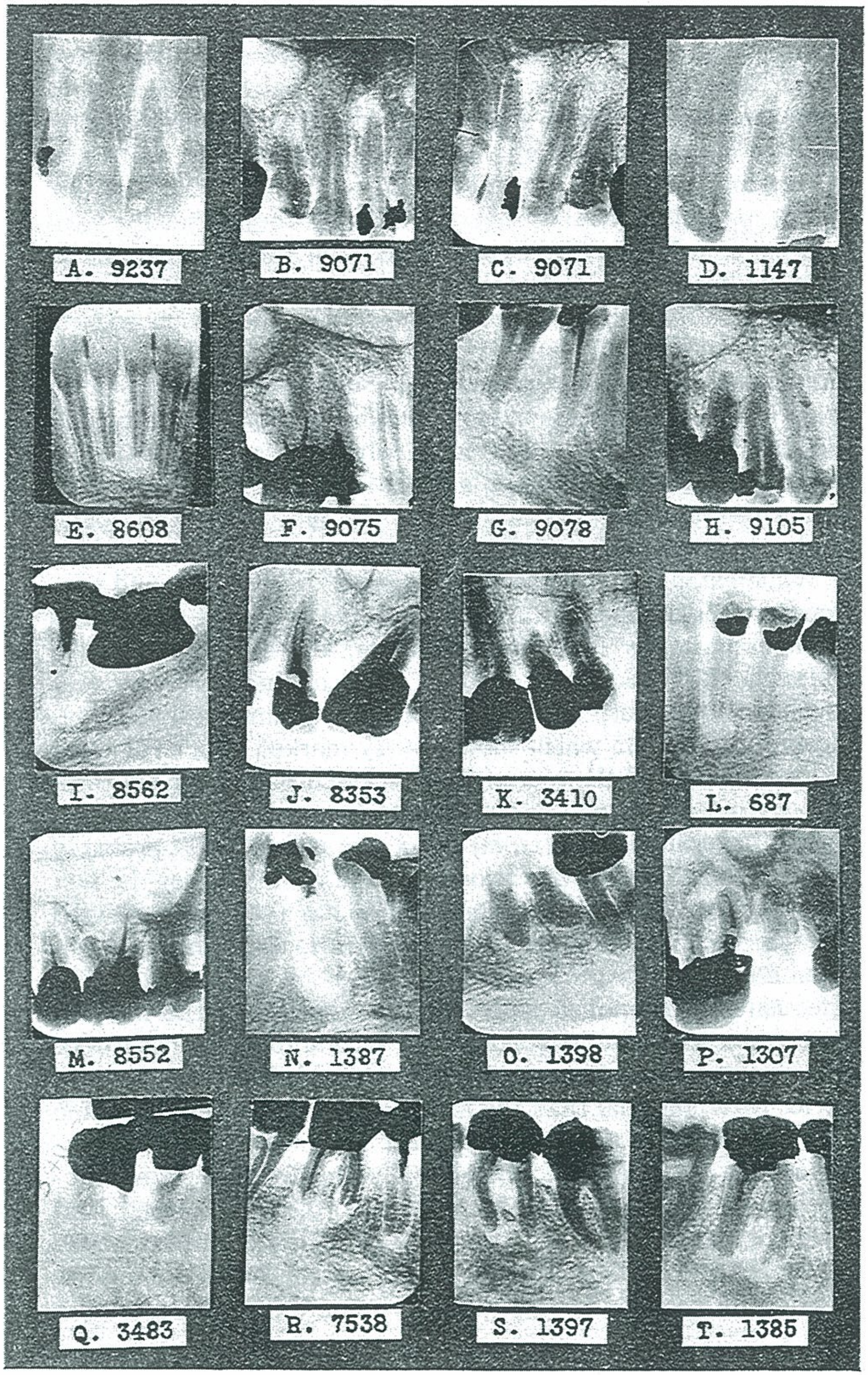


FIGURE 38. A GROUP SHOWING EXTENSIVE RAREFYING OSTEITIS ABOUT INFECTED TEETH.

occurred about the first bicuspid. Just as in geology the recorded depositions may represent entirely different conditions because they occur in different periods, similarly, recorded bone changes may chronicle entirely different types of reaction because they belong to different periods; in other words, one of the important elements involved is the environment, together with the fact that a small quantity of irritation may be sufficient to produce just enough stimulation to make a deposition; whereas, a much larger quantity goes beyond the point of stimulation to irritation and produces rarefaction.

If, as we have suggested, individuals may change their capacity for reaction to a given dental infection, we should find evidence of this about teeth which have not been subjected to surgical interference or medication. Such a condition is shown in Figure 36 in which in A we have the extent of the zone of rarefaction about an infected bicuspid root and which six months later, as shown in B, has a smaller area. It is not probable that the infection has become less serious either in virulence or quantity. There is an important relationship, however, to the patient's health which will come out in the next chapter.

We have, accordingly, studied carefully a very large number of roentgenograms covering all of those in a busy clinical practice for many years, and have found evidence in the roentgenograms to suggest their classification into three main groups on the basis of the type of structural change produced in the supporting structures about infected teeth. We have, accordingly, made up a page of each of these three groups: namely, those in which there is evidence in the roentgenogram of a vigorous reaction about the tooth, expressing itself with extensive absorption of alveolar bone and in some instances with absorption of the roots themselves. We have placed the centrals and laterals at the top, bicuspids next, and molars at the bottom. Figure 38 shows the group with this splendid reaction.

In the next group (Figure 39) we have presented those in which there is evidence of a change in the type of reaction in which there has been originally a very abundant absorption, but about the periphery of which absorption area there is evidence of a condensed layer in contrast with the preceding group in which in many or most instances the zone diffuses off into cancellous bone. Whereas the medullary spaces are frequently enlarged and have direct connection with the absorption chamber, in this group there

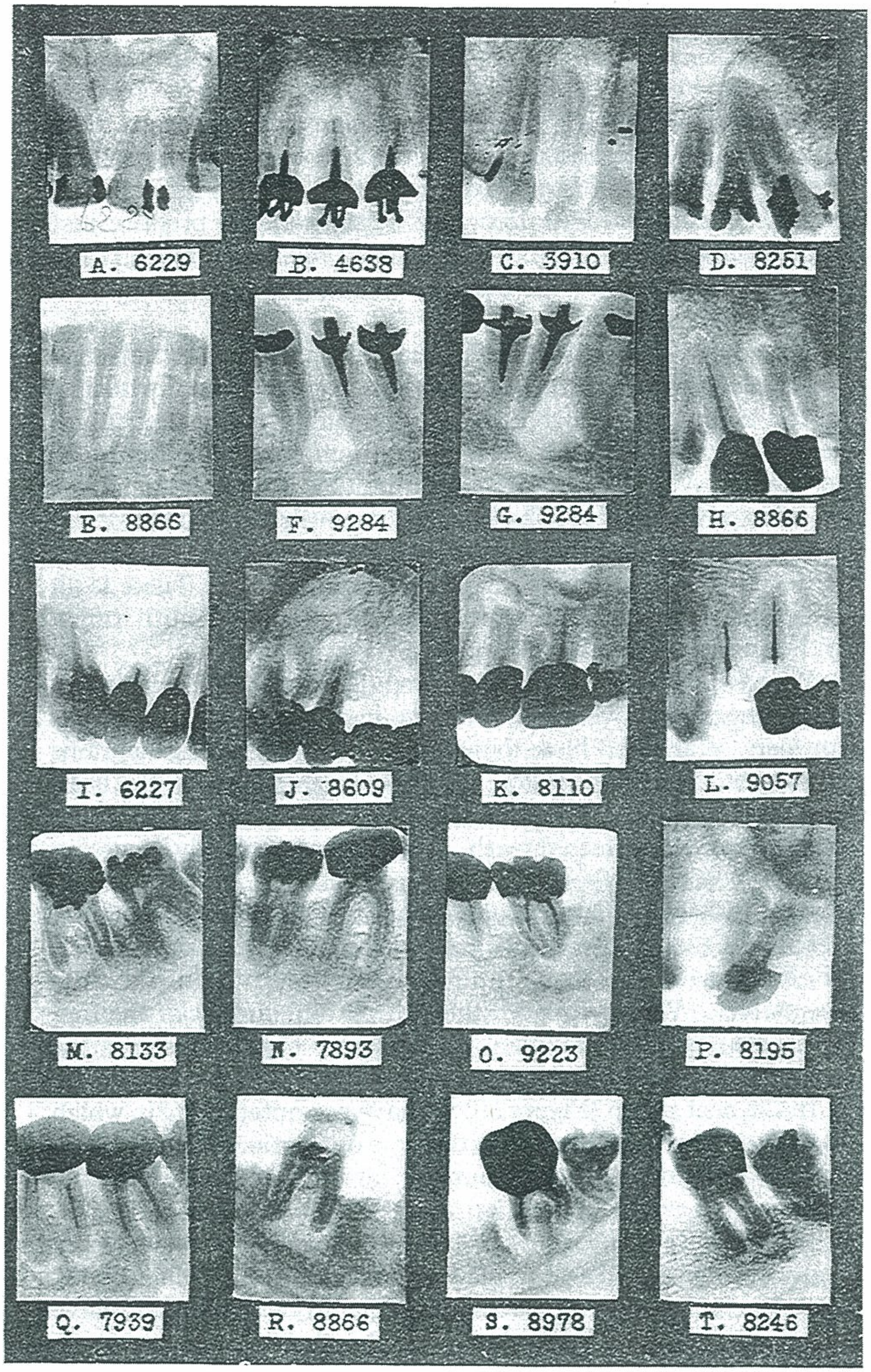


FIGURE 39. A GROUP SHOWING A ZONE OF CONDENSING OSTEITIS SURROUNDING A ZONE OF RAREFYING OSTEITIS ABOUT INFECTED TEETH.

is a reduction in the medullary spaces with an enlargement of the trabeculae adjoining the absorption chamber, which latter is frequently surrounded by a dense continuous bony shell, often polished like the inside of an egg shell, with occasional or very few connections with the medullary spaces. There is evidence of distinct change in the progress of this reaction; whereas there was one time an active absorption with enlargement of the chamber, there is now a deposition at the periphery of the granulomatous tissue with the obliteration of the medullary spaces.

A third group is shown in Figure 40, in which there has never been an extensive zone of absorption about the root apices and in many instances there seems to have been much obliteration of the cancellous structure of the bone. The medullary spaces are small or lost by the fusing of the trabeculae. In this group there is no evidence of there ever having been a fistula. The teeth were seldom, if ever, tender. There is very little reaction about the tooth and what there is, is largely a deposition process about a very small zone of rarefaction.

Clinically, there are many characteristics in common in the members of these groups but in contrast with those of the other groups. For example, in the teeth of the first group there is generally a fistula with recurring exacerbations of tenderness or painfulness. This is continued to the present. The sockets left after the extraction of the teeth of the first group tend to heal readily without treatment or tendency to local infection. The members of the second group often show the evidence of an old scar of a fistula but none, of late. Formerly the tooth got tender but not so, recently. These teeth tend more to infection of the sockets following extractions than those in Group 1 and require more postoperative care after exodontia. Those in Group 3 never had a fistula; seldom, if ever, became tender; the sockets tend very readily to infection after exodontia, often very painful requiring careful postoperative care, for both this group and the one preceding tend readily to develop what is frequently referred to as dry socket following extraction, which the former group in Figure 38 never does. Another characteristic of the groups will be that the teeth in Group 1, Figure 38, tend to be extracted easily, those in Group 2, Figure 39, quite difficult, and those in Group 3, Figure 40, often exceedingly difficult. With regard to infiltrative anesthesia, those in Group 1 are very readily anesthetized; those in Group 2 less so; and those in Group 3 tend to be very difficult.

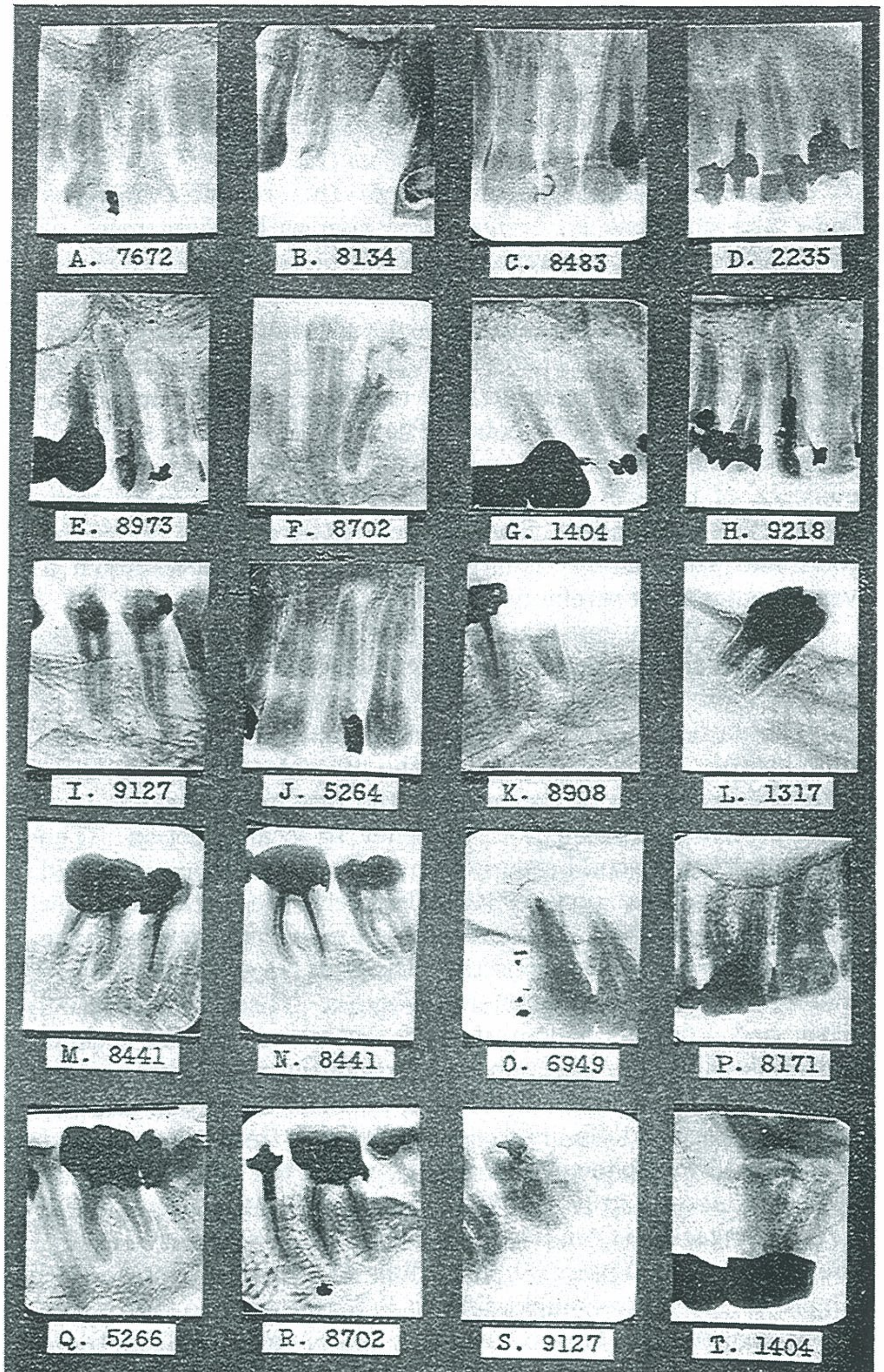


FIGURE 40. A GROUP SHOWING LIMITED REACTION, WITH OR WITHOUT CONDENSING OSTEITIS, ABOUT INFECTED TEETH.

SUMMARY AND CONCLUSIONS.

1. An analysis of these data reveals that the first premise—namely, *that infection will express itself in bone as absorption*—is not a constant finding, assuming that the quantity of infection that would be represented by an infected pulp of a single rooted tooth represents approximately the same quantity for a given tooth in various individuals. The extent of absorption is not the same in all these individuals, for there may be very extensive absorption in one case and in the very next case very little absorption or a previous deposition of bone, or both. And this difference is not explainable on the basis of quantity; for, indeed, it will often be found that whereas in some cases the local effect of the quantity of infection represented in large pulps which are putrescent, therefore large quantities of infection, may be exceedingly small; and in other cases with even very small pulps or nearly filled pulp chambers, or even completely root-filled canals, there will be enough irritation to produce very large areas of absorption.

2. With regard to the matter of grouping of individuals, it is very evident that members of the same family tend to have the same type of reaction about their teeth, immediately suggesting an evidence of grouping.

3. Frequently in the study of individuals we find cases with quite extensive absorption but which areas are surrounded with zones of apparently dense bone. These individuals seem clearly to have had a change come into their lives. They have at one time been in the first group and have finally come to be in the second group. As a class these individuals are so characteristic that they can readily be put into a group by themselves on the basis of the local structural change about their teeth; for the inoculation of experimental animals with cultures taken from dental sources has revealed variations in conditions which vary through a wide range, from very marked absorption of bone to very definite deposition within the bone.

We can therefore briefly summarize as follows:

(a) Dental infection in bone may express itself as absorption, even extensive absorption, or may be attended by very little or no absorption, or may even produce a marked increase in the density of the bone. (b) A given dental infection will not express itself in the local tissues of the mouth approximately the same in all people. People tend to divide into groups with regard to this matter of local reaction, which groups are very dissimilar.

CHAPTER IV.

SYSTEMIC STRUCTURAL AND FUNCTIONAL CHANGES PRODUCED BY DENTAL INFECTIONS.

PROBLEM: Are all human beings similar or sufficiently so that they may be considered comparable in their reactions to dental infections?

INTRODUCTION.

If, as seems indicated from the preceding researches, the presence or absence of systemic involvements and their nature is not dependent primarily upon biological characteristics of the organisms involved, and if the local structural changes about the teeth are not the same in different individuals for a given or similar type of infection, there is strong evidence that other factors must be responsible for the presence or absence of local and systemic effects from dental infections. Historically, about all that has appeared in the literature has been a reference to a rheumatic diathesis influenced by racial, climatic, geographic, industrial, and environmental conditions.

EXPERIMENTAL AND DISCUSSION.

A fundamental law of biology, physics, and chemistry, is that similar causes produce similar effects; and since this is a biological problem, it has been very natural to suppose that even though the factor involved in the invading organism is variable, the factor involved in the host is a common and universal one. In fact, a department of applied medicine is based on this assumption: namely, clinical pathology or diagnosis. Not that all people exposed to typhoid develop a typical case of typhoid fever, but that those who do become a prey to this infection develop similar symptoms; and upon this symptomatology the disease is identified. This presupposes (1) that the invading typhoid organisms will, in general, have the same powers of attack varying within certain limits in severity, selecting the same tissues, and (2) that the reactions in the patient will be clinically similar. This being true of a great majority of pathogenic organisms has made it possible for a very general and quite effective classification of infectious diseases to be made, whether the infection entered the system by the respiratory tract, the digestive tract, or by implantation within the tissues. The invasion of the organisms has resulted in local and general tissue reactions which have been termed symptoms, and on this the whole department of clinical medicine or clinical diagnosis has been based. The near universality of this rule of symptomatology has made it very natural and logical that the same general fundamental principles would be understood to apply to both the aggressive qualities of the organisms in dental infections and the reactions of the host to such an infection if it existed.

Applying this general law of clinical medicine, we have concluded that people who are well, or are not ill, are not infected; and, similarly, teeth that are well, or at least not ill, are not infected; and teeth that have been ill and have become apparently well have ceased to be infected. The answer to the questions "What constitutes a sick or afflicted tooth?" and "What are the symptoms of an infected tooth?" has been determined by the same law of symptomatology. Since, with practically all infections the severity of the symptom is an expression of the severity of the involvement, therefore we have argued that teeth with serious inflammatory processes, whether in the pulp which is so capable of expressing abnormal states or in the peridental membrane, a tissue nearly as responsive, are or are not seriously infected in proportion to the pain and evidences of inflammation involved. This has implied that the attacking power of the invading organisms, the reacting power of the host, and the resultant of the reaction—namely, the clinical symptoms—will be comparable in various individuals as well as in various teeth of the same individual.

To determine this we have made a very careful analysis of fourteen hundred cases in order that we might ascertain the correctness of this presumption. We have undertaken to establish, so far as was feasible, the following factors: The characteristics of the host, including susceptibility to caries and susceptibility to gingival infections or periodontoclasia; the characteristics of the local histopathology; the tendency to systemic involvements and their kind; whether there is a definite susceptibility to the type of organism usually found in dental infections; whether that factor has developed recently, or whether it has been the characteristic throughout life; whether it is an inherited characteristic, whether its dominance is marked, and whether inherited from both sides of the ancestry; and the effects of overloads and the nature of those most involved.

We have found it very difficult to establish what might be considered a normal individual; and, in any group, to know how divergent they of that group are from the normal; or, of the individuals studied, what percentage any group might form of the total of society. We have undertaken to establish what might be considered normal individuals by selecting from those examined those most free from systemic involvements. We have accordingly found a very constant grouping of individuals which we shall later analyze in detail. The Chart in Figure No. 41 shows the general result of this study.

INTERPRETATION OF RESULTS OF SUSCEPTIBILITY EXPERIMENTATION

Many very interesting and unexpected units of information have been revealed, which seem to establish very definitely that human beings are not similar in the sense that the same rules can be applied to all and various individuals for the interpretation of the immediate symptoms or of their significance. In general we find that all individuals divide naturally into two main groups so far as the presence or the probable presence of systemic reactions to dental infections is concerned. We have chosen to call these groups *non-susceptibles* and *susceptibles*. The latter group, the *susceptibles*, naturally subdivides into two. The first is a group whose susceptibility is an *acquired* factor. It is made up of people who belong in the non-susceptible group, but who, because of various forms of overload which will be discussed later, have come into the group we have termed *acquired susceptibles*. There are many things which distinguish this sub-group from the balance of the general groups we have called susceptibles. The second division of the susceptibles has characteristics which are so outstanding that they make themselves readily recognized. They have never been in the first group, or non-susceptibles, and while they may not have broken during long periods of their life, they have tended to break with the presence in the system of the type of infection requisite for making manifest that inherited susceptibility. This type of infection, as will be seen, is found in practically all dental lesions. There seems to be truly an inherited quality which, as is disclosed, shows very definitely evidences of the laws of mendelism. Accordingly, we have divided all individuals into three main groups: the first, those with non-susceptibility; the second, those with an acquired susceptibility; and the third, those with an inherited susceptibility. An illustration familiar to everyone from earliest recollection will be the common phrase "Heart disease runs in this or that family."

A detailed description of what is presented in the chart in Figure 41 is as follows: The letters of the alphabet, called "Alphabetical Group," represent the first letter of the patient's name in order that a comparison may be made of as many different groups as there are letters of the alphabet for the purpose of showing the constancy of percentages, etc. The succeeding columns are "Total Number per Alphabetical Group, Average Age, Number of Males, Their Average Age, Number of Females, Their Average Age." The next section of the chart is entitled "Number of Rheumatic Group Lesions (past and present) of the Individual Being Studied" under which we have Tonsils, Rheumatism, Heart, Neck, Nerves, Internal Organs, and Special Tissues. The next division entitled "Number of Relatives Affected" indicates the number of instances in the group where Brothers, Sisters, Father, Father's Relatives, Mother, Mother's Relatives were affected with rheumatic group lesions. The next column is a combination to show the number of Severe, and Severe and Mild instances of rheumatic group lesions of the patient and family. These include the subdivisions of Tonsils, Rheumatism, Heart, Neck, Nerves, Internal Organs, Special Tissues, Total Severe Lesions (by which we mean those severe enough to incapacitate them or put them to bed or cause death), Percentage Severe, Total Severe and Mild Lesions, and Percentage of Severe and Mild.

The balance of the chart, which is divided into three main groups, consists of a study of the number of lesions severe, and severe and mild, in the three susceptibility groups, *inherited*, *acquired*, and *absent*. In all of these three groups we have in the first column the number of patients in the group, followed by the severe lesions, the average severe lesions, the severe and mild lesions, the average severe and mild lesions. It will be noted that if the figures showing percentage in any of the three groups, as, for example, Average Severe or Average Severe and Mild, are read downward, they show a marked similarity in those collections of patients which would be represented by the first letters of their names.

The figures at the bottom of the chart show the average number or percentages as per the various groupings as expressed. The heavy black leaded summary at the bottom gives the condensed summary of this very extensive study. The data here revealed constitute what I believe to be one of the most fundamental and far-reaching, but generally overlooked, forces operating to determine the nature of the systemic expressions of dental infections.

In a group of non-susceptibles consisting of 102 persons, which included all of those who did not have rheumatic group expressions (degenerative diseases) and never had had, either from dental or other origin, which were found in 681 cases selected from about 1400 because the records of their cases were considered sufficiently detailed and exact for scientific and comparable study, we find that this factor of immunity of the persons of this group consisting of 102 individuals, pertains in general also to all the other known members of those family circles; by which, we mean the immediate individuals in three generations: namely, the brothers and sisters of the individual, his or her father and mother and their brothers and sisters, and the grandparents of the individual.

We have termed the group of lesions produced by streptococcal infection the *Rheumatic Group* lesions, and have selected for our classification only such as were severe enough seriously to distress or incapacitate the patient. When we compare first the individuals in these three groups of absent susceptibility, acquired susceptibility, and inherited susceptibility, we find a very remarkable difference in their tendency to affections of the rheumatic group lesions. But even more striking and significant it is to find that this same quality tends to run as a constant law through the other members of their families; for, whereas the average number of severe rheumatic group lesions in the individual and the entire family as outlined is in the absent group only 4.2, in the acquired group it only increases to 6.8, but in the inherited group jumps to the remarkable figure of 15.05. And even more striking is the comparison of the average incidence of the severe and mild rheumatic group lesions which in the absent group is 7, acquired susceptibility group 10.7, and in the inherited susceptibility group jumps to 21.1.

I repeat this is one of the most important new truths that has been presented on this whole problem of the relationship existing between focal infections and the deficiency and degenerative diseases. Its significance cannot possibly be appreciated by simply reading it; for when the diagnostician thoroughly visualizes this great new truth, he will find himself playing the game of diagnosis with marked cards. He can pretty nearly make out either the patient's chart from the family record or *vice versa*. Immediately it will be realized that if the factor of danger is entirely different in these different groups, the factor of responsibility increases with that danger, and also that the individuals in the latter group must have an entirely different set of standards as to what shall be condemned or retained, from those with the much larger factor of safety. This will be developed in detail in the succeeding chapters.

The reader should have in mind in studying these results that the patients coming to a dental clinic such as this, include a much larger percentage of individuals with definite involvements than those found in an ordinary practice, since we have been specializing in dental diagnosis, dental pathology, and dental research. The percentages cannot, therefore, be considered as directly applicable to either the groups that would be made up in some other practice, or with the average people on the street.

We desire at this point to explain in a general way the method of making these examinations, and the detailed analysis of the method of diagnosis will be found in Chapter 59. On the presumption that there is more or less danger of exposure to rheumatic group lesions for every individual, differences in the prevalence of a break might furnish information that would help to point to the characteristics of individuals which have or have not a high defense, and by studying large groups we would be more likely to select those fundamental qualities which are characteristic and causative, or at least contributing factors. We have assumed that many individuals know whether or not they have had acute pain in their joints which has put them to bed, or have had a sense of being short of breath and exhausted from going upstairs, which their physician has told them was due to a condition of their heart, etc.; and that they could be depended upon in a general way to have information that would be of value for comparison, if not always exact. I can anticipate with relative certainty, based on past experience, that it will be argued that an individual will not know whether the parents and their brothers and sisters died of heart trouble or Bright's disease, or had rheu-

COMPARISONS OF

Susceptibility Class	Age	No. 100 M	No. 100 F	Patients' Lesions in % Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues	Brothers	Sisters	Fathers	Fathers' Relatives
Averages of Total No.	41.3	29	71	53	63	**26**	61	81	60	70	51	51	73	39
Groups of 15														
Absent	40.7	53	47	27	13	**0**	13	13	7	7	0	0	27	7
Acquired	47.9	40	60	40	47	**20**	53	87	53	53	27	7	13	0
Inherited 1 side mild	43.4	33	67	53	93	**13**	60	80	47	47	33	47	60	40
Inherited 2 sides mild	40.9	20	80	60	87	**40**	73	80	67	73	60	73	80	47
Inherited 1 side strong	39.4	27	73	47	93	**27**	80	100	67	87	33	40	67	40
Inherited 2 sides strong	33.9	7	93	80	73	**80**	93	100	93	87	80	73	100	100

FIGURE 42.

matism or neuritis; to which I will reply that we have excluded nearly half of the records taken because the patients did not know sufficient detail regarding these facts; and also, that if the cause of the parent's death was sufficiently impressed upon the minds of the patients that they could say clearly that it was from Bright's disease or heart, etc., they were generally correct, which we have determined by making a large number of verifications by communications with family physicians. I would also emphasize that since if the patient did not feel sure no entry was made, the effect on the record would be that the individual in question was clear and the total effect would be to make the case less strong than it should be rather than more strong than it should be. If then our percentages seem high with incomplete records, we are very sure that they would be much higher if we had complete records in all cases. That this is true, we have demonstrated to our satisfaction by establishing groups where we were able to get very complete records for all or most of the individuals involved, and in those cases, as will be shown presently, the percentages are very much higher.

We selected fifteen families in each of the following groups: non-susceptibility or absent, acquired susceptibility, inherited susceptibility one side mild, inherited susceptibility both sides

TIBILITY GROUPS

	Average No. of Lesions in Family								Local Expressions of Dental Infections					
	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues	Total Severe	Total Severe and Mild	Caries	Pyorrhea	Open	Locked	Rarefying	Condensing
3	2.48	1.35	1.01	5.37	3.12	1.94	11.34	16.35	75	23	25	75	32	26
7	.47	.13	.13	.47	.47	.13	**1.07**	**2.07**	40	40	40	60	67	0
)	.73	.27	.60	2.47	.93	1.00	**4.20**	**6.40**	80	33	33	87	33	20
)	3.00	.87	1.20	4.40	1.87	1.27	**9.60**	**13.40**	67	33	40	87	33	20
)	4.07	1.93	1.27	6.13	3.73	2.00	**15.30**	**20.53**	93	20	27	80	40	33
7	3.40	2.00	1.80	7.00	5.20	2.07	**17.20**	**22.53**	80	20	20	80	27	33
3	7.07	5.13	3.33	16.93	7.07	7.60	**32.20**	**50.27**	93	0	0	87	7	67

mild, inherited susceptibility one side strong, and inherited susceptibility both sides strong. The figures for these groups are shown in Figure 42, from which it will be noted in the last two columns under "Average No. of Lesions in Family" that the total severe rheumatic group lesions per family in the non-susceptibility or absent group is reduced to 1.07, and the severe and mild rheumatic group lesions to 2.07; the group with acquired susceptibility, severe lesions, 4.2 severe and mild lesions 6.4; inherited susceptibility, mild one side of ancestry, severe 9.6, severe and mild 13.4; inherited both sides mild, severe 15.3, severe and mild lesions 20.5; inherited one side strong, severe lesions 17.2, severe and mild lesions 22.53; inherited two sides strong, severe lesions 32.2, severe and mild lesions 50.27.

The above is an analysis of the ancestry on the premise of the patient's condition. When we reverse this and use the ancestry as the basis on which to judge the progeny, results are quite as striking, as shown in Figure 43; from which it will be seen that in eight families with an average of 7.3 children per family with no apparent susceptibility, the average number of children affected per family was .63; which means that during the entire lifetime up to the time of the record less than one child per family had shown at any time a rheumatic group lesion. The percentage

MENDELIAN FACTORS

Relative number of children affected in families when
(a) Patient has *absent* susceptibility.
(b) Patient has *acquired* susceptibility.
(c) Patient has *strongly inherited* susceptibility.

	No. of cases	Average No. of children per family	Average No. of children per family affected	Percent of children affected	
(a)	8	7.3	0.63	**9%**............	**Susceptibility absent**
(b)	8	7.2	1.2	**17%**............	**" acquired**
(c)	8	9.0	4.0	**44%**............	**" inherited**

FIGURE 43.

A PROGRESSIVE STUDY OF THE RELATION OF THE SUSCEPTIBILITY FACTOR OF INDIVIDUAL PATIENTS TO THAT OF THEIR RELATIVES.

Group	No. of Families in Group	Average Total Severe Rheumatic Lesions per Family	(Applied Mendelism) Average No. of Individuals Per Family Who Have Had Lesions In: Joints & Muscles (Rheumatism)	Nerve Tissues	Heart	Digestive Tract and Kidney	Percentage of Individuals with: Extensive Caries	Locked Dental Infection	Periodontoclasia
1 Dental Patients with *No* Developed Susceptibility	35	**3.7**	**1.1**	**1.0**	**0.5**	**1.0**	**51%**	**58%**	**23%**
2 Dental Patients with an Apparently *Acquired* Susceptibility	12	**4.7**	**1.3**	**1.3**	**0.6**	**1.6**	**91%**	**75%**	**33%**
	27	**7.7**	**2.2**	**2.0**	**1.2**	**2.2**	**78%**	**48%**	**26%**
3 Dental Patients with a Susceptibility and with one or both Parents acting as *Carriers Only*	16	**10.0**	**3.8**	**2.3**	**1.9**	**2.3**	**81%**	**44%**	**25%**
4 Dental Patients with a Susceptibility and with only *One Side of Ancestry*, including the Parent Involved	8	**13.6**	5.2	3.4	3.3	2.1	**88%**	**75%**	**0**
5 Dental Patients with a Susceptibility and with *Both Sides of Ancestry*, including Both Parents Involved	7	**37**	12.4	8.0	9.0	7.0	**100%**	**86%**	**0**

FIGURE 44.

of all the children affected in this group was 9. In the second series of eight families with an average number of children per family of 7.2, where there was what we have termed an acquired susceptibility, the average number of children affected per family was 1.2, or a total for this group of 17%. In a third group of eight families with an average number of children per family of 9, the average number of children affected per family was 4, and the percentage of affected children in this group jumps to 44.

Realizing how critical one must be of his findings when searching for fundamental new truths because of the danger of seeing the thing one is looking for, I have made or have had made several separate and independent studies for the purpose of checking one against the other. The individual compiling the last group of 681 selected cases not only did not know the content of the previous compilation, but did not even know that one had been made. She was kept in ignorance of this fact for her own protection, and it is exceedingly important to find that in several particulars those records analyzed in 1919 furnish totals which are strikingly similar to those recently completed. (1922)

Figure 44 shows an analysis of five different groups slightly differently selected as follows: Group No. 1, Dental patients with no developed rheumatic group susceptibility; Group No. 2, Dental patients with an apparently acquired susceptibility; Group No. 3, Dental patients with a susceptibility, and with one or both parents acting as carriers only (parents' ancestry involved, but parents not yet); Group No. 4, Dental patients with susceptibility with only one side of the ancestry, including that parent, involved; Group No. 5, Dental patients with a susceptibility, and with both sides of the ancestry, including both parents, involved.

Among the many similarities, note that in the first group of 35 non-susceptibles the average total severe rheumatic group lesions per family is 3.7; whereas, in the recent analysis of 102 non-susceptibles, this is shown to be 4.2; (See Figure 41) and in Figure 42, the 15 strongly absent cases, 1.07. [See column "Total Severe" under "Average No. of Lesions in Family."] The corresponding groups of acquired susceptibles in these three studies show in the figures of 1919 (Figure 44) 4.7 severe lesions and in Figure 42, 4.2 severe. Groups 3 and 4 of Figure 44 are not exactly comparable to groups 3, 4, and 5 of Figure 42, though quite similar in classification of individuals. Group 3, Figure 44,

A STUDY OF SUSCEPTIBILITY OF VARIOUS GROUPS OF PATIENTS WITH RHEUMATIC LESIONS WHICH ARE APPARENTLY RELATED TO DENTAL FOCAL INFECTIONS

Group	No. of Families in Group	Age Range	Average Age	Average Total Severe Rheumatic Lesions per Family	Percentage of Patients in Group Who Have Had Lesions In: Joints & Muscles (Rheumatism)	Nerve Tissues	Heart	Digestive Tract and Kidney	Average No. of Individuals Per Family Who Have Had Lesions In: Joints & Muscles (Rheumatism)	Nerve Tissues	Heart	Digestive Tract and Kidney
1 Dental Patients with *No* Developed Susceptibility	35	19 to 85	46	**3.7**	0	0	**0**	0	**1.1**	**1.0**	**0.5**	**1.0**
2 Dental Patients with *Rheumatism* Most Dominant in Patient and Family	14	17 to 55	40	**14.8**	100%	95%	36%	79%	**6.9**	**2.8**	**2.0**	**3.3**
3 Dental Patients with *Nerve Lesions* Most Dominant in Patient and Family	3	31 to 42	35	**16.0**	67%	100%	33%	100%	**4.3**	**6.0**	**3.3**	**3.0**
4 Dental Patients with Lesions in *Digestive Tract and Kidney* Most Dominant in Patient and Family	8	12 to 56	40	**17.4**	50%	88%	50%	100%	**4.1**	**2.9**	**3.3**	**7.3**
5 Dental Patients with Lesions of *Rheumatism and Nerves* Most Dominant in Patient and Family	9	26 to 63	44	**18.3**	100%	100%	44%	78%	**6.4**	**5.7**	**2.6**	**3.7**
6 *Appendix Infections* in Children and Grandchildren where Lesion was Dominant in One Side of Ancestry	3	12 to 48	31	**22.0** (**23.3**)x	67%	67%	67%	100%	**6.0** (**8.3**	**4.0** **4.0**	**3.3** **3.3**	**8.7** **9.7**)x
7 Dental Patients with Lesions of Rheumatism and Heart Most Dominant in Patient and Family	14	18	42	**24.0**	100%	93%	**100%**	71%	**7.4**	**4.4**	**6.9**	**3.5**
8 Dental Patients with *Heart Lesions* Dominant in Patient and Family	7	30	43	**28.2**	71%	86%	**100%**	86%	**7.0**	**6.1**	**9.7**	**5.0**
9 Dental Patients with *Rheumatism, Nerves, Digestive Tract and Kidney* Most Dominant in Patient and Family	6	21	35	**27.8**	100%	100%	50%	100%	**9.2**	**7.7**	**3.7**	**7.7**

x—Children included.

FIGURE 45.

has severe rheumatic group lesions 10 per family, and Group 3, Figure 42, has 9.6; Group 4, Figure 44, 13.6, Groups 4 and 5, Figure 42, 15.3 and 17.2 respectively; Figure 44, Group 5, strong inherited susceptibility from both sides, 37, and Figure 42, Group 6, similar strong inheritance, 32.2.

Similarly, Figure 45 made in 1919, in which the dominance in its relation to the particular type of tissue is involved, shows progressively percentages beginning with 3.7 for non-susceptibility and increasing to 27.8 for susceptibility to lesions in several types of tissues. The average total severe rheumatic group lesions per family for the group with absent susceptibility, marked "No Susceptibility," is 3.7; those with rheumatism the most dominant in the patient and family, 14.8; nerve lesions most dominant in patient and family, 16; digestive tract and kidney, 17.4; a combination of muscle and nerve involvements as rheumatism and neuritis, 18; appendix infections where lesion was dominant in one side of the ancestry, 22; where rheumatism and heart affections were both expressed in the family, 24; heart affections alone dominant in the family 28.2; and where a combination of affections as rheumatism, nerves, digestive tract, and kidney, were all dominant in the family, 27.8.

A further comparison of data developed in the two series of studies is of value. At that time one hundred consecutive families were studied (See Figure 46) and the ratios of lesions in various types of tissue recorded for comparison. The recent analysis (1922) of seven hundred cases (See Figure 41) selected from dental practice (as distinguished from individuals studied under other conditions to be discussed later), when compared with the former determinations of 1919, reveals the following remarkable similarity of figures: Joints and muscles, former, Figure 45, 65%, recent, Figure 41, 63%; Nerves, former 70%, recent 81% (we are recognizing more nerve lesions than formerly with increased information); Heart, former 28%, recent 26%; Internal Organs, former 61%, recent 60%; Tonsils, former 62%, recent 53%.

A comparison of the severity and frequency of lesions in the two sexes, when compared in the two sheets of studies, again repeats an important similarity. In the former studies of one hundred patients with severe lesions, 75% were females and 25% were males; and in the recent study of 681 cases, 71% were females and 29% were males. In this connection, it is interesting

COMPARISONS OF TWO SEPARATE STUDIES						
		Relative Prevalence of the Following Lesions in Affected Patients				
Summary Made In	No. of Affected Patients	Joints and Muscles (Rheumatism)	Nerves	Heart	Internal Organs	Tonsils
1919	100	65%	70%	28%	61%	62%
1922	681	63%	81%	26%	60%	53%

FIGURE 46.

DOMINANCE OF SPECIAL TISSUE LESION IN BOTH PATIENTS AND FAMILIES (TEN)

Group	No. of Males	No. of Females	No. of Lesions in Ten Patients							No. of Lesions in Families									Local Expressions of Dental Infections					
																	Total							
			Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues	Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues	Severe	Severe & Mild	Caries	Periodontoclasia	Open	Locked	Rarefying	Condensing
*Rheumatism	2	8	5	**10**	2	6	4	4	3	8	**59**	7	9	19	19	10	104	131	9	1	1	6	2	3
Heart	3	7	7	6	**10**	5	7	3	8	12	24	**57**	6	25	13	19	121	156	10	1	2	8	3	5
Nerves	2	8	5	6	2	7	**10**	7	7	10	15	9	10	**142**	28	19	180	233	9	3	3	9	5	5
Internal Organs	2	8	6	4	0	6	9	**10**	6	6	13	9	10	30	**90**	12	136	170	7	3	3	7	4	3

* Type of susceptibility—inherited.

FIGURE 47.

MENDELIAN FACTORS									
Cases	Average No. of children per family	Average No. of children affected	Fathers affected	Fathers' Relatives affected	Mothers affected	Mothers' Relatives affected	Dominance in ancestry	Dominance in children	Mendelism
42	5	2.7	76%	61%	50%	55%	61%	54%	88%

FIGURE 48.

to note that in the progressive classification of cases from absence of susceptibility through mild to severe, the ratio of males to females keeps changing in a definite and geometric proportion; whereas, in the group Figure 42 of absent susceptibility, the ratio of males to females in the cases selected at random was 53% males to 47% females; acquired susceptibility, 40% males and 60% females; inherited susceptibility, mild one side only, 33% males and 67% females; inherited strong one side, 27% males and 73% females; inherited two sides mild, 20% males and 80% females; inherited two sides strong, 7% males and 93% females. This last group represents persons with very severe lesions. (This tells a tragic story which is discussed later in the chapter on Overloads and is largely a record of the terrific charge that Nature has made against motherhood.)

An analysis of these data throws a very important new light on the nature of an inherited susceptibility, which is augmented by careful application of these observations in clinical practice. We have thought of susceptibility in terms of a systemic defense. After we make an analysis, for example, of hearts in 681 selected cases, consecutive except that there have been eliminated all cases in which we could not secure a sufficiently complete record, in these we find that 26% of the patients, approximately 1 in 4, have some heart disturbance. This in itself is a tragic revelation, especially when we contemplate that 1 in 10 of the deaths of all ages in modern civilized communities is from heart affection. An examination of the heart column in Figure 42 shows a progressive column of ratios of patients with heart involvements; from 0 in Group 1, those without streptococcal susceptibility, 20% in the acquired group, 13% inherited one side mild, 27% inherited one side strong, 40% inherited two sides mild, to 80% inherited two sides strong. Comparing this with the data in Figure 45 under the column "Percentage of patients in group who have had lesions in various tissues," we find that the percentage starting in Group 1 as 0, advances progressively so that in the group in which the heart was dominant in the family, in seven families which were studied at that time, and many more of which have been added, the ratio is 100%. While these were unusually severe instances of susceptibility to heart lesion, it is most striking to see how frequently the susceptibility to heart is dominant throughout the family with complete absence of other rheumatic group lesions; and, similarly, kidney, muscles and joints, digestive tract, and special tissues, each dominant in the particular family group while all other tissues remain very low or completely absent. This is strikingly revealed in a section of Figure 42 entitled "Average No. of Lesions in Family" and in Figures 43 and 44, and in the totals of Figure 41. It is, however, true, as revealed in all of the charts, that the heart or some other tissue may be dominant by inheritance and there may be an acquired susceptibility for some other tissue, which other tissue is in the majority of cases the nervous system.

Contrary to our expectations, we do not find that the inheritance of a susceptibility to rheumatism necessarily carried with it a susceptibility to heart or *vice versa*. This is strikingly illustrated in Figure 47 which shows many instances of heart's being dominant in the family and no rheumatism; and likewise, rheumatism dominant in the family and no cases of heart. In a study of the relation of other infections, such as syphilis, to heart involvements we show that this is an important factor as a source of heart lesions and that these individuals frequently, if not generally, have distinctive characteristics aside from their history and Wasserman reactions. The data in the chart in Figure 47 is exceedingly important because it shows that in forty selected families where the chief lesion of the patients studied was one of the four following—rheumatism, heart, nerves, or internal organs—in every instance that lesion by far outnumbered all others in the individuals constituting the patients' relatives, as, for example, in ten patients affected the number of instances of rheumatism in other members of the family was 59, whereas no other lesion of the rheumatic group exceeded 19. Where heart was the chief lesion of the patient there were 57 cases of heart in the immediate family and no other lesion exceeded 25. Where nerve lesions were the chief lesions in the patient there were 142 instances of severe nerve lesions in the family, whereas no other lesion exceeded 28; and where the lesion of the patient was in the internal organs there were 90 instances of lesions in the internal organs of other members of the family, whereas no other tissue exceeded 30.

When we take the total number of heart cases in 681 family groups, we find it to be 916 hearts, or an average of 1.35 per family. An actual count of the families having at least one case of heart reveals that of the 681 families, only 413 have any heart lesions, an average of 2.2 per family; and of these, 100 families had over half of the lesions. An analysis of the families included in Figure 42 shows that in each of the six groups of fifteen families each, there is not an instance where the patient being studied had developed a heart lesion, where there was not a record of a heart lesion in the brothers or sisters, or fathers' and mothers' families; and in practically all instances where the number of hearts per family group is greater than the average for the entire group, the patient is recorded as having a heart lesion, and the severity of the heart lesion is in striking proportion to the dominance of the lesion in the family. The groups of fifteen in Figure 42 show more striking conditions than the average. Similarly and quite as strikingly, we might study kidney, joint tissues, and other tissues, though not necessarily the nerve tissues which apparently may have a susceptibility by inheritance or be an acquired factor.

An analysis of the data has been made to see the evidence of mendelism. This is shown in Figure 48. In 42 cases of marked susceptibility, the average number of children per family was 5. The average number of children affected was 2.7, which you will note is 54%. In these 42 families, 61% of the two ancestral families had been afflicted with severe rheumatic group lesions. The dominance, therefore, will be seen to be 88% of that in the preceding generations, which we may take as a factor of mendelism.

It will be of interest to note the relation of inherited susceptibility to both the mothers' and the fathers' sides of the ancestry. In the patients, 73% showed inheritance of the chief severe lesion, and 27% an acquired susceptibility. Of the inherited, 44% were singly, that is from one ancestral branch; only 29% from both sides of the ancestry. Of the singly inherited, 23% showed inheritance through the maternal side, and 21% through the paternal side.

Where the susceptibility was apparently an inheritance quality, we found that in involvements of joints and muscles, there were 4 males to 5 females; nerve tissues, 2 males to 3 females; heart, 3 males to 4 females; digestive tract and kidney, 4 males to 5 females; nervous system, 3 males to 5 females; making a general average of 3 males to 4 females. One of the most striking results of this study has been to find that when an *acquired susceptibility* obtains—in other words, where the individual with normally an ample defense has been overloaded—in the great majority of individuals the break came in the nervous system. Overloaded tissues in general tend to break and become a prey to streptococcal infection or toxic irritation.

Reading downward in most of the columns of the various charts you will see evidences of a progression, some of which are very striking. We shall later discuss in Chapter 21 the forces which tend to make tissues susceptible, which are not limited to inheritance but have to do with injury and overload of various types. This extensive study of the characteristics and relationships between systemic involvements and individual susceptibilities has brought out many other very striking new facts, such as the type of the tissue reaction about apical involvements in relation to the systemic susceptibilities, the relationships of susceptibilities to dental caries and periodontoclasia (pyorrhea alveolaris), which have been made the subjects of special researches and will be discussed in this order in the three succeeding chapters.

SUMMARY AND INTERPRETATION.

To summarize:

(1) The evidence that we have secured to date on the problem of the variations in susceptibility, while not sufficient for a final statement, is ample to suggest what will be a much safer interpretation until further data are available. To recapitulate briefly, individuals instead of being similar and therefore comparable in their susceptibilities to infection, divide themselves into three groups: Non-susceptibles, acquired susceptibles, and inherited susceptibles.

(2) The tendency of an individual to develop both general and special systemic involvements has a direct relation with, and proportion to, the susceptibility of the various members of that family circle.

We would therefore restate the fourth fundamental as follows:

Individuals as a whole do not react sufficiently similarly to justify the premise that they could all be judged by the same standards and therefore be considered comparable; that individuals can be classified into groups, the members of which are sufficiently similar to be judged by the same general standards, and they may therefore be considered comparable.

CHAPTER V.

RELATIONSHIPS BETWEEN LOCAL AND SYSTEMIC REACTIONS.

PROBLEM: Is it true, as generally presumed, that there are no distinguishing characteristics which relate the type of local periapical reaction to the nature and extent of systemic reaction?

INTRODUCTION.

Problem No. 3 was a study of the local characteristics of the periapical lesions as they develop in various individuals, and revealed (a) that infection may or may not produce extensive absorption in bone, and (b) that a given or similar dental infection will not necessarily express itself in the local tissues of the mouth approximately the same in all people; that these expressions tend to be of three different types which we have as a grouping for individuals, and that the individuals of this group therefore are comparable in this regard. The differentiating characteristic of these three groups was that the first had very extensive absorption of alveolar bone about the apex of an infected tooth, a marked rarefying osteitis; the second was similar except that it tended to have a zone of condensing osteitis surrounding the zone of rarefying osteitis; and the third with a similar quantity of infection had a relatively limited area of rarefying osteitis frequently surrounded by a condensing osteitis.

Problem No. 4, Chapter 4, was an analysis of the presumption that all individuals are comparable. The research data strongly indicated that they are not; that while there is a variation through a wide range, that variation still permits of individuals' being roughly grouped into three general classes with regard to the presence or absence of systemic lesions of the types which we have classified as the rheumatic group affections frequently, if not generally, produced by streptococcal invasion.

This research, which we have designated as Problem No. 5, is a study to determine whether or not there is a relationship between the groupings disclosed in the researches of these last two chapters. This problem may then be stated briefly as "The Relationships between Local and Systemic Reactions".

EXPERIMENTAL AND DISCUSSION.

Since, according to the presumption, all individuals are similar, and since dental infections are entirely dependent for their characteristics upon the type of organism which has chanced to secure access, therefore there are no characteristics of the local tissue pathology which are related to the degree of susceptibility or nature of systemic involvement. Our problem more specifically stated is "What relationship, if any, exists between the forces which make individuals group with regard to their local tissue reactions about infected teeth, to those forces which make them group with regard to their systemic involvements into absent susceptibility, acquired susceptibility, and inherited susceptibility."

In order to determine this we have made a very careful study of the local tissue reactions found about the teeth in the 1400 cases from which the 681 cases have been selected as being sufficiently complete in detail to be worthy of comparison. Our charts have been provided with spaces for recording in detail the type of dental pathology found in the various cases. In Figure 49 (also shown in Figure 42 of Chapter 4) there is a progressive increase in the dominance of rheumatic group lesions with the dominance of the inheritance, heart lesions, for example increasing from 0 to 80 per cent, and lesions of other tissues increasing in about the same proportion. The last division shows under "Local Expressions of Dental Infections" that open dental infections, by which we mean those that have a communication with the mouth cavity, were found present in 40 per cent of the individuals of the absent group, 33 per cent of the acquired group, 40 per cent inherited one side mild, 27 per cent inherited two sides mild, 20 per cent inherited one side strong, and 0 inherited two sides strong. Locked infections increase through these groups from 60 per cent to 87 per cent. Rarefying osteitis decreases from 67 per cent in the class of absent susceptibility to 7 per cent in the inherited susceptibility two sides strong. Condensing osteitis is present in 0 per cent of cases of the class of absent susceptibility and increases progressively to 67 per cent inherited two sides strong. It is significant that the individuals making these figures had not the slightest conception of what they were for or what information was going to be developed.

The significance of this important new truth cannot be realized suddenly. It is only by seeing patient after patient of these

RELATION OF LOCAL STRUCTURAL CHANGES TO SYSTEMIC SUSCEPTIBILITY

Susceptibility Class	Age	M	F	Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues	Open	Locked	Rarefying	Condensing
Groups of 15														
Absent	40.7	53	47	27	13	0	13	13	7	7	40	60	67	0
Acquired	47.9	40	60	40	47	20	53	87	53	53	33	87	33	20
Inherited 1 side mild	43.4	33	67	53	93	13	60	80	47	47	40	87	33	20
Inherited 2 sides mild	40.9	20	80	60	87	40	73	80	67	73	27	80	40	33
Inherited 1 side strong	39.4	27	73	47	93	27	80	100	67	87	20	80	27	33
Inherited 2 sides strong	33.9	7	93	80	73	80	93	100	93	87	0	87	7	67

FIGURE 49.

various classes and observing how constantly the local reaction about the teeth corresponds with phases of the systemic susceptibility to rheumatic group lesions that one visualizes its full significance. When we compare these data with the charts shown in Figures 38, 39, and 40, of Chapter No. 3, we find that the individuals whose cases were found to be typical of the type of structural change which we have represented in Figure 38, all belong to the class which we have referred to as having absent susceptibility. The structural change in alveolar bone about infected teeth of this group is that of an extensive alveolar absorption as a rarefying osteitis diffusing into the medullary spaces without condensing osteitis. The group represented in Figure 39 corresponds and is, we find, the same group of individuals that make up our lists, when studied from the standpoint of systemic involvement, of those of acquired susceptibility. They have evidence of an extensive rarefying osteitis surrounded by a zone of condensing osteitis, sometimes thin, sometimes of considerable depth. And, similarly, the group which we have expressed in Figure 40 as having a very limited zone of rarefying osteitis in comparison with the quantity of infection, surrounded by a blurring of the medullary spaces or with a condensing osteitis diffusing into a general bone condensation, corresponds with the group of strongly inherited susceptibility to rheumatic group lesions.

To be more specific, Figure 50 shows the condition in the mouth (as revealed by the roentgenograms of two individuals from each of the following groups: A and B, absent susceptibility; C and D, acquired susceptibility; E and F, mildly inherited susceptibility; G and H, strongly inherited susceptibility;) to be as follows: There is very extensive alveolar destruction about infected roots, particularly the first molar. This includes also a marked tendency to root absorption, with the result that the mesial root of the molar is practically obliterated. There was a fistula over each one of these three teeth, with exudate oozing on compression. This man, fifty-two years of age, was physically in excellent condition with but one exception: limitation of the use of his eyes in reading, not sufficient to produce lesions but requiring glasses. After the removal of his dental infections which were extensive, he found he could read without limitation and that without his glasses. Not only was his system excellent and free from rheumatic group lesions notwithstanding this abundant infection, but he had seven brothers no one of whom had ever suffered from any of the rheumatic group lesions, and three sisters none of whom had suffered from any of the rheumatic group lesions. A study of his father and the father's brothers and sisters, and the mother and her brothers and sisters, reveals but one mild instance of a suspected rheumatic group lesion on either side of the ancestry, including the grandparents on both sides. This is clearly a case of absent susceptibility.

In Figure 50-B showing extensive alveolar absorption about the apices of the roots of a molar and bicuspid, we have a condition in the mouth of a man of twenty-five years of age. Note the extensive absorption of bone and tendency to absorption of root ends. Physically, this man has scarcely known a limitation. Material aspirated from an apical area showed many giant cells, which particular cell we have only found in cases of very high defense where there is absorption of root apices. His physical classification is also absent susceptibility. His family history is as follows: He has not had during his lifetime a single break of the rheumatic group lesions. His brother and four sisters have also been entirely free. His father is fifty years of age and has not had a symptom of any of the rheumatic group lesions, nor have any of the father's three brothers or two sisters, his father's

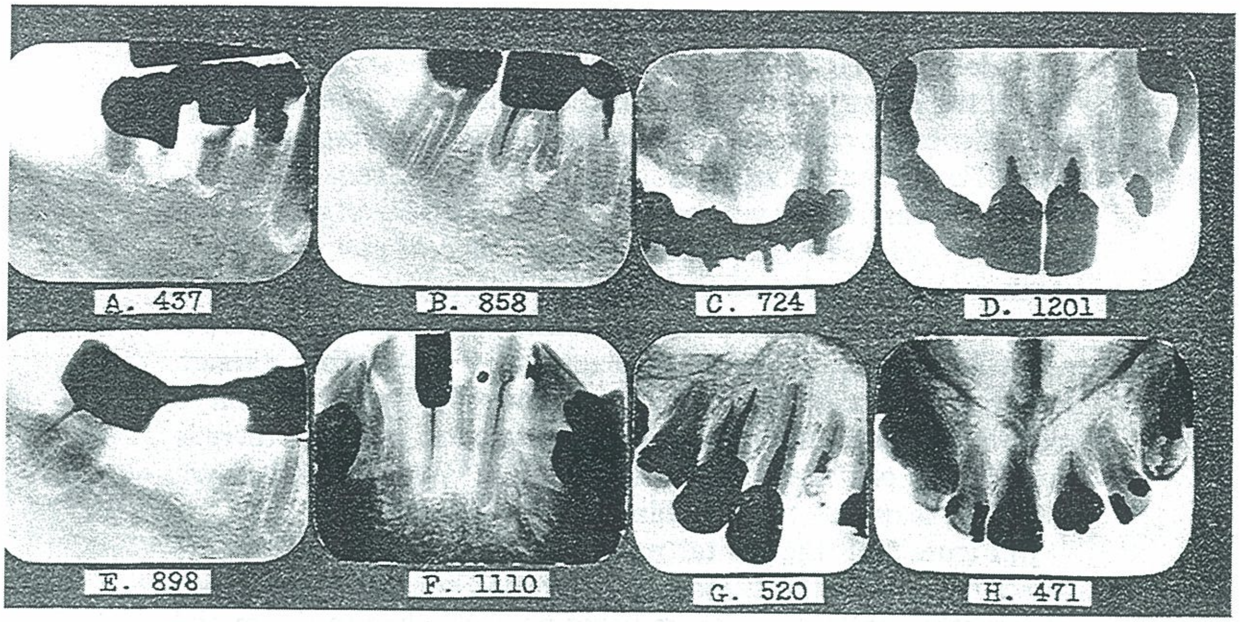


FIGURE 50. TYPICAL ILLUSTRATIONS OF THE LOCAL REACTIONS IN THE DIFFERENT GROUPS.

father and mother living to ninety years of age each. His mother at fifty-two years of age has had hardening of the arteries and her father had some rheumatism. Otherwise the record is perfectly clear on the mother's side. The mother's mother is living and very strong at seventy-five.

In Figure 50-C we have a patient with very extensive absorption of alveolar bone but with a zone of condensed bone surrounding the immediate zone of rarefaction at the apex and about the bicuspid root. This patient has been free from rheumatic group lesions during his lifetime until recently when he has developed neuritis. He is sixty-seven years of age, has had five brothers and six sisters, one of the latter being ninety years of age. His father died of typhoid at fifty-eight and his father's father and mother both lived to ninety. His mother died at ninety-two and her mother at ninety-two. There has not been a single case of rheumatic group lesion on either side of the ancestry. This man's classification is clearly one of an acquired susceptibility.

In Figure 50-D we see the local dental expression to be one of quite extensive alveolar absorption above the right central which shows a record of condensing osteitis surrounding the rarefying. Physically, the patient presented with pain of recent development in his right shoulder and right knee. He has had three brothers and three sisters. One of the former has had a heart involvement.

Otherwise the brothers and sisters have been free from rheumatic group lesions. His father died at sixty of a stroke and the father's side of the ancestry with three brothers and three sisters was free from rheumatic group lesions, his father's mother dying at eighty-five. The patient's mother is living at seventy-seven, is in excellent health, and has never been ill except one attack of pneumonia and some asthma. His mother's father died at eighty and her mother at eighty-nine, and these with her four brothers and six sisters were entirely free from rheumatic group lesions. There is a possibility that the heart involvement of his brother, though not accompanied by rheumatism, may have had some relation to his own rheumatic development, but we assume not since there is no evidence of it elsewhere in the family. We would therefore classify this as one of an acquired susceptibility.

In Figure 50-E the dental pathology as revealed in this case shows much less absorption of the alveolar bone surrounding an apparently unfilled root, there being no root filling in the mesial canal of the third molar and not as extensive absorption about the roots of the first molar as we should expect. A study of this patient's susceptibility reveals that he has had mild attacks of rheumatism and some slight evidence of neuritis. He has one sister. She has had mild rheumatism. His father died at seventy of anemia and has a history of mild rheumatism. His father's father died at seventy-six of kidney and heart involvement, probably related to the rheumatic susceptibility; his father's mother at eighty-six with a definite history of rheumatism. His mother is living at seventy-six with a definite history of rheumatism and gall-stones. The mother's mother had heart and kidney involvement and the mother's sister very severe rheumatism and heart involvement. There has therefore been a very definite tendency to rheumatism in this family; and while it has been on both sides, neither of the parents has suffered severely. We would therefore record it as a mildly inherited susceptibility.

Figure 50-F shows three lower incisors, one of which has a putrescent pulp without root filling, yet neither it nor the adjoining teeth show extensive absorption. This patient is fifty-five years of age and presents with mild neuritis and rheumatism of recent development in his hands. Otherwise he has been entirely clear during his lifetime. His two brothers and a sister have not had disturbances. His father died at seventy-nine, having had severe attacks of rheumatism and neuritis. His father's father

died at seventy-four. The patient's mother died of pneumonia at thirty-seven, the mother's father at sixty-six, the mother's mother at eighty-four, all without symptoms of rheumatic group lesions. Since there is a definite history of rheumatism and neuritis with the father, not in other members of the ancestry and not previously in the patient, we would classify this as a case of mildly inherited susceptibility.

Figure 50-G shows a lateral of a girl only twenty-three years of age. There has been a moderate absorption but no fistula and no local pain. She presents with quite severe rheumatism and severe lassitude. Even at the age of twenty-three she has had quite severe rheumatism, neuritis, nervous breakdown, and digestive disturbance. She has two brothers and four sisters. Each one of the six brothers and sisters has had rheumatism and each, like herself, has been operated for tonsils. Her brothers and sisters have also all had acute neuritis. Her father died at fifty-six, having had acute rheumatism and neuritis and acute digestive disturbance. His father died at eighty-one, having been a sufferer from rheumatism and neuritis. The father's mother had also suffered from rheumatism, neuritis, and digestive disturbance, as did also the father's brothers, three of whom and the patient had kidney involvement. The patient's mother is living at sixty-one but has suffered severely during her life from rheumatism, neuritis, and digestive disturbance. Her mother died at seventy, having suffered from these severely; and the mother's mother died at thirty-nine of acute rheumatism. The mother's brothers and sisters also suffered from rheumatism. We have, then, a family in which there have been sixteen cases of rheumatism, eight cases of acute digestive disturbance, and twelve cases of neuritis. This girl is breaking at twenty-three, not because of severe overload but in spite of the absence of severe overload. Of her two brothers and four sisters ranging in age from twenty-one to forty, everyone has similarly broken. Her father and mother began breaking seriously at about forty years of age and scarcely were free from disturbance thereafter. This girl by inheritance has an exceedingly marked susceptibility to not one but three at least of the severe rheumatic group lesions, involving muscles and joints, nerve tissues, and digestive tract tissues. We therefore record her case as one of strongly inherited susceptibility.

The dental pathology as shown in Figure 50-H is very misleading. There is very little evidence indeed of severe involvement of the root filled centrals; yet on extraction the root apices were badly discolored and therefore showed marked involvement though there was very little destruction of alveolar bone. She has presented with a very severe heart involvement and severe nervous breakdown. Her life was being despaired of. She had been troubled during that time a great deal with neuritis, nerve lesions, and heart involvement. Her age is forty-two. Her goiter has developed since the birth of her two children. She has had symptoms of rheumatism. She has had four brothers and four sisters, one brother and two sisters having had rheumatism. All four of her sisters have had severe nervous breakdowns as has also one brother. One sister has a severe heart lesion. Her father died at seventy-five of rheumatism and heart involvement. Her father's father died at forty, and three of her father's brothers and her father's sister all died of heart involvement. Her mother died of heart involvement at seventy-eight, having suffered severely from nervous breakdown and rheumatism. This patient has shown a marked susceptibility to these lesions during her lifetime, as have also her brothers and sisters and both sides of the ancestry. We would therefore classify her case as one of strongly inherited susceptibility.

If, now, we will review these four groups, we find the following: The two individuals of the first group (A and B) have never yet had any of the rheumatic group lesions notwithstanding various overloads, nor did the members of their families or their ancestries. They therefore classify readily as having an absent susceptibility. C and D had a history similar, in general, so far as they themselves and their ancestry were concerned except that they have lately broken. With each there has been a distinct physical overload of overwork. The development of their rheumatic group lesions has been a recent, though quite severe, disturbance. With the removal of the dental infections without a change in the overload of business cares, their conditions have cleared up completely and promptly. E and F are two patients who have developed acute rheumatic group lesions as rheumatism, which disturbances were definitely present in the ancestry. They are therefore classified as mildly inherited susceptibility. With removal of their dental infections they both had complete relief from their rheumatic disturbances notwithstanding they have maintained very busy business careers, which, no doubt, had contributed to their breaking. G and H are two cases of severe break with rheumatic group lesions that have tended to develop for some time, which lesions are very strongly present in the other members of the family and ancestry. They are accordingly classified as strongly inherited susceptibility. Each has shown very marked improvement with removal of the dental infections. The latter has taken up her home duties again as a mother and the management of a large home and is enjoying excellent health.

The question of prognosis in these various cases will be discussed in detail later. It will, however, readily be seen that the prognosis becomes less and less favorable for complete relief and freedom as the condition gets to be more strongly an inherited quality. This matter is discussed in detail in other chapters.

If, then, we will take a single tooth, let us say a lateral with all the infection of a putrescent pulp, and follow it through these different groups, we will see in detail the difference in the local structural pathology as well as in the physical systemic reactions. This we have done in Chapter 12 while discussing the relation of the quantity of absorption to the danger.

It is exceedingly significant that when we picked out a group of individuals expressing each of the different classes from the standpoint of a structural change about the teeth, and again made groups typically characteristic, and strongly so, of the different groups or classes on the basis of their systemic susceptibility, we were amazed to find that out of the hundreds of cases from which we had selected these few typical illustrations we not only had similar individuals but in a very many cases we had the identical individuals that had been selected to illustrate a group on the other basis of classification.

This one important new truth accounts for a very large part of the confusion amounting almost to bitter antagonism, that has existed in the medical and dental professions regarding whether or not dental infections are a serious factor in systemic involvements and degenerative diseases. It has been insisted by the group on one hand, on the basis of effect being in proportion to cause, that since the enormously large number, if not large percentage, of individuals presenting in the outpatient clinics of hospitals with the most abundant infections that could be found in any group, as evidenced by the quantity of pus flowing from broken down and decayed teeth, do not have any systemic involvements, therefore, it cannot be true that dental infections are an important causative factor in the systemic involvements. If any individuals would be affected, surely this group would be and they are not.

It now is demonstrated that the physical change about the root of the tooth is not a measure of the infection but a measure of the quantity and type of the reaction, not a factor primarily relating to the invading organism which is inside the infected tooth but a factor primarily relating to the activity of the defense of the individual; and when seen in this light, this whole confusion ceases to be a paradox and is completely intelligible. The individuals having the extensive rarefactions have an active mechanism of defense, which for them establishes an adequate quarantine immediately about the tooth. So long as that quarantine is maintained there is an extensive zone of rarefaction with little tendency to condensation of bone about this rarefying area. The vascularization is excellent. This also explains why these individuals without systemic rheumatic expressions tend continually to have alveolar fistulae. These are the individuals with absent susceptibility; but when their defense is broken from the many causes that will be shown in subsequent chapters, they take on a condition which we have termed an acquired susceptibility. Incidentally, their local defense goes down; the quarantine is not maintained; a changed reaction takes place about the tooth with the change in local defense; there is a condensing osteitis surrounding the rarefying osteitis; the quarantine not being maintained about the tooth, the warfare must take place in other parts of the body. These individuals probably now for the first time have the organism passing extensively from their dental infections through their bodies, and in a sense it is the first time the tissues of their bodies have been exposed to this infection. After the overloads are removed they will tend strongly to come back to their normal which is high; but in the third group with the inherited low defense there always has been a struggle, there never has been a normal local quarantine, teeth never have had fistulae because there was never an ample local reaction the by-product of which would have been a fluid which must have an exit, spoken of as pus from a fistula; the warfare has always had to be carried on in the various tissues of the body rather than in the specially designed and developed tissue which Nature establishes for the purpose of maintaining the quarantine about the root apex.

This group is always in danger. Their type of local reaction about their teeth has been what it has because of a lack of the capacity for a defensive reaction.

SUMMARY AND CONCLUSIONS.

The researches of this chapter have disclosed that individuals with a high systemic defense against rheumatic group lesions have also a very extensive zone of absorption about infected teeth; that with an overload these individuals tend to lose that high defense and develop the rheumatic group lesions, in which state they have less extensive absorption and a tendency to condensing osteitis surrounding the already existing rarefying osteitis; or if a new dental infection develops at and during this period, there is much less absorption of alveolar bone than that which occurred prior to the development of this acquired susceptibility; that the individuals of this group tend to come rapidly back to their high normal defense with the removal of their dental infections; that in most instances there has been a combination of dental infections plus overload. We assume that other focal infections have precisely the same relationships to their overloads.

In contrast to these two groups, all the members of which tended to belong to the absent susceptibility during the period of normal high resistance, we have found another group which we have subdivided into various degrees of inherited susceptibility, which susceptibility has been largely in proportion to the severity and dominance of the same expression in other members of the family and ancestry. The individuals of this group have had very much less alveolar absorption for a given dental infection, usually without a fistula. The teeth did not tend to become painful or tender as in the preceding groups. While there was relief from the acute systemic symptoms produced by the removal of the dental infections, there was a very marked tendency to recurrence. This group was termed those with inherited susceptibility ranging from mild to severe.

The prognosis was shown to be progressively more favorable with the absence of the inherited susceptibility factor. This new interpretation satisfactorily accounts for the clinical conditions and histories of the various types of individuals that are found in hospital clinics and private practice; and as the local defense is high, the absence of the systemic involvements prevails; and as the local defense is low, the prevalence of systemic involvements prevails. Therefore the individuals with the extensive areas of absorption with fistulae should be expected to be distinctly more safe than those with less effective local reaction, thus removing the paradox that has more than any other, or perhaps all other factors, blinded the professions to the role of dental infections in degenerative diseases.

We would therefore restate the fundamental which expresses the relation of local to systemic disturbances as follows:

> **Local dental pathology about an infected tooth has variations which make grouping and classification easily possible on this basis, which groups have a direct relationship with similar groupings that can be made on the basis of susceptibility to rheumatic group lesions. The local and systemic expressions are not only related, but are both symptoms of the same controlling forces and conditions.**

CHAPTER VI.

ARE APICAL ABSORPTION AND TOOTH INFECTION SYNONYMOUS?

PROBLEMS: Is it essentially true (a) *that a tooth without visible absorption at its apex is not infected, and* (b) *that a tooth with visible absorption at its apex is infected?*

EXPERIMENTAL AND DISCUSSION.

The entire system of dental diagnosis of today would seem to stand or fall on the correctness of these premises. This is true to so great an extent that the medical and dental professions of the country probably depend more on this one diagnostic means than all others combined. One has only to go to the ordinary diagnostic laboratory, whether that of a medical or dental roentgenologist, and observe the procedure. The patients, whether they come in with or without a letter of reference, will be assigned to a technician for making a complete or partial set of dental roentgenograms of the mouth. The roentgenologist or one of his assistants takes the films one at a time and holds them between himself and the light, in the better equipped laboratories often, so arranged as to be very comfortable and convenient with a hole in the top of the table covered by a piece of opal glass and an electric light beneath. A form is filled out as film after film is handled, and a decision is made in an instant on the basis of the above presumption that a tooth without visible absorption at its apex is not infected and that a tooth with visible absorption at its apex is infected.

Allowing ten films to each mouth, a little calculation readily suggests the amount of time that can be taken with each film. We know of several laboratories that claim to take complete sets of roentgenograms for fifty to one hundred patients a day, and have heard of laboratories reaching twice that maximum number. We would not suppose that even a skilled operator would undertake to interpret the films for more than one hundred patients per day. Assuming that the areas where teeth are absent are studied as well as the areas where teeth are present, which should be done, this operator would have to study the condition of several thousand teeth, or their sockets, a day. Assuming eight hours continuous study of this very exacting problem and two thousand teeth to be studied, it would allow less than fifteen seconds per tooth. We are not here presuming to discuss whether the decision is or is not correct, but simply the fact that some fundamental law must prevail, and be very constant, to make it possible for these dental diagnosticians to decide the fate of so many hundreds of teeth per day on such few seconds of observation of a record of their condition. There is no question but that this law is supposed to obtain: namely, that infection in a tooth will express itself as absorption at the root apex; and, conversely, that absorption at a root apex can have only one meaning: namely, dental infection.

This research has been made to verify the correctness of these premises. Figure No. 51-A shows roentgenograms of the central and lateral incisors of a young man, none of which would appear from the Roentgen-ray shadows to be infected; nor would the shade of any of them suggest that they were infected or abnormal. The testing of each tooth individually for a temperature response revealed the fact that the upper left central gives no response. A careful examination of the tooth and tissue surrounding it revealed a small fistula opposite about the middle third on the labial surface of the root. The tooth was opened; and a smear stained and examined immediately was found to contain an abundance of very small diplococci. B and C show roentgenograms from different angles of the tooth with a flexible gutta-percha point placed in the fistula. E and F show two views, one a direct and the other lateral, of the extracted tooth with its granuloma in position on the side of the root, also revealing apical absorption not revealed in the original roentgenograms. D shows a lateral roentgenogram of this tooth and discloses a very large lateral foramen which had its exit beneath the granuloma. A study of this tooth in Figure 51-A discloses a zone of condensing osteitis about the apex in the position which would usually be occupied by a zone of rarefying osteitis. In this chapter we are discussing the significance of what is apparent in the roentgenogram. It will readily be seen that a decision on the basis of what is apparent in the roentgenogram is utterly misleading in this case, for this patient had not only this serious local involvement but had also a systemic expression apparently influenced or caused by this involvement. Cultures were made under controlled conditions, all of which grew out long and short chained streptococci and diplococci in both aerobic and anaerobic conditions.

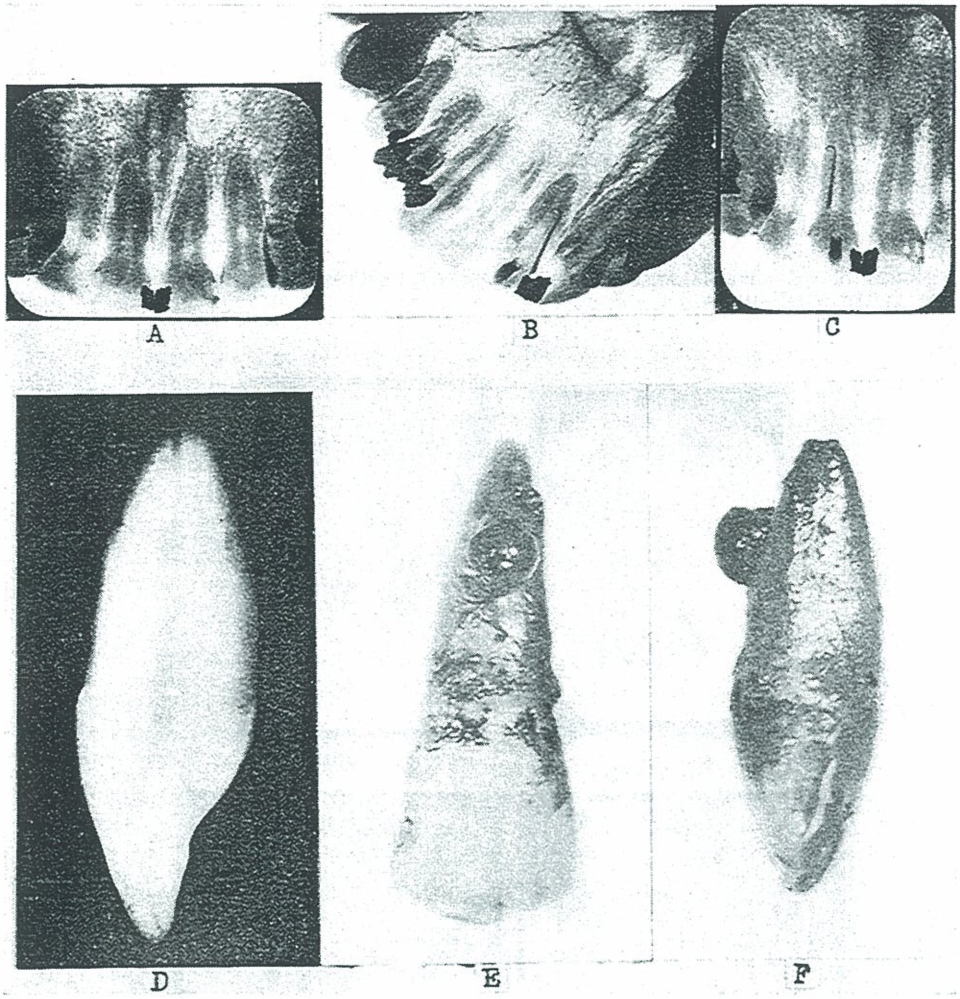
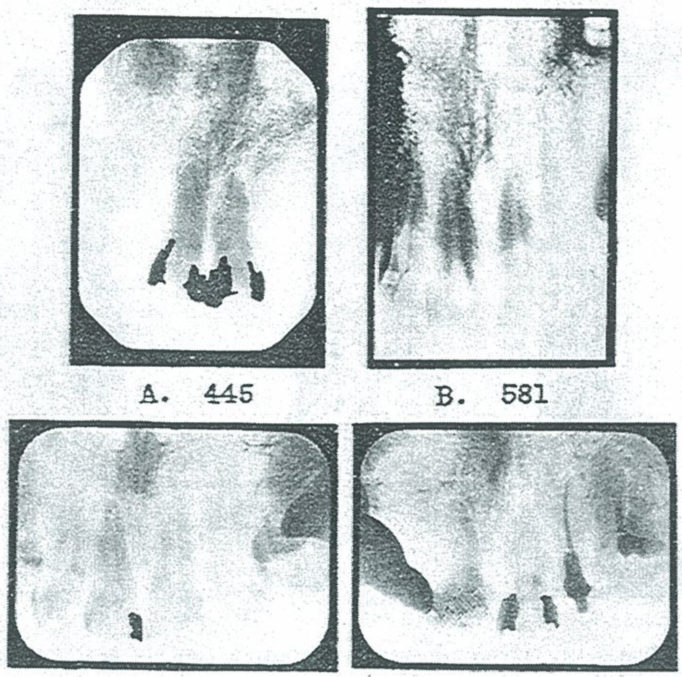


FIGURE 51. DIFFERENT VIEWS OF AN INFECTED TOOTH WHERE THE ROENTGENOGRAPHIC EVIDENCE IS NEGATIVE.

Had this lateral canal lain at right angles to the direction of the incidence of rays, the zone of rarefaction would have been on the side of the root instead of in front of or behind it, and would then probably have been disclosed. Such a case is shown in Figure 52.

Figure No. 53 shows a series of four cases of the roentgenograms of similar central incisors that have been found to be definitely and seriously infected where little or no roentgenographic evidence is apparent. In the many hundreds of cases here being reported, in more than ten per cent of instances teeth were found by other methods of study to be infected where the Roentgen-ray completely failed to reveal or even suggest that condition. To illustrate the importance of this latter we will detail the seriousness of these cases.

FIGURE 52. A LATERAL ABSCESS ON THE SIDE OF A BICUSPID; ALSO APICAL ABSCESS. (SEE TOOTH WITH FILLING.)



A. 445 B. 581

C. 911 D. 706

FIGURE 53. FOUR CASES WITH PUTRESCENT CENTRAL INCISORS. CONDITION NOT REVEALED ROENTGENOGRAPHICALLY.

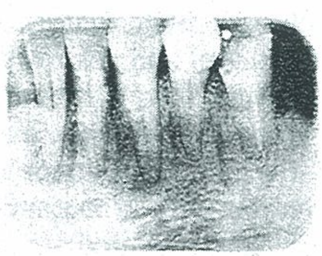


FIGURE 54. A MANDIBULAR CUSPID WITH APICAL RADIOLUCENCY BELOW A VITAL TOOTH.

A shows a woman bedridden for many months with heart and gall-bladder involvement, whose condition was so severe that her death was awaited hourly. She finally passed a large number of gall-stones after which she greatly improved, and as soon as she could be moved was brought to us for study. One of the centrals shown in A is non-vital and the seriousness of its infection was demonstrated by animal inoculation with the culture. With the removal of this and a couple of other teeth showing also a very little evidence of infection, and whatever benefit she derived from the passage of the gall-stones (which, no doubt, was very considerable) her condition has so greatly improved that for five years she has had no recurrence and is again doing her household duties with little or no evidence of heart involvement.

B shows the roentgenograms of the teeth of a young married woman, age thirty-two, who was brought to us with a heart infection. This occurred some years ago when we had more courage based on ignorance, and we consented to treat and root-fill this tooth, and make a curettage of the root end. A couple of other teeth were extracted at this time. The patient's condition improved a great deal and she resumed the responsibilities of her home. During the overload of a subsequent pregnancy her heart became very badly involved again, at which time she was carried to us; and as she lay in the ward, had so much dyspnea that about every fifth breath she would gasp. Even while lying quietly her pulse was 120. With the removal of this tooth, which (neither at the time of its first discovery and treatment or at this time, a couple of years after its root filling,) has never had any tenderness to suggest that there was anything wrong with it, the patient has had an apparently quite complete recovery, and has been taking care of her household duties, going up and down stairs, etc., for two years. She looks and feels splendid. In Chapter 60, on Circulatory System, I review this case with illustrations of the heart lesions produced in rabbits by the inoculation of the culture from this root filled and curetted tooth. The important item for us to stress is that so seriously an infected tooth gave no physical symptoms of being abnormal and, as in the last case, practically no roentgenographic evidence that it was not normal.

C shows a case of very severe rheumatism which crippled the man so severely that he could walk only by shuffling his feet

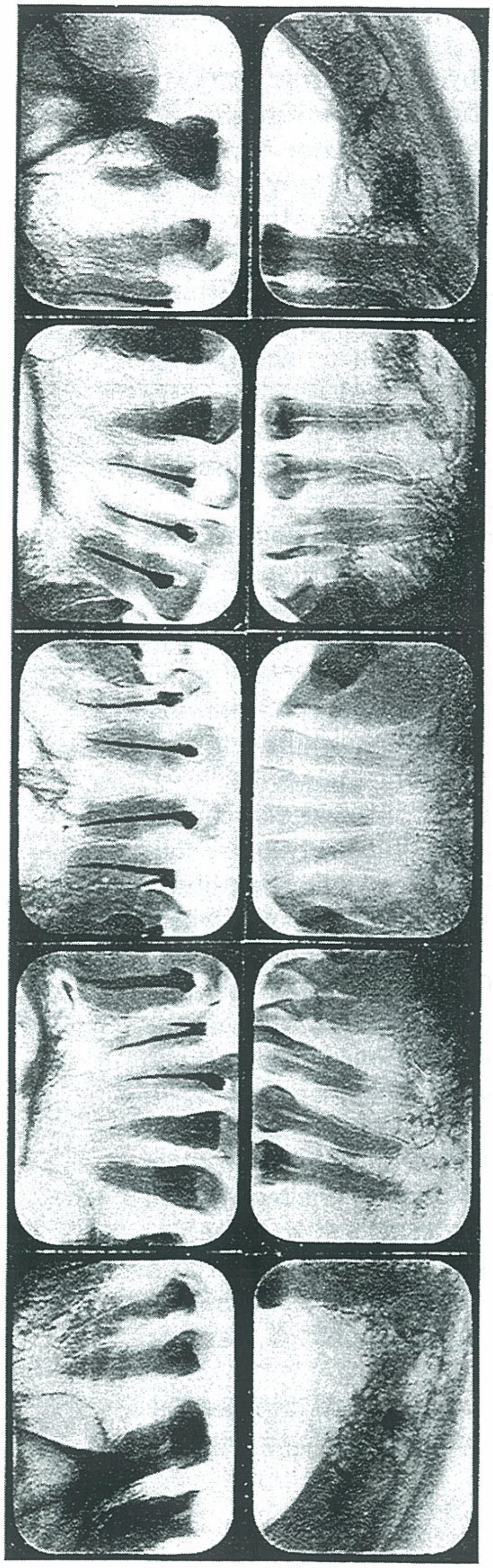


FIGURE 55. MAXILLARY ANESTHESIA.

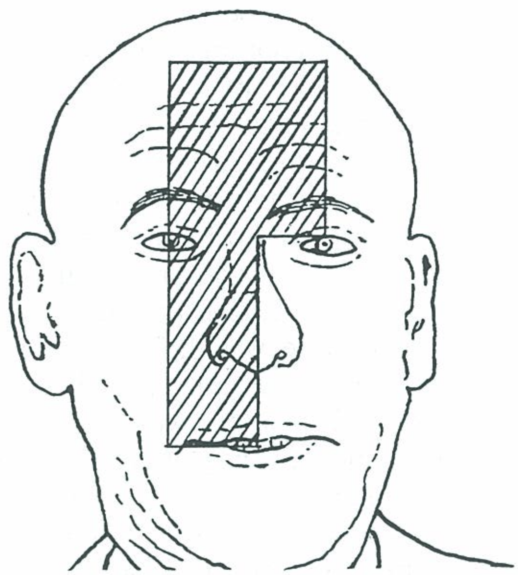


FIGURE 56. SHOWING ZONES OF EXTERNAL ANESTHESIA IN THE CASE OF THE PREVIOUS FIGURE.

slowly. His hands were equally helpless. With the removal of this infection his rheumatic symptoms, which had been recurring for some time, entirely and quickly disappeared and have not returned in four years. There was no change in the color of this tooth to suggest or indicate its condition and the patient vigorously protested against its removal though he is now extremely grateful. Figure 10, Chapter 1, illustrates the result of animal inoculation with this culture.

Figure 53-D shows the roentgenograms of the central incisors of a young lady suffering from a very severe neuritis in her neck. She had had a serious fall and there was a fear at one time that her neck had been broken. This pain had persisted for months though with markedly changing severity. It is not known how much of the improvement that occurred in this case was due to the removal of dental infection but there seemed to be considerable.

These are just a few typical illustrations of the extreme need for more efficient methods of dental diagnosis, than the very questionable presumption that the extent of the dental infection is that which is suggested in the roentgenogram. As shown in these four cases there was practically no evidence, for in the last case (D) the tooth that was involved was one carrying a porcelain filling, which looks like a cavity in the roentgenogram, and is not the one showing the two gold fillings and which does suggest the possibility of a periapical involvement. A close observation, however, of the involved tooth discloses a zone of condensing osteitis surrounding the apex of the upper right central.

PROBLEM NO. 5B: Does apparent absorption about a root apex necessarily reveal infection?

Figure No. 54 shows a lower left cuspid with very definite evidence of absorption of bone about the apex of the root. By both the thermal and electrical tests, and by drilling of the dentin, the tooth responds with complete normality. This tooth would readily be condemned for root filling or extraction if complete dependence is to be made upon the evidence of the roentgenogram.

Figure No. 55 shows the present condition of the upper incisors, each with areas of absorption, all of which according to history were getting larger. A careful study of the history, as furnished by the patient and the dentist, revealed the following: The woman was a colored servant in a home in another city. One day

FIGURE 57 SHOWS AREAS OF ARSENICAL NECROSIS ON DOG'S TONGUE FROM DEVITALIZED TEETH.

FIGURE 58 SHOWS SEVERAL VIEWS OF TREATED TEETH, SOME WITH ARSENIC, OTHERS WITH FORMALIN.

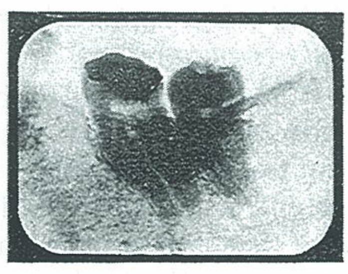


FIGURE 59. AN ENLARGED APICAL MEDULLARY SPACE RESEMBLING APICAL INVOLVEMENT.

she observed that some of her teeth felt numb when she tapped them. She went to her dentist and he tested with thermal change and by drilling, and found that the central incisors had no sensation and proceeded to remove the pulps which, as he described it to me afterwards, notwithstanding that the teeth were dead, bled like live pulps and that there was absolutely no life in them. He treated these teeth with tricresol-formalin and changed the medicine frequently. One after another, as he tested additional teeth, he found that they did not respond to temperature or to drilling, and he proceeded to remove the presumably lifeless pulps. These he also treated with tricresol-formalin and to his amazement, notwithstanding the fact that he changed the medicine very frequently, after weeks of continuous treatment he was not able to stop the apparent infection at the apices, for the abscesses, as he described them, grew larger and larger. A careful examination revealed that not only the teeth but the soft tissues of the entire upper jaw of this patient's mouth had no sensation. What had been taking place clearly was that teeth with normal pulps, but to which the nerve supply had been destroyed, had been removed, and sterile pulp chambers had been saturated with irritating medicament which penetrated the apices and produced an ever increasing zone of necrosis, and which areas were the result of irritation of a kind other than bacterial. Figure 56 shows an outline of the zones of anaesthesia. In seven years there has been little change in the boundaries of the zones of anaesthesia, though it is becoming more complete.

Figures 57 and 58 show the tongue and teeth of a dog, in whose teeth I placed arsenic and covered it carefully with amalgam fillings in cement to make a very tight seal. The dog was killed at the end of a week. The photograph shows large areas of necrosis on the tongue where the arsenic had penetrated from the pulp chamber (which is very small in dogs' teeth) through the root walls and apices to the supporting structures which were definitely necrosed and even produced extensive lesions on the dog's tongue as shown.

There was a time in the history of the practice of dentistry, and not far removed, when many dentists—we hope none now—were continually using arsenic for the devitalization of dental pulps; and I have frequently seen, when that practice was in vogue, areas of necrosis produced by the arsenic, which could easily be mistaken for zones of infection, and which could readily

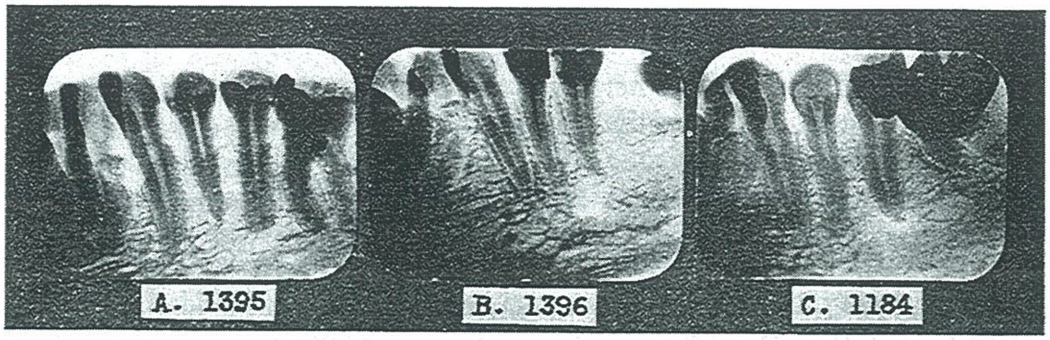


FIGURE 60. MENTAL FORAMINA WHICH MIGHT BE MISTAKEN FOR APICAL INVOLVEMENTS.

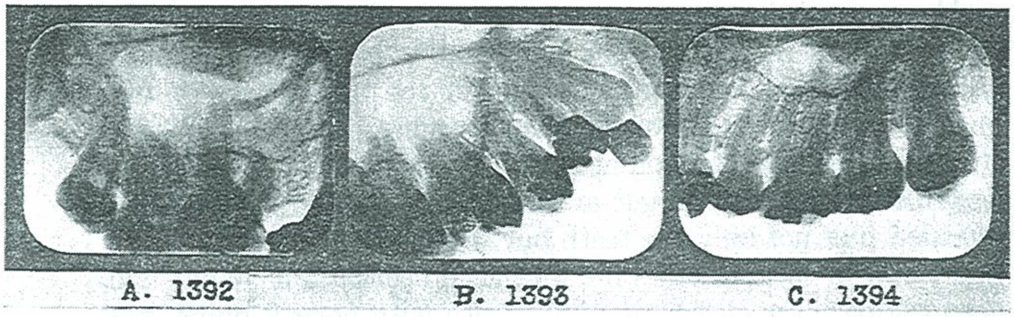


FIGURE 61. ANATOMICAL RELATIONSHIP OF MAXILLARY SINUS AND PALATE, SUGGESTING APICAL INVOLVEMENT.

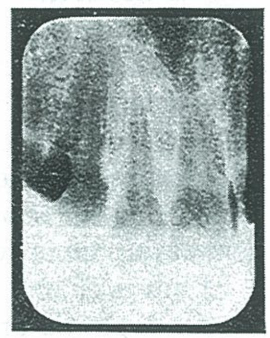


FIGURE 63. THICKENING OF PERIDENTAL MEMBRANE, DUE TO ORTHODONTIA.

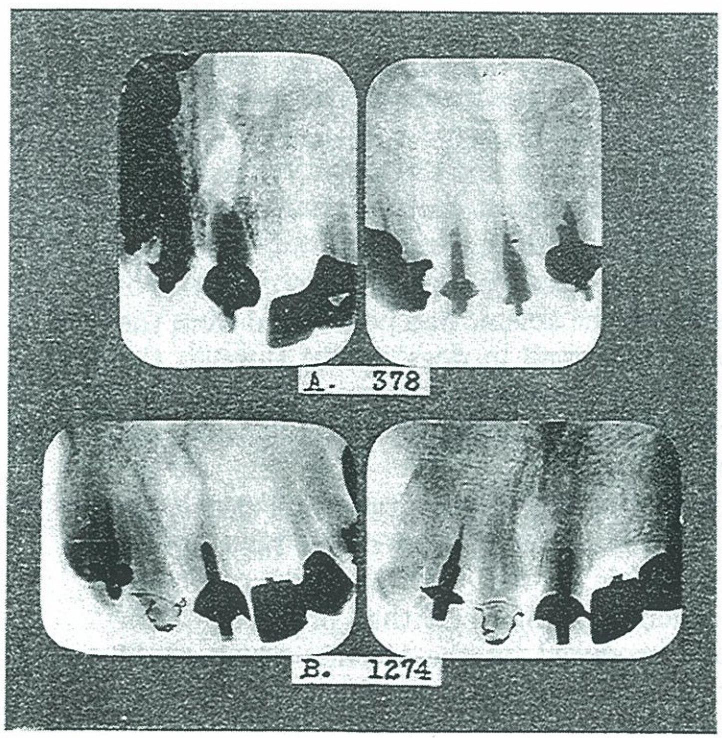


FIGURE 62. TWO VIEWS OF NASOPALATINE FORAMINA, EASILY MISTAKEN IN ONE VIEW FOR APICAL INVOLVEMENT.

FIGURE 64. PUTRESCENT BICUSPID WITHOUT APICAL INVOLVEMENT.

become focal on the entrance of bacteria. There can be no doubt that great damage has been done by this practice.

In Chapter 14 on Tooth Medications, I report our studies on the irritating effects of the medications currently used. Much work has been done since, demonstrating that dental medication may be a very definite cause of bone absorption about the apices of the treated teeth.

We very frequently see reports from non-dental diagnosticians, including roentgenographic interpretations of dental conditions, which specifically condemn and assign for extraction certain teeth which show evidence of apical absorption. Figure 59 shows a third molar with a zone of apparent decalcification which is in all probability an anatomically large medullary space, for this tooth is by all physical tests normal. Figure 60 shows lower bicuspids in which the mental foramen has been mistaken for an apical absorption. Figure 62 shows two centrals which have been condemned because of the apparent apical involvement but are due to the angle at which they are taken, throwing the root apex in such a relationship to the nasopalatine foramen, that it appears as an apical absorption. Figure 61 shows three illustrations of an anatomical relationship as it is disclosed by the roentgenogram, and which appears like an abscess on the palatal root of the upper molars, and is produced by the transparent zone of the maxillary sinus in alignment with the anteroposterior grooves.

Another type of irritation which can easily be misunderstood is that due to trauma, such as the overload in a tooth which frequently produces a very marked thickening of the peridental membrane, which, to the untrained, would readily be mistaken for a pericemental involvement. The removal of the overload, whether traumatic occlusion or the carrying of a fixed bridge, will usually entirely correct this condition. Another type of apical involvement also due to trauma of a different kind is produced in certain orthodontic procedures where, with the movement of the tooth, there is a distinct zone of rarefaction disclosed about it. Such a condition is shown in Figure 63.

It is, of course, understood that much of the data in this chapter should be common knowledge and is inserted here for the benefit of those not familair with these facts. The need for it has been suggested by a very large number of interpretations that have come to my hands, which indicated that the diagnosticians making them were not familiar with these facts.

It is common practice to depend upon the roentgenograms of teeth for a final decision as to whether they are or are not infected. I have previously stated that in our practice, approximately ten per cent of the teeth that we find to be infected and seriously injuring the patients, do not have the same revealed by the roentgenograms. Figure 64 shows a bicuspid tooth with a putrescent pulp, which condition is not suggested by changes in the supporting structures. In this patient's mouth several teeth were found similarly non-vital without any local symptom to suggest it. The patient was suffering from a nervous breakdown and her condition greatly improved following the removal of the non-vital teeth.

SUMMARY

We are led by these studies to conclude:

(1) That dental infection will not of necessity produce bone absorption at the apex of the root of the involved tooth; and

(2) That absorptions, when they occur, are not of necessity a result of bacterial irritation.

CHAPTER VII.

THE RELATION OF CARIES TO PULP INFECTIONS.

PROBLEM: What is the relation of the health of pulps without exposure to shallow and deep caries?

EXPERIMENTAL AND DISCUSSION.

Dental literature and dental practice have taken for granted that teeth, with exposed pulps as a result of dental caries, may be and probably are infected; but that teeth, with a dental caries which has not reached and uncovered a pulp chamber, are not yet infected, and are available and safe for filling by the indicated procedures. Clinical experience, however, has indicated to all observing operators that teeth with large fillings tend to develop symptoms, more or less severe, of pulp involvement, very often observed as hypersensitiveness to thermal change, and later pulpitis and pericementitis. So many teeth were found having non-vital pulps in which there were no symptoms whatever that the patient could distinguish that a series of careful studies was made to determine, if possible, the extent to which caries must have advanced before there was danger of pulp involvement, and what the early expressions in pulp tissue would be.

A practice, still more or less common but more generally practiced a few years ago, was that of pulp capping, by which some operators undertook to place over the zone of pulp involvement a non-irritating protection. Some of these were very ingeniously devised by making a little hood of gold or platinum into which was placed medicated paste, and the capping placed over the exposure and covered with cement, over which the permanent filling was placed. Statistics indicated that this operation was more likely to succeed in hot climates than in cold.

One of our experiences of years ago convinced us that there was danger in some of these pulp involvements to a degree far exceeding that which we had expected. A boy of fifteen years presented with a very acute rheumatism, being brought by a nurse from a Visiting Nurse Association. The history showed that four weeks previously he was compelled to leave school because of an acute tooth-ache definitely located in the left mandible. This acute pain lasted only a few hours; then it entirely disappeared. About a week or ten days later it was found that he could not get up from his seat in school and had to be carried home with acute rheumatism which kept him in bed for two weeks, after which he was hustled back to school. The teacher reported that he did not seem to care to play with the other children and seemed very tired, which she and the visiting nurse interpreted as being due to the lingering rheumatism. To our amazement we found a very bad endocarditis, with the heart greatly enlarged and already some cyanosis.

Examination of his mouth revealed deep caries without pulp exposure in the lower left first molar. The tooth was extracted and the boy ordered to bed under strict control of the district physician, which directions were not properly carried out; and he was buried in about seven months as a result of a complete breakdown of his heart. Before the tooth was extracted it was determined that the pulp responded nearly normally to irritation and thermal change. After extraction the tooth was sterilized externally, including the pulp cavity, and after the removal of the caries, culture was made from the pulp, which pulp, macroscopically, was nearly normal except that it showed slight congestion. Of thirty rabbits inoculated from this culture, 93.3 per cent, (28) developed acute endocarditis within a few days, and 100 per cent developed acute rheumatism. This, with many other experiences, made us very suspicious of the pulps of teeth with deep caries and prompted these special studies.

Teeth with more or less deep caries were accordingly selected for study. Figure 65 shows the pulp tissue underneath a caries that did not extend more than half way to the pulp but which was slightly abnormal to temperature change. The capillaries are enlarged and there is a marked infiltration of leucocytes just beneath the odontoblastic layer, with an area of necrosis. This patient had symptoms of an acute neuritis, which, incidentally, were greatly relieved by the extraction of the tooth; and it is very clear that a focal infection within the pulp is mechanically so situated that its toxic products must of necessity go into the lymphatic and hematogenous circulations; and, as necrosis proceeds, organisms can readily enter each of these two circulations.

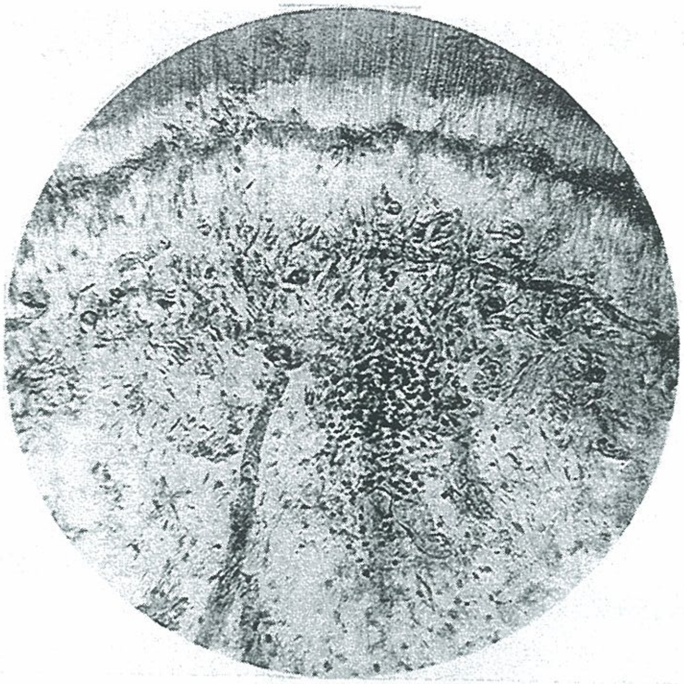


FIGURE 65 SHOWS THE PERIDENTAL LAYER OF THE PULP IMMEDIATELY BELOW A DENTAL CARIES. SEE FRONTISPIECE FOR HEMORRHAGE ASSOCIATED WITH PULPITIS.

In this case, as in many, the local symptom of pain and the reactions in the sympathetic nervous system were entirely out of proportion to the direct toxic reactions.

Figure 66 illustrates a very common type of pulp involvement following and accompanying deep caries. Figure 66-A shows a cross-section through the pulp of the three canals and the caries cavity on the mesial surface of an upper left second molar. It will be noted that the caries had extended only about half way to the pulp. Figure 66-B shows a roentgenogram of the tooth in the mouth with the metallic filling in place. In A is seen the location of a very large pulp stone, which type of calcification is expressing itself in many places as islands, as shown in Figure 66-D. There is a marked congestion of the blood vessels in the vicinity, which are shown in E. This was taken from the mouth of a man about sixty-five years of age, and his symptoms, as in the last case, were largely elsewhere; and it was only on testing that it was found that the pulp was abnormally sensitive to reactions from irritants. Figure 67 shows, similarly, extensive calcifications with multiple islands in a tooth with moderately deep caries but without pulp exposure.

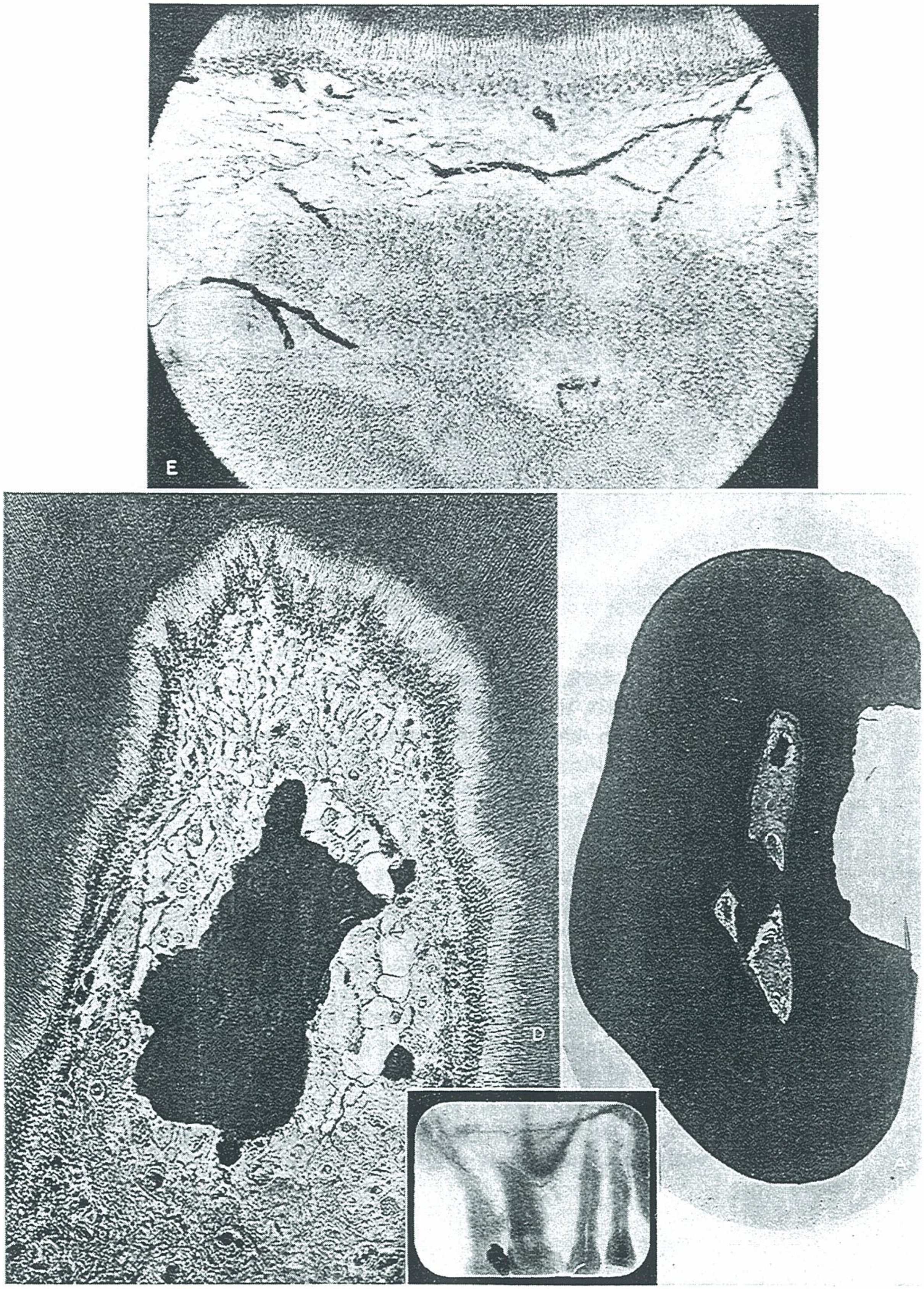


FIGURE 66. PULP INVOLVEMENT AND METALLIC FILLING: B, ROENTGENOGRAPHIC; A, CROSS SECTION OF CAVITY AND PULP; D, PULP STONES; AND E, HYPEREMIA AND FIBROSIS.

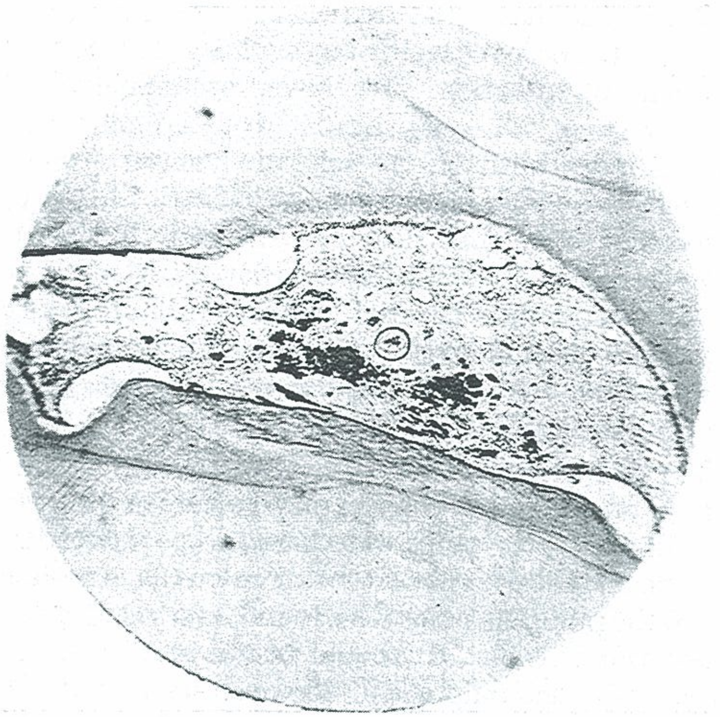


FIGURE 67. CALCIFICATIONS WITHIN PULP BENEATH MODERATE CARIES.

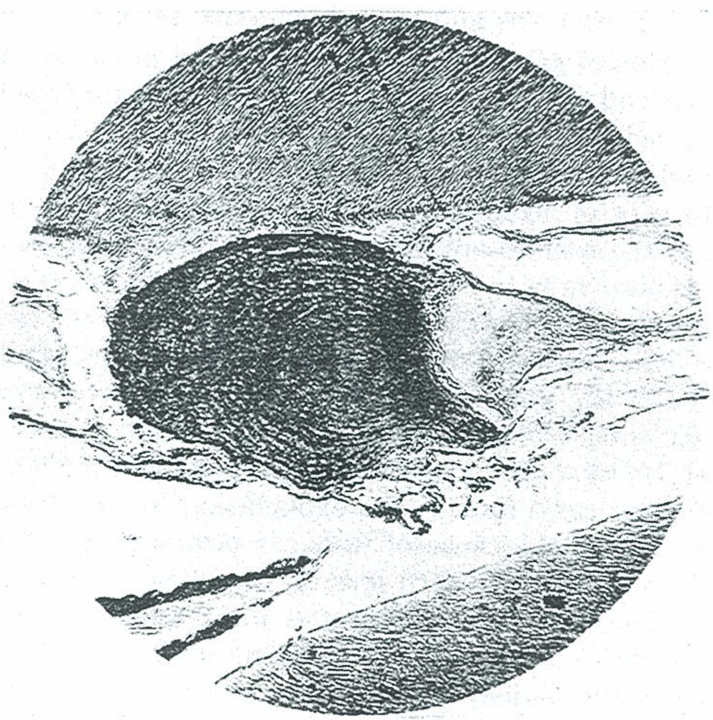


FIGURE 68. EXTENSIVE CALCIFICATION IN PULP OF TOOTH OF A BOY FOURTEEN.

It might be expected that a condition such as shown in Figure 66 might be found in the pulps of teeth of individuals of an advanced age, independent of irritation. We have, accordingly, made studies of the pulps of young individuals to determine whether the condition is independent of the sclerotic changes attendant with advancing years. Figure 68 shows a calcification in the pulp of a molar tooth of a boy about fourteen, the tooth having deep caries of long standing. It will be noted that the island is very large and shows distinctly a lamellar laminar structure, indicating that it was laid down in layers. It can readily be understood that, notwithstanding that this tooth had not yet given trouble, it would be very strange if it did not in the future.

A bacterial examination of the pulps of teeth with caries has revealed that in practically all cases of pulp culture of deep caries without pulp exposure, the pulps were found to be infected; and in teeth with moderate caries not extending more than one-fourth the distance to the pulp, the pulps were found infected in approximately 50 per cent of cases. It should not be implied that the presence of germs in the pulp would demonstrate that the pulp must later become a focus of infection, for it is definitely demonstrated that infections may involve the circulation temporarily as a result of acute colds and special epidemic infections.

This condition of calcification may be located in islands or be adherent to a wall of the pulp chamber, or may surround the pulp tissue on all sides and almost entirely obliterate it. Figure 69 shows two degrees of magnification of a section of the pulp through the root of a tooth where the calcification has reduced the lumen of the root to approximately one-eleventh of the original cross-section area. The dark area in the center is the pulp tissue; the granular layer is a calcified zone and is an osteoid structure. This tooth did not present local symptoms of abnormality, though the patient had very distressing symptoms of neuritis. Cultures from this tooth inoculated into animals developed very marked disturbances and pathological changes.

Some of these cases furnish the explanation for the so-called obscure neuralgias, the cause of which is ordinarily not found; and the patient may suffer both severely and long. Or it may be that the involved tooth may be located and proved to be one in which the dentist has placed a filling some months before; and the attitude of the patient will probably be one of very definite criticism of the judgment of the operator, assuming that he should

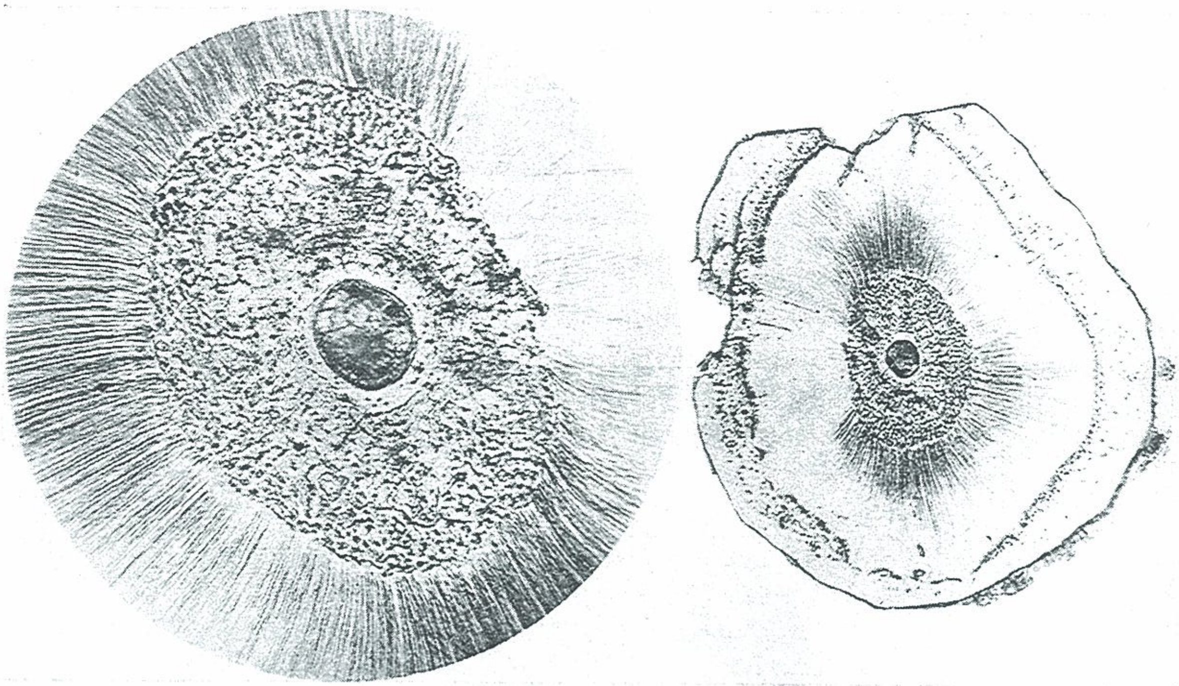


FIGURE 69. THE FILLING IN OF THE PULP CHAMBER WITH AN OSTEOID BONE.

have known whether or not it was safe to place an expensive gold inlay over the pulp of the tooth, even though, up-to-date, it had not given trouble. Unless the operator knows the probability of this type of pathology, he will find it very difficult to make a defense or explanation that will be satisfactory to the patient; and until the patients know of this danger they will continue entirely to misjudge the operator.

Figure No. 70 shows a case that illustrates this point. The patient has presented with an obscure neuralgia involving the left ear and the temple, with a symptom of hypersensitiveness to temperature change, which she locates in the upper teeth. No tooth is tender or has had a localized pain; yet the condition is severe. Careful examination reveals that the tooth that is abnormally sensitive to temperature change is the lower left second molar. An inlay placed three months before is in excellent condition. The history of the tooth is that the progressive caries had extended deeply, about half way through the dentin on both the occlusal and distal surfaces. On removal of the inlay, the dentin beneath a protecting layer of cement responded normally. It was decided that the pulp should be condemned and that this

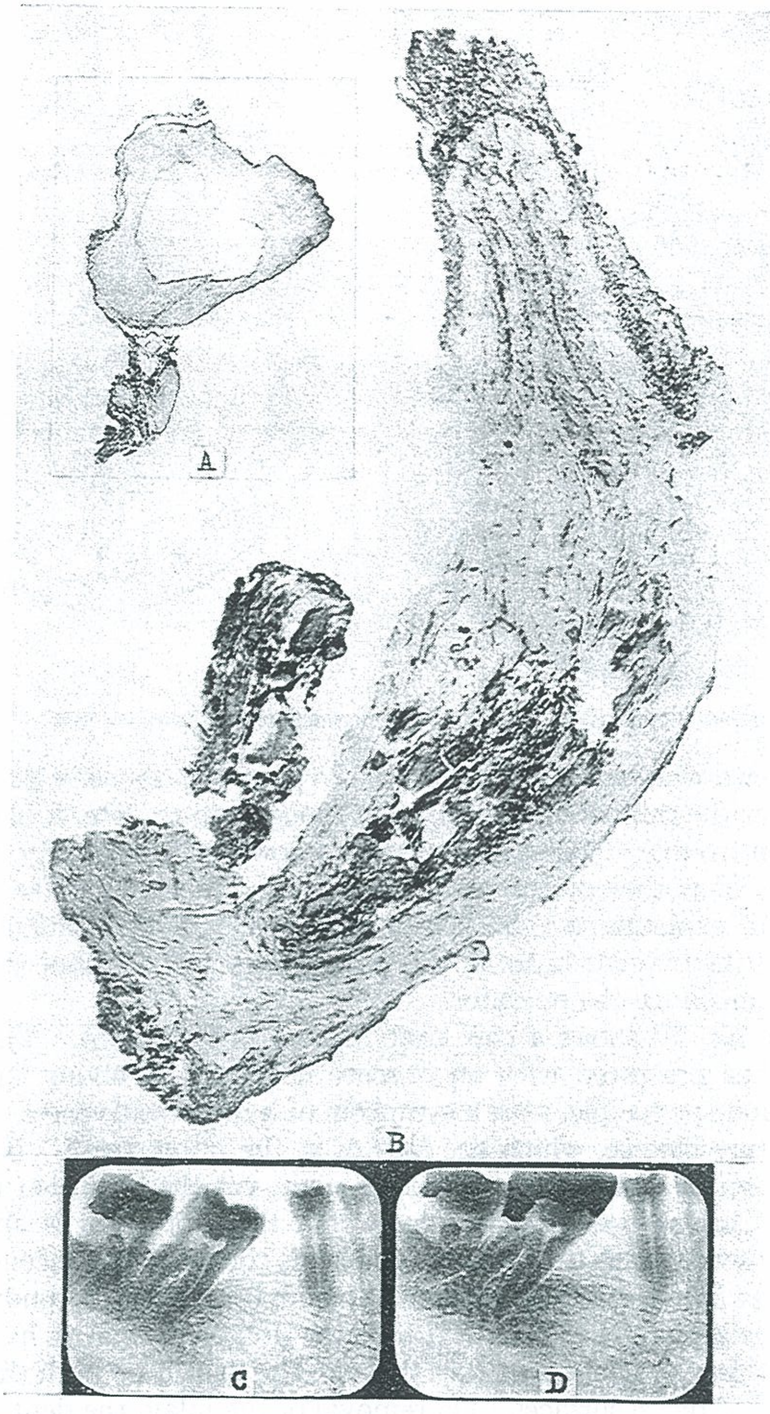


FIGURE 70. PULP CHANGES RESULTING FROM IRRITATION OF CARIES PLUS IRRITATION OF FILLING: C, ORIGINAL CARIES CAVITY IN SECOND MOLAR; D, SAME WITH GOLD INLAY; B, DEGENERATION CHANGES IN PULP WITH FIBROSIS AND CALCIFICATION; AND A, LARGE PULP STONE IN CORONAL PORTION OF PULP, NOT DISCLOSED BY ROENTGEN-RAY.

patient could have the roots of this tooth filled, provided good access could be had. The pulp was removed under mandibular anaesthesia and sectioned. The pulp is shown in Figure 70 to have very extensive zones of calcification.

Other cases are shown in Chapter 66 on "The Nervous System and the Sense Organs."

Since the process of dental caries involves decalcification, the roentgenogram becomes a most important aid in detecting obscure zones and early stages of dental caries. This process of decalcification may be exhibited as extending from its beginning in the tooth to, or nearly to, the pulp. This is illustrated in Figure 71, in which a zone of decalcification is shown extending from a caries which has entered beneath an alloy filling and then has followed the dental tubuli directly toward the pulp. This is also shown clearly in Figure 72, in which the zone of decalcification can be traced in the roentgenogram from the open cavity directly toward the pulp. Figure B shows the pulp tissue directly beneath this zone of decalcification, and Figure C islands of calcification within this pulp tissue. The process by which the decalcification is carried on by bacteria and the systemic factors involved in it are discussed in Chapter 29 "Etiological Factors in Dental Caries."

I desire to present in this chapter an illustration of the progress of dental caries pulpward through the dentin. Figure 73 shows the details of the base of the cavity of dental caries in two magnifications revealing the etching of the tubuli and the advancement into these openings of the bacterial growth having entered the

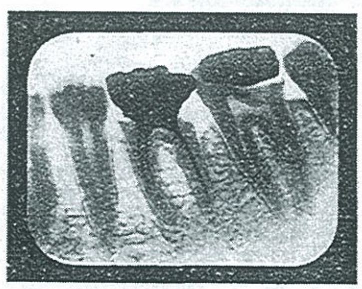


FIGURE 71. A ZONE OF DECALCIFICATION EXTENDING FROM THE CARIES CAVITY TOWARD THE PULP.
(See second molar)

tubuli. The direction of this can most readily be made toward the pulp. The organisms furnish their own tools for dissolving the dentin, and progress as fast as they have enlarged the tubuli. B shows the enlargement of the tubuli far below the surface caries. The toxic substances produced by these organisms have as direct

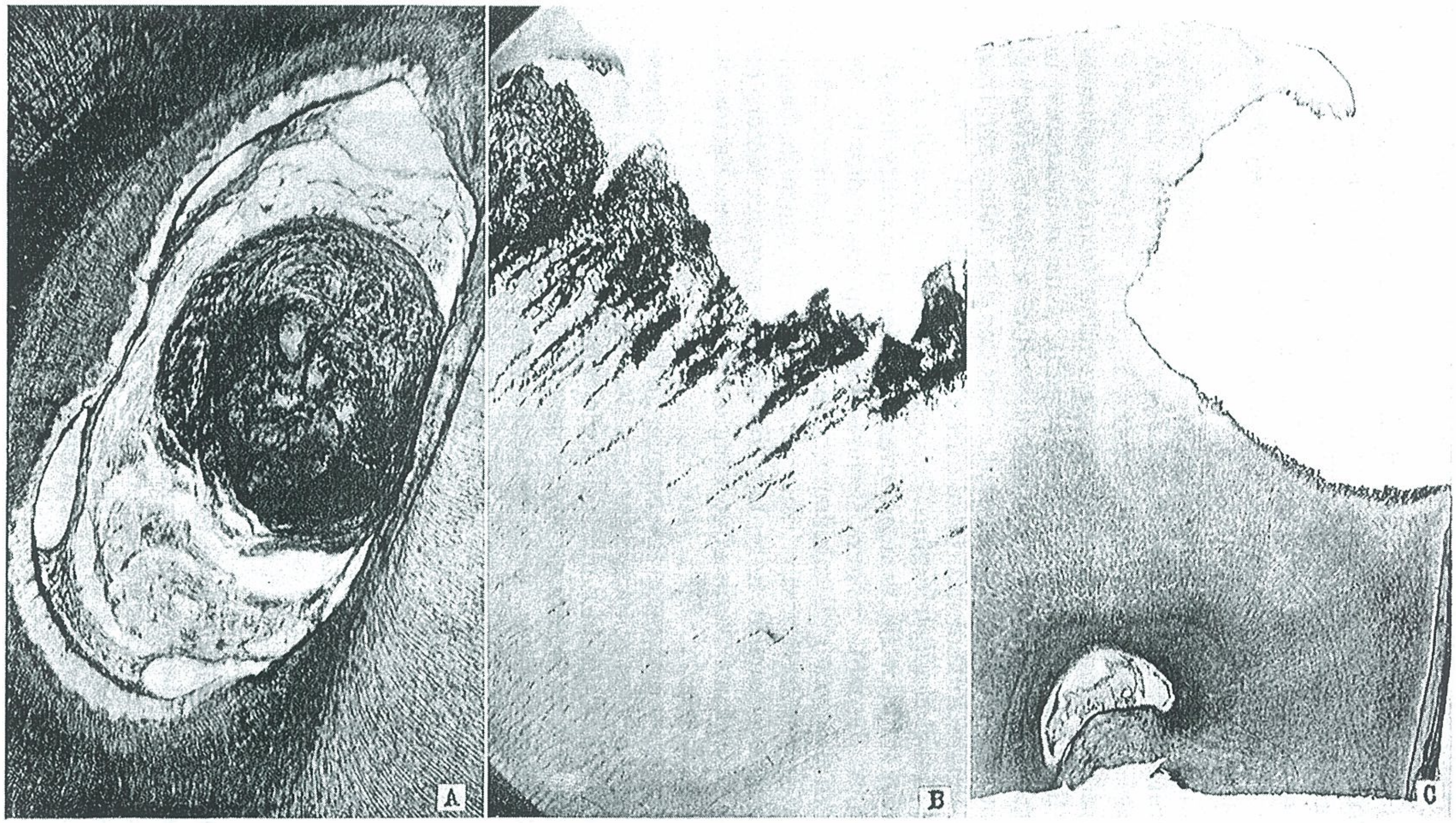


FIGURE 72. SECTIONS OF A TOOTH WITH DEEP CARIES, TRACING CHANGES TO PULP: C, A CROSS SECTION OF TOOTH SHOWING THE RELATION OF CAVITY TO PULP; B, MAGNIFICATION OF DENTIN SHOWING ADVANCEMENT OF DECALCIFICATION FOLLOWING TUBULI; A, A PULP NODULE AND FIBROSIS IN PULP CHAMBER.

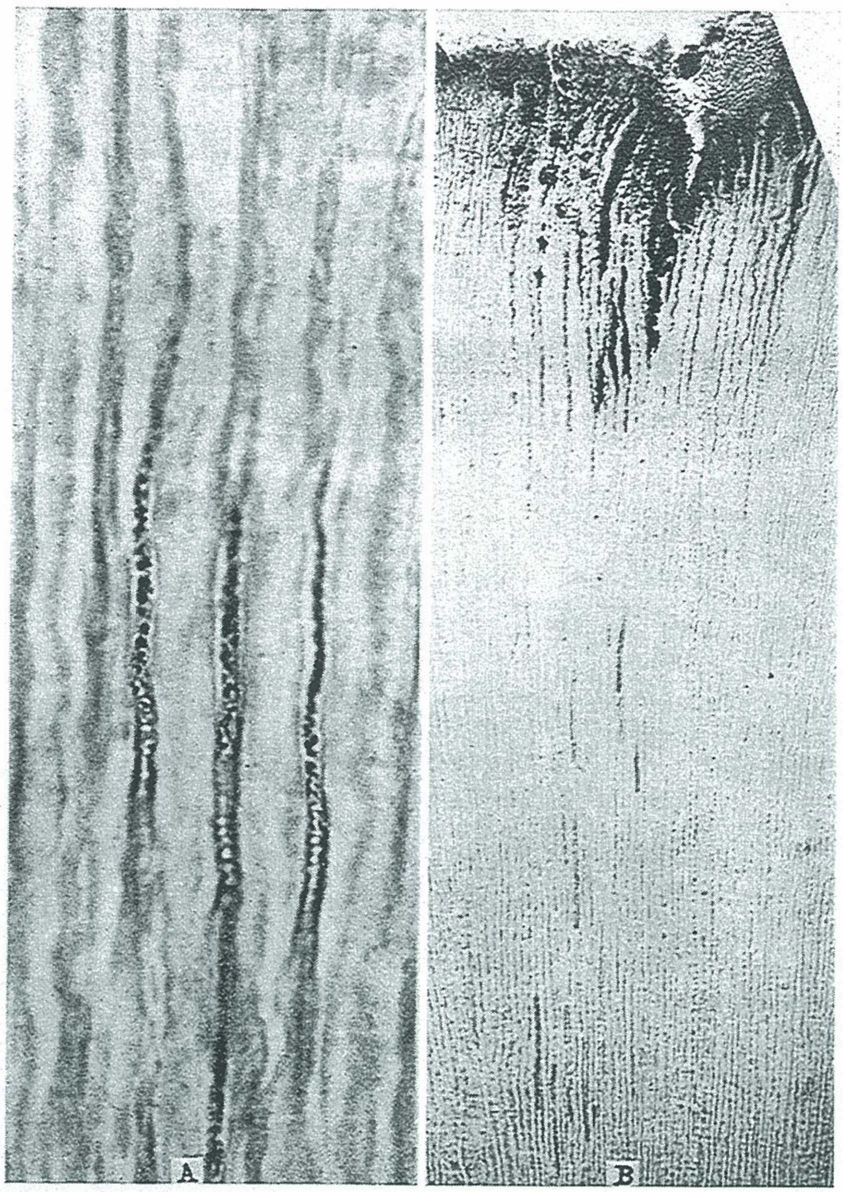


FIGURE 73. AN ILLUSTRATION OF THE DEPTH OF DECALCIFICATION FROM A SUPERFICIAL CARIES: B, FROM THE BASE OF THE CARIES CAVITY INWARD, SHOWING THE ENLARGED DENTAL TUBULI; A, ENLARGEMENT OF THE DENTAL TUBULI, SHOWING BACTERIAL ADVANCEMENT FAR TOWARD THE PULP.

access to the pulp as to the open cavity, and will most naturally develop in that direction, because it is in the direction of their food, since they live on the disintegrating tissue material which their own toxins devitalize in their advance.

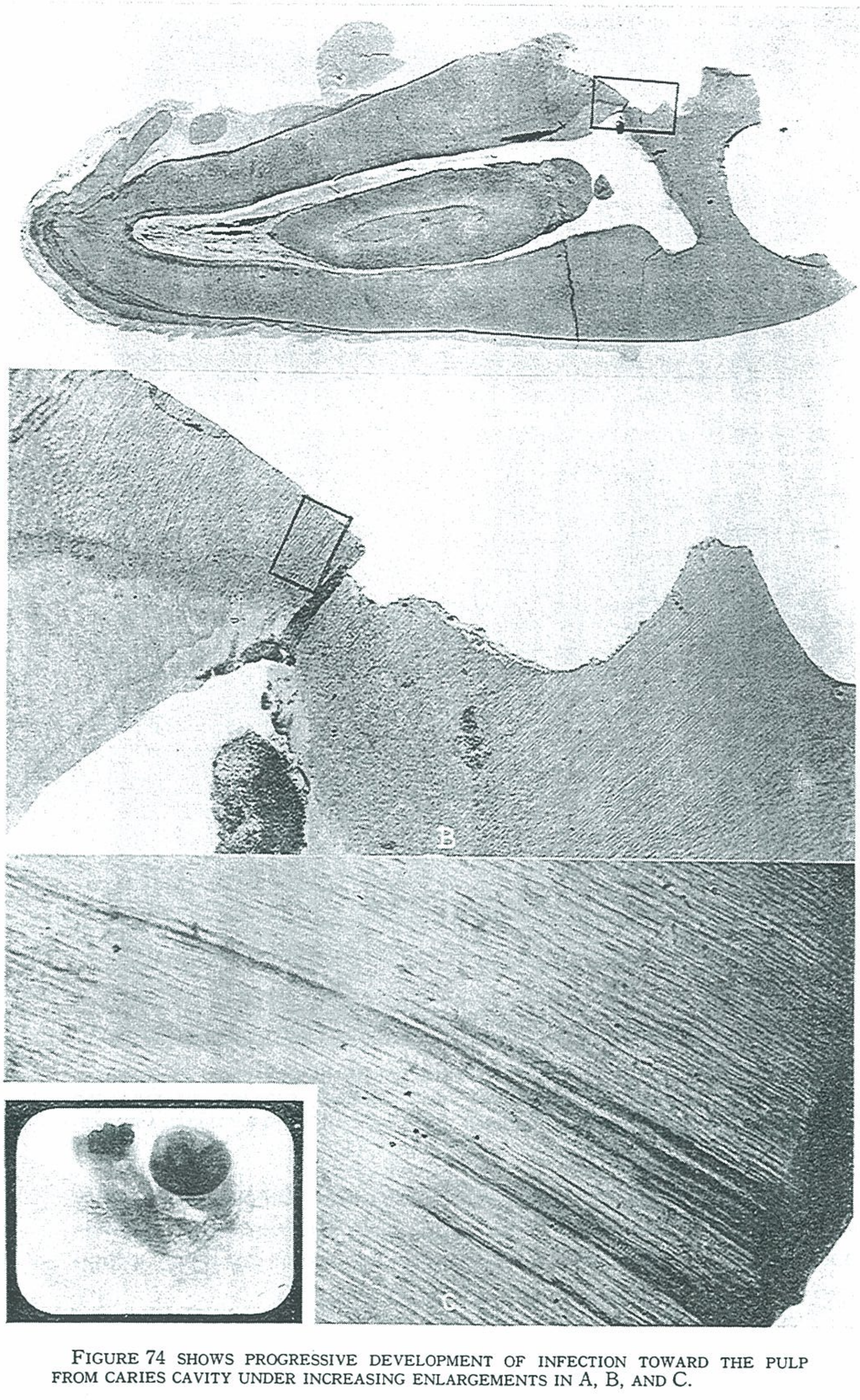


FIGURE 74 SHOWS PROGRESSIVE DEVELOPMENT OF INFECTION TOWARD THE PULP FROM CARIES CAVITY UNDER INCREASING ENLARGEMENTS IN A, B, AND C.

In Figure 74-A we have a cross section of the molar shown in the roentgenogram in 74-D. In the latter you will note a large alloy filling in the occlusal surface of the molar, and a distal cavity of caries at the point of contact with the malposed third molar. This tooth was vital and producing sympathetic disturbances from pulpitis. In B we have a magnification of the zone included in the oblong outlined in the margin of the caries cavity of A, but enlarged about 200 diameters; and in C we have a zone from the base of this area of the cavity, which is outlined in the oblong in B, enlarged to about 1200 diameters. In this latter the dentinal tubuli are shown enlarged to a point where the etching out of their lumina can be distinctly seen. The irritation upon the pulp tissue has been in progress for a long time, with the result that there has been a progressive degeneration, first as a congestion, then a fibrosis and calcification, the latter of which is shown in A at the point of fusion of the mesial and distal canals. This zone is shown enlarged in Figure 75-A; and it will be noted that in addition to the large islands that were so clearly seen and outlined in Figure 74-A, there are myriads of small ones resulting from the fibrosis. In Figure 75-B, a zone of the dentin beneath and at the margin of the leaking alloy filling is shown enlarged to about a thousand diameters; and in C, enlarged to about 1800 diameters. It would not be difficult to find arguments for and against the conclusion that the bodies shown are microörganisms. We will not introduce a discussion of this phase of the problem in this chapter. Whether these are organisms or products resulting from bacterial growth, the resultant irritation upon the pulp tissue has been very positive.

But these inflammatory irritations are not limited to the pulp itself, but are extended to the peridental membrane and the supporting structures. This is shown in Figure 76, A and B. In A we have marked changes in the vascularization. The large, dark, round area is a calcification within the lumen of a blood vessel. Between it and the cementum another blood vessel is shown in the process of degeneration. Transverse fibers are invaded with round cells and leucocytes, with much evidence of inflammatory process. These irritation processes produce at some points degeneration with absorption, and at others, prolif-

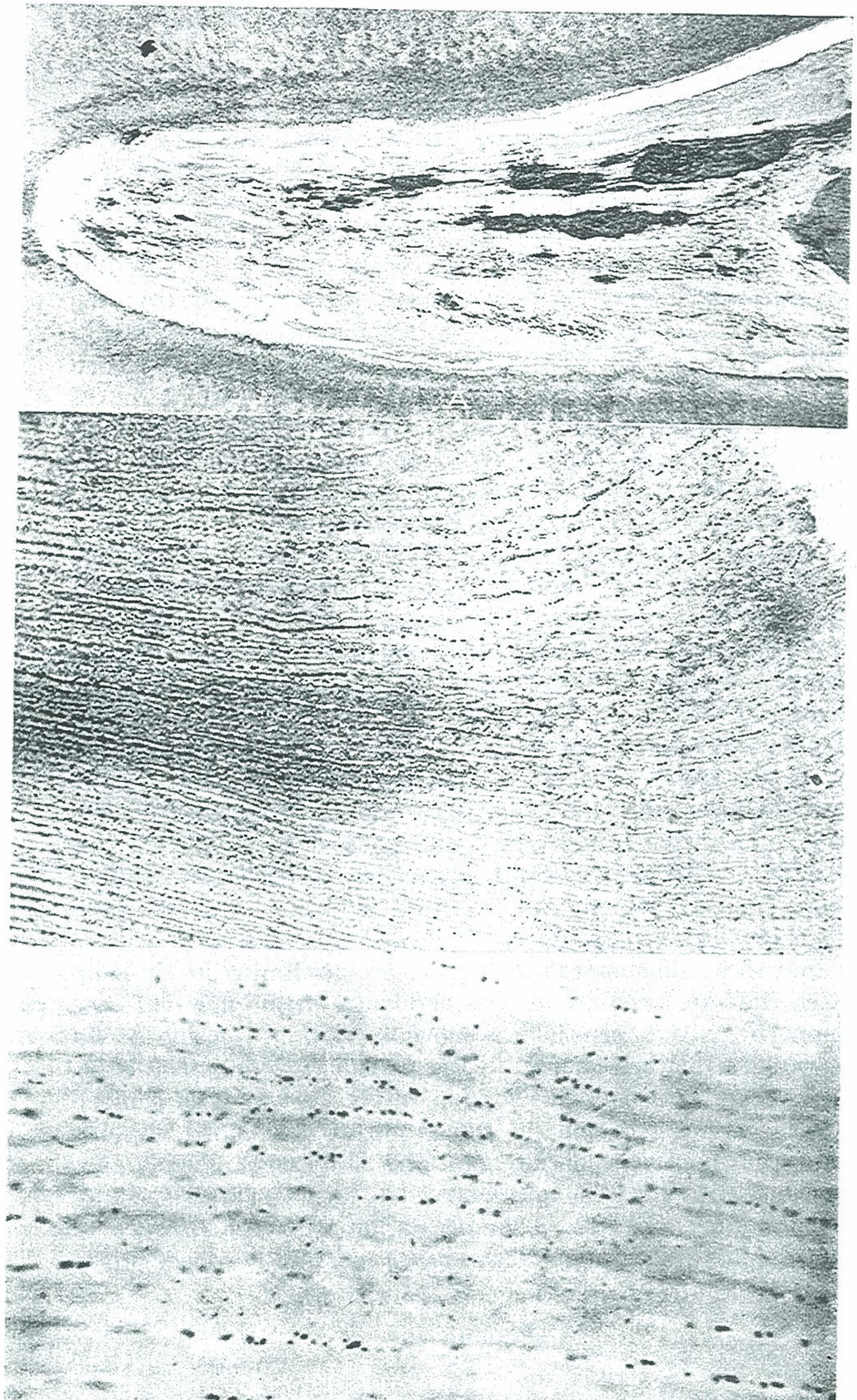


FIGURE 75 SHOWS CALCIFICATION ZONES IN PULP IN A; THE DENTIN BENEATH A LEAKING ALLOY FILLING IN B; HIGH POWER OF SAME IN C.

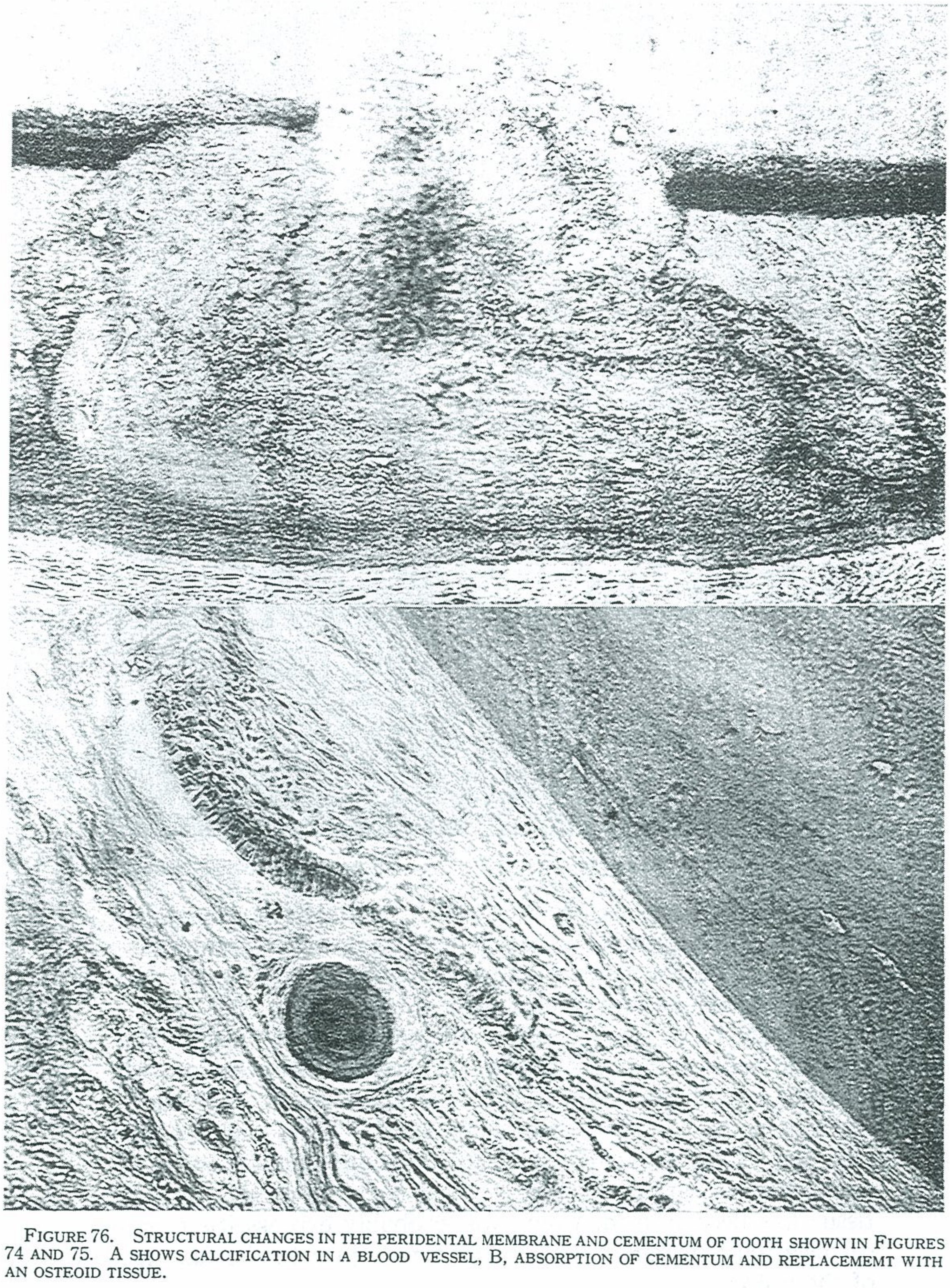


FIGURE 76. STRUCTURAL CHANGES IN THE PERIDENTAL MEMBRANE AND CEMENTUM OF TOOTH SHOWN IN FIGURES 74 AND 75. A SHOWS CALCIFICATION IN A BLOOD VESSEL, B, ABSORPTION OF CEMENTUM AND REPLACEMEMT WITH AN OSTEOID TISSUE.

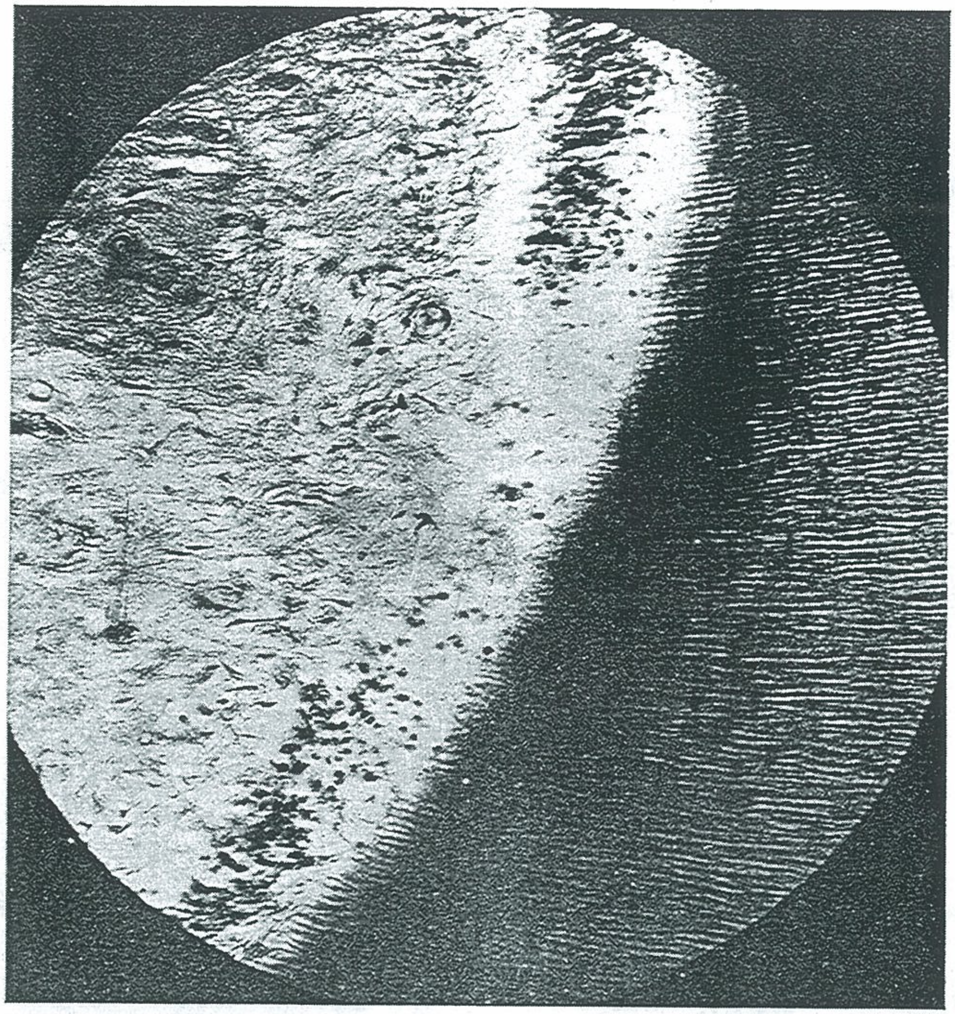


FIGURE 77 SHOWS A ZONE OF DEGENERATING OSTEOBLASTS BENEATH A ZONE OF CARIES.

eration with hyperplasia, as shown in B, in which an absorption cavity in the cementum has been later built in with osteoid structure; and these structures will always remain as scar tissues and with less than a normal defense against infection.

In Figure 77 we have a zone of the odontoblastic layer of the pulp just beneath a zone of caries. The toxic substances from the bacterial growth are penetrating into the pulp tissue. The odontoblasts in this zone have been devitalized. Their nuclei ceased to stain. Connective tissue is proliferating abundantly; and whether infection has already invaded the pulp or not, the scar tissue has been formed, which will probably express itself as a calcification and remain as a permanent injury to the function of the pulp.

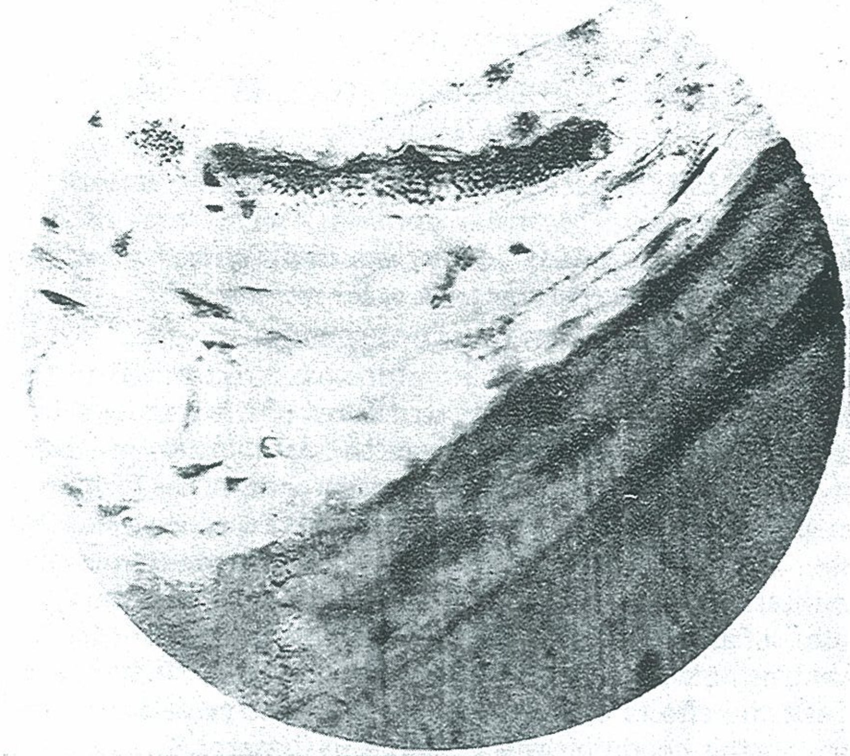


FIGURE 78. BACTERIAL INVASION AND NECROSIS IN DENTAL PULP BENEATH CARIES.

The bacterial invasion of the pulp usually progresses through the various stages of inflammation, first with congestion as part of the heroic reaction of the tissues to stamp out the invasion, then the paralyzing of the tissues and their degenerative forces with the resultant degeneration with the subsequent necrosis. At some stages of an acute pulpitis, bacterial invasion can be disclosed in the vital pulp. Such a case is shown in Figure 78, shown here stained with Giemsa stain. In this case there is marked degeneration of the odontoblastic layer, with fibrosis.

SUMMARY AND CONCLUSIONS.

This research has included the sectioning, culturing, and animal experimentation on many teeth with deep caries which were found to have abnormal reactions though nothing was disclosed by the roentgenograms; and has demonstrated to us that

> **Teeth with deep caries generally, and moderate caries frequently, already have their pulps involved from that source and cannot be dismissed as normal without further study by means other than the Roentgen-ray.**

CHAPTER VIII.

THE RELATION OF PERIODONTOCLASIA TO PULP INFECTIONS.

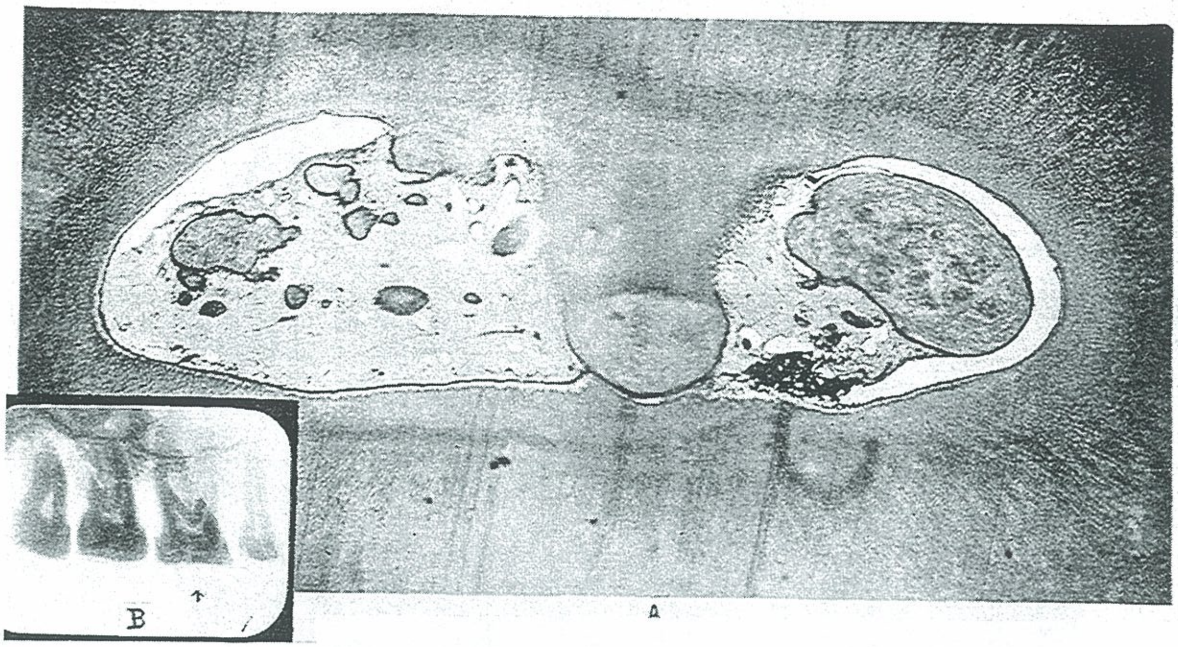
PROBLEM: Are the pulps of teeth influenced or injured by periodontoclasia (pyorrhea alveolaris) unless and until that pathological process has mechanically severed or invaded the vessels entering the tooth at the root apex?

EXPERIMENTAL AND DISCUSSION.

Dental practice and dental literature have recognized in the inflammations of the supporting structures of the teeth a direct danger of systemic involvement from the pathological processes about the tooth, but have taken for granted that the pulps are not in general involved except in very far advanced stages of this disease. These studies have accordingly been made to establish the earliest expressions of pulp involvement resulting from periodontal inflammation and infection. This particular chapter does not include the etiology of periodontoclasia. It has to do only with its effects on pulp tissue. Our interpretation of the etiology of this condition will be found in Chapter 28.

The earlier conceptions of this disease considered it as well nigh incurable, an opinion which does not generally obtain today. The newer conception regarding its amenability to treatment, however, has not recognized that even though the tissues about the tooth are put in relatively normal and safe condition, changes may have occurred in the pulp before that treatment was established, which may permanently jeopardize the pulp. Figure 79 illustrates such a case. In this case chronic periodontoclasia (pyorrhea alveolaris) had existed for years. The teeth were put under modern and efficient treatment; the disease was supposedly arrested; and the teeth became quite solid and comfortable. The old infection had, however, produced very extensive changes in the pulp tissues of these teeth. The pulp of this root had been divided by calcification which had extended across the entire pulp chamber. Very many pulp stones had formed. One is shown nearly half obliterating the remaining lumen. This tooth was still responsive to thermal change but was producing systemic disturbances. B, the insert, shows the roentgenogram of the tooth.

III-1



'IGURE 79. A DEGENERATING PULP, DUE TO PERIODONTOCLASIA. A, MULTIPLE PULP STONES, FIBROSIS; B. ROENTGENO-
PHIC APPEARANCE.

The irritation from periodontoclasia may produce calcifications within the pulp chamber in a number of different forms. Whereas the tendency is quite largely to the formation of islands, it not infrequently occurs that a fibrositis develops within the pulp tissue, which later becomes calcified. We cannot conceive of a pulp's retaining its capacity for normal functioning, when such conditions develop, for it is embarrassed both by the osteoid structure within it which lowers the vitality of the pulp tissue, and by the abundant infection present in the vicinity which is available for infecting very quickly any pulp tissue in which the defense has become lowered. Such a case is shown in Figure 80. In this it will be seen that the pulp at the apex where it was subjected to the direct irritation of the toxic products is almost solidly calcified. Some nutrient vessels passing between these fibers maintain the vitality of the coronal portion of the pulp. These teeth have an entirely different relationship to the patient and his health in the early stages, when his defense is high and before the resistance of the pulp has been lowered, and it has been in-

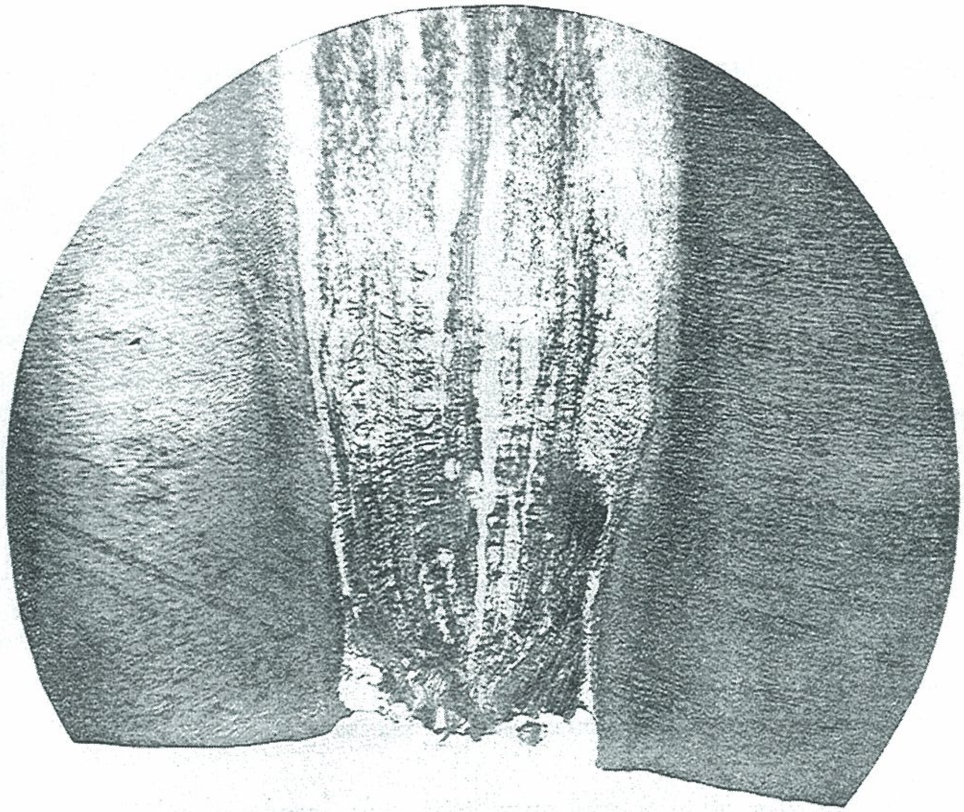


FIGURE 80. A FIBROSITIS OF THE PULP, FOLLOWED BY CALCIFICATION OF SAME.

volved, from the condition which exists in the patient with lowered defense and with this serious pulpal disease. From the patient's standpoint, and perhaps from most operators' standpoints, the tooth in the latter condition might be considered less serious because it is less loose than it had been previously. It may, however, have taken on a condition in which, with the patient's lowered defense, it may seriously jeopardize his health, or start a degenerative process in some organ or tissue of the body. This phase will, however, be discussed in later chapters.

I started investigations on this problem in 1916 while directing the activities of The Research Institute of the National Dental Association, which were carried out and reported by Dr. Katherine R. Collins,[2] which report was given before The National Dental Association in Chicago in 1917, and published in the

[2] See bibliography.

Journal of the National Dental Association, VI, 1919, 164-170 (Information to Date on Infections within the Root and Periapical Tissues;) and pages 370-373 of the same journal (Preliminary Report on Bacteria Found in Apical Tissues and Pulps of Extracted Teeth).

Following this work some special studies were made by Drs. Hartzell[3] and Henrici,[3] University of Minnesota, and reported in the Research Jnl., 1, 1919, 419-422 (The Bacteriology of Vital Pulps).

SUMMARY AND CONCLUSIONS.

From these few selected illustrations of a general pathological reaction, it is evident to us that there may be a pulp irritation resulting from periodontoclasia long before a pocket has advanced to the vicinity of the apical third of the root. This irritation may express itself in very marked structural changes which may often be of a nature (because of calcifications), which will permanently destroy the normal functioning of the pulp. That such pulps can become the source of a focal infection involving the entire system has been abundantly demonstrated and is discussed in succeeding chapters, as is also the pathology of the changes which occur in pulp tissue, cemental tissue, and dentin. The evidence at hand suggests to us that

Deep pockets of periodontoclasia generally, and shallow pockets of periodontoclasia frequently, have already injured the pulp of the tooth, which tooth may be potentially much reduced in its safety from normal though it may have very great value and efficiency for mastication, and being potentially a danger must be continually watched.

[3] See bibliography.

CHAPTER IX.

THE RELATION OF DENTAL CARIES TO SYSTEMIC DISTURBANCE.

PROBLEM: Is there a relationship between susceptibility to dental caries and to systemic involvements of the rheumatic group lesions?

EXPERIMENTAL AND DISCUSSION.

We do not find in the literature or the fundamental thought of dental practice a clearly expressed conviction indicating the existence of a relationship between susceptibility to dental caries, and susceptibility to systemic involvements, and this study has been made to determine, if possible, whether there be any such relationship. We have carefully tabulated the presence of dental caries as a dominant factor in the life, or at some period during the life, of each of the cases here reported, inasmuch as that part of a tooth, that is once lost by decay, is always lost, for even though the tooth is filled, the mark of that ravage has been written in a form that cannot be obliterated. If the teeth are absent, true they may have been lost because of periodontoclasia. If, however, the teeth are present, showing very extensive fillings or open cavities, or absent, with no evidences of periodontal disease, it can be quite accurately determined that caries has been conspicuously dominant in the mouth being studied.

By dividing the patients into groups ranging from absence of systemic affection through mild to severe, and noting the proportion of dental caries in the mouths of these various groups, we are able to study relationships between these two types of disturbances. In Figure 81, we have in the first column the names of the groups in terms of their susceptibility to systemic involvement: namely, absent, acquired, inherited one side mild, inherited two sides mild, inherited one side strong, and inherited two sides strong; in Column two, the number of severe rheumatic group lesions in fifteen patients and their families; in Column three, the number of severe and mild rheumatic group lesions in fifteen patients and their families; and in Column four, the percentage of the group of fifteen individuals having extensive caries. From

Relation of *Caries* to Susceptibility to Rheumatic Group Lesions. Fifteen Typical Families in each Group.			
Susceptibility	No. of Lesions per Family		Per cent with Caries
	Severe	Severe and mild	
Absent	16	31	**40**
Acquired	63	96	**80**
Inherited 1 side mild	144	201	**67**
Inherited 1 side strong	258	338	**80**
Inherited 2 sides mild	227	308	**93**
Inherited 2 sides strong	483	754	**93**

FIGURE 81.

this chart it will be seen that in the first, the absent group, the percentage of individuals with caries, including mild and severe, is 40; in the second, the acquired group, 80; inherited one side mild, 67; inherited one side strong, 80; inherited two sides mild, 93; and inherited two sides strong, 93.

Even more striking is the parallelism between caries and the tendency to rheumatic group lesions, as shown in Columns two and three. In Column two, the total number of severe rheumatic group lesions found in the individuals and their families, with an absent susceptibility, is 16; acquired susceptibility, 63; inherited susceptibility, one side mild, 144; inherited, one side strong, 258; inherited, two sides mild, 227; inherited, two sides strong, 483. In Column three, the combined severe and mild rheumatic group lesions, respectively, are 31, 96, 201, 338, 308, and 754.

Relation of *Caries* to Susceptibility in 681 Individuals		
Absent Susceptibility	Acquired Susceptibility	Inherited Susceptibility
73	130	327

FIGURE 82.

In Figure 82 we have divided all cases of caries into three fundamental groups of individuals: namely, absent, acquired, and inherited; and we find that in 681 individuals, 73 were classified as having absent susceptibility and caries, 130 acquired susceptibility and caries, and 327 inherited susceptibility and caries. The reader must distinguish that these are not ratios expressing caries in terms of susceptibility, but the number of individuals in the various groups who have caries. These studies show clearly that there is a very marked increase in susceptibility to caries with increased susceptibility to rheumatic group lesions whether or not either is causative to the other.

We have, accordingly, made an analysis of the different types of susceptibility for the purpose of determining whether or not caries bears a higher percentage of dominance in individuals with

Relation of *Caries* to Type of Rheumatic Group Lesions	
	Per cent
Digestive Tract	70
Internal Organs	70
Nerves	90
Rheumatism	90
Heart	100
Kidney	100

FIGURE 83.

marked susceptibility to the various types of rheumatic group lesions. In Figure 83, we have related caries to each of the following: a susceptibility to digestive tract, internal organs, nerves, rheumatism, heart, and kidney; and it will be seen that these severe affections have been found in our groups to be associated with caries in ratios running from 70 to 100 as follows: Digestive tract, 70; Internal organs, 70; Nerves, 90; Rheumatism, 90; Heart, 100; Kidney, 100.

RELATION OF CARIES TO SYSTEMIC SUSCEPTIBILITY

	Inheritance Group	No. of Families in Group	Percentage of Individuals with Extensive Caries
1	Dental Patients with *No* Developed Susceptibility	35	**51%**
2	Dental Patients with an Apparently *Acquired* Susceptibility	12 27	**91%** **78%**
3	Dental Patients with a Susceptibility and with one or both Parents acting as *Carriers Only*	16	**81%**
4	Dental Patients with a Susceptibility and with only *One Side of Ancestry*, including the Parent Involved	8	**88%**
5	Dental Patients with a Susceptibility and with *Both Sides of Ancestry*, including Both Parents Involved	7	**100%**

FIGURE 84.

In Figure 84, we have made a classification on the basis of susceptibility both where the parents acted as carriers only and where they were themselves involved from one or both sides of the ancestry, and have figures, as will be seen, ranging from 51 per cent in the absent group to 100 per cent in the inherited, both sides strong with both parents involved. This chart was made in 1919 and agrees in general with the recent findings of 1922, shown in Charts 81, 82, and 83. It shows dental patients with absent susceptibility, percentage of individuals with extensive caries, 51; acquired susceptibility, 91; another group of the same classification, 78; inherited susceptibility, one or both parents acting as carriers only, 81; inherited susceptibility, with one side of ancestry, including that parent involved, 88; with both sides of ancestry, including both parents, 100.

SUMMARY AND CONCLUSIONS.

From these data we feel compelled to conclude that one of these three conditions must prevail: First, that rheumatic group lesions are causative in the production of caries; or second, that dental caries is directly causative in the production of rheumatic group lesions; or third, that both are symptoms of a systemic condition. This will be discussed later.

We would accordingly change our previously accepted fundamental which concluded that there is no relationship between caries and systemic involvements to the following:

> **Susceptibilities to dental caries and to rheumatic group lesions are proportional, whether as cause and effect or as related symptoms.**

CHAPTER X.

THE RELATION OF PERIODONTOCLASIA TO SYSTEMIC DISTURBANCE.

PROBLEM: Is there a relationship between susceptibility to periodontoclasia (pyorrhea alveolaris) and susceptibility to systemic involvements of the rheumatic group lesions; or stated differently, is it not true that with an increase of susceptibility to periodontoclasia there is a marked increase in sustibility to rheumatic group lesions?

EXPERIMENTAL AND DISCUSSION.

In undertaking the study of this problem, as herewith outlined, we seem to be wasting time with a fundamental that is so generally accepted that it must of necessity be correct. Surely with the increase in prejudice of various affections, from a relative indifference to dental decay and some apprehension for apical involvements and putrescent pulps, we come by progression, in the thought of the laity, and we think of the professions, in so-called periodontoclasia to the most dreaded of dental lesions. Many of the laity are as alarmed over the thought of periodontoclasia's having attacked their mouths, as they would be over some terrible affliction like leprosy. Such a firm conviction, it would seem, can only be born in fact. Probably few, if any, headlines in the advertisements of current literature will be so largely read as the advertisements for cures for periodontoclasia. We have, accordingly, taken up the study of this scourge with an expectation amounting to a confidence that our data will show conclusively that an increase in susceptibility and dominance of this affection will be continually accompanied by an increase in severity and susceptibility to the rheumatic group lesions.

In our careful analysis of these 681 cases, we have found 579 have had rheumatic group lesions, and only 102 were without rheumatic group lesions; and of the 681, 508 have extensive caries and only 155 periodontoclasia.

Relation of Periodontoclasia to Susceptibility to Rheumatic Group Lesions.
Fifteen typical families in each group. Group Two.

Susceptibility	No. of lesions per family		Per cent Caries	Per cent Periodon-toclasia
	Severe	Severe and mild		
Absent	16	31	40	**40**
Acquired	63	96	80	**33**
Inherited 1 side mild	144	201	67	**33**
Inherited 2 sides mild	227	308	93	**20**
Inherited 1 side strong	258	338	80	**20**
Inherited 2 sides strong	483	754	93	**0**

FIGURE 85.

In Figure 85, we have related periodontoclasia to systemic involvements in association with dental caries as presented in Figure 81 of the previous chapter, No. 9. These data show in the last column, Group 1, that the percentage with periodontoclasia was 40, corresponding to the percentage with severe and mild caries; in Group 2, acquired, periodontoclasia 33%, caries 80%; Group 3, inherited one side mild, periodontoclasia 33%, caries 67%; Group 4, inherited two sides mild, periodontoclasia 20%, caries 93%; Group 5, inherited one side strong, periodontoclasia 20%, caries 80%; and in Group 6, inherited two sides strong, periodontoclasia 0%, and caries 93%; and in the entire 681 cases, periodontoclasia 23% and caries 75%. Columns three and four show respectively the total number of severe, and severe and mild lesions of the groups consisting of fifteen individuals and their families.

In Figure 86, in the chart made in 1919, Group 1, Patients with no developed susceptibility, severe periodontoclasia 23%, extensive caries 51%; Group 2, Acquired susceptibility, periodontoclasia 33%, dental caries 91%; Group 3, Inherited susceptibility with one or both parents acting as carriers only, periodontoclasia 25%, caries 81%; Group 4, Inherited susceptibility strong on one side of ancestry, including that parent, periodontoclasia 0, extensive caries 88%; Group 5, Inherited susceptibility with inheritance strong on both sides, including both parents, periodontoclasia 0, extensive caries 100%. By referring back to Figure 44, Chapter 4, the relationship of these figures to the increasing dominance of rheumatic group lesions will be readily seen.

Analysis of the 681 cases shows that in that number 167 had periodontoclasia, distributed in the three groups—absent, ac-

RELATION OF PERIODONTOCLASIA TO SYSTEMIC SUSCEPTIBILITY

Group One

Group	No. of Families in Group	Percentage of Individuals with: Extensive Caries	Percentage of Individuals with: Periodontoclasia
1 Dental Patients with *No* Developed Susceptibility	35	51%	**23%**
2 Dental Patients with an Apparently *Acquired* Susceptibility	*12 *27	91% 78%	**33%** **26%**
3 Dental Patients with a Susceptibility and with one or both Parents acting as *Carriers Only*	16	81%	**25%**
4 Dental Patients with a Susceptibility and with only *One Side of Ancestry*, including the Parent Involved	8	88%	**0**
5 Dental Patients with a Susceptibility and with *Both Sides of Ancestry*, including Both Parents Involved	7	100%	**0**

* Two groups.

FIGURE 86.

RELATION OF PERIODONTOCLASIA TO SYSTEMIC SUSCEPTIBILITY

	Susceptibility: Absent	Susceptibility: Acquired	Susceptibility: Inherited
Total No. of Patients	102	174	405
No. of Patients having Periodontoclasia	49	51	67
Percentage of Patients having Periodontoclasia	**48**	**29**	**16**

FIGURE 87.

quired, and inherited—in the following percentages: Absent susceptibility, 48%; acquired susceptibility, 29%; and inherited susceptibility 16%. (Figure 87.)

In Figure 88, we have made groupings in accordance with the type of lesion as we did in the previous chapter on caries. Digestive tract, periodontoclasia 20%, caries 70%; Internal organs, periodontoclasia 30%, caries 70%; Nerves, periodontoclasia 30%, caries 90%; Rheumatism, periodontoclasia 10%, caries 90%; Heart in patient and family, periodontoclasia 10%, caries 100%; Kidney, periodontoclasia 10%, caries 100%; Rheumatism and heart, periodontoclasia 0, caries 100%.

Relation of Periodontoclasia to Type of Rheumatic Group Lesions		
	% Caries	% Periodonto-clasia
Digestive Tract	70	20
Internal Organs	70	30
Nerves	90	30
Rheumatism	90	10
Heart	100	10
Kidney	100	10

FIGURE 88.

In Figure 89, we have arranged a group of typical illustrations of gingival affections. In these, we have selected, as nearly as possible, the same irritant in the different classes of cases (and that a well known and all too common cause) namely, the impinging and irritating edge of bad fillings and open contacts producing food packs. In A, we see very extensive absorption of alveolar bone; in B, much less marked; in C, a slight area of absorption surrounded by an area of condensed bone; in D, practically no absorption of alveolar bone and a marked zone of condensed bone. When we relate these to susceptibility to the rheumatic group lesions, we find that A tends to be typical of those in the absent group; B and C, those in the acquired or mildly inherited groups; and D, those in the strongly inherited and strongly involved group. A survey of the illustrations that are used throughout the various studies, as in all the preceding chapters, will show how generally these conditions obtain.

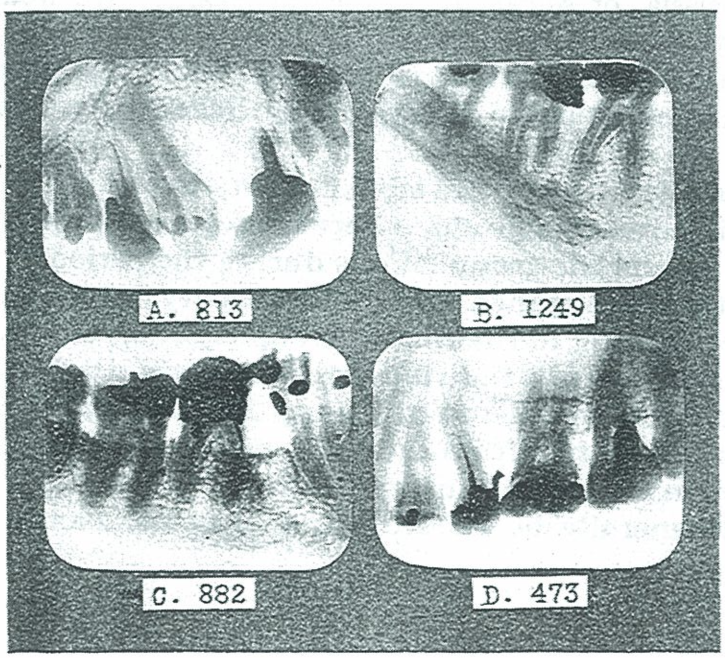


FIGURE 89. FOOD PACKS AND GINGIVAL IRRITANTS, WITH DIFFERENT TYPES OF REACTION.

In general, it is not true that, with an increase of susceptibility to periodontoclasia, there is an increase in susceptibility to the rheumatic group lesions; but on the contrary, those with no susceptibility to rheumatic group lesions, whether inherited or acquired, have a larger percentage of periodontoclasia (pyorrhea alveolaris) than any of the groups, ranging from mild to severe, of susceptibility to rheumatic group lesions. Our interpretation of these new and important phenomena will be given in the development of this general interpretation, in their places in subsequent chapters.

SUMMARY AND CONCLUSIONS.

From these data, we find an apparent contradiction to the thought and teaching of dental and medical science that is nothing short of bewildering. We have gone over our figures time after time to see if we have not made a mistake, but do not find such. Figure 86 made in 1919, and Figures 85 and 87 made recently, 1922, were made by different individuals. Those making the last charts not only did not know the figures of the previous study, but did not know that such a comparison had ever been worked out.

We are, apparently, dealing here with fundamental new truths which will have a great bearing on the explanation of the confusion which has been so dominant throughout the entire history of dental and medical science; and no conundrum has more completely and continually baffled solution than has the etiology of periodontoclasia, or so-called pyorrhea alveolaris. We would, accordingly, re-state the accepted fundamental, that with an increase of susceptibility to periodontoclasia, there is a marked increase in susceptibility to rheumatic group lesions to the following:

Individuals with marked susceptibility to periodontoclasia have, as a group, a decreased susceptibility to the rheumatic group lesions during the period of its active development (In its secondary stages it may contribute to rheumatic group lesions); or expressed otherwise, individuals with a very marked susceptibility to rheumatic group lesions tend, in general, to be free from extensive periodontoclasia; and when rheumatic susceptibility does develop, it would generally be classed as an acquired factor.

CHAPTER XI.

RELATIONSHIPS BETWEEN PERIODONTOCLASIA (PYORRHEA ALVEOLARIS) AND THE EXTENT OF PERIAPICAL ABSORPTIONS.

PROBLEM: Is there a relationship between the root end expression of irritation and the gingival expression of irritation; or otherwise expressed, is there in a given case no relationship between the size of apical abscesses from infected roots and extent of periodontoclasia?

EXPERIMENTAL AND DISCUSSION.

We do not find in the literature or the opinions of dental practice, evidence other than would tend to demonstrate or verify that there is no connection between the extent of the absorption, in case of apical abscess, and the extent and type of gingival absorption, as occurs in so-called pyorrhea alveolaris. These studies have been made to determine whether or not there be any such relationship.

In Figure 90, we have in the teeth of the upper arch (upper right first molar, upper left biscuspids and first molar) evidences of extensive periodontoclasia. The supporting alveolus has been very extensively destroyed, the gingival crest completely obliterated, and the lamina dura disintegrated to, or nearly to, the apices of the roots. In the lower arch will be seen a lower incisor and a cuspid root, each with periapical involvement, not of moderate degree but of great extent. This patient is fifty-two years of age, has never had any of the rheumatic group lesions, and not only has excellent health but carries an enormous overload of care and work all the time with apparent ease.

In Figure 91 we have a case with a marked tendency to periodontoclasia that has existed for many years but has passed from the active to the chronic stage. The patient is suffering at this time from an abscess in her scalp which she said seemed like a boil, though it has persisted in discharging for many weeks. She is fifty-seven years of age and has carried all this infection without injury until recently, when the above trouble developed, and also eye trouble. The upper left first bicuspid shows distinctly a

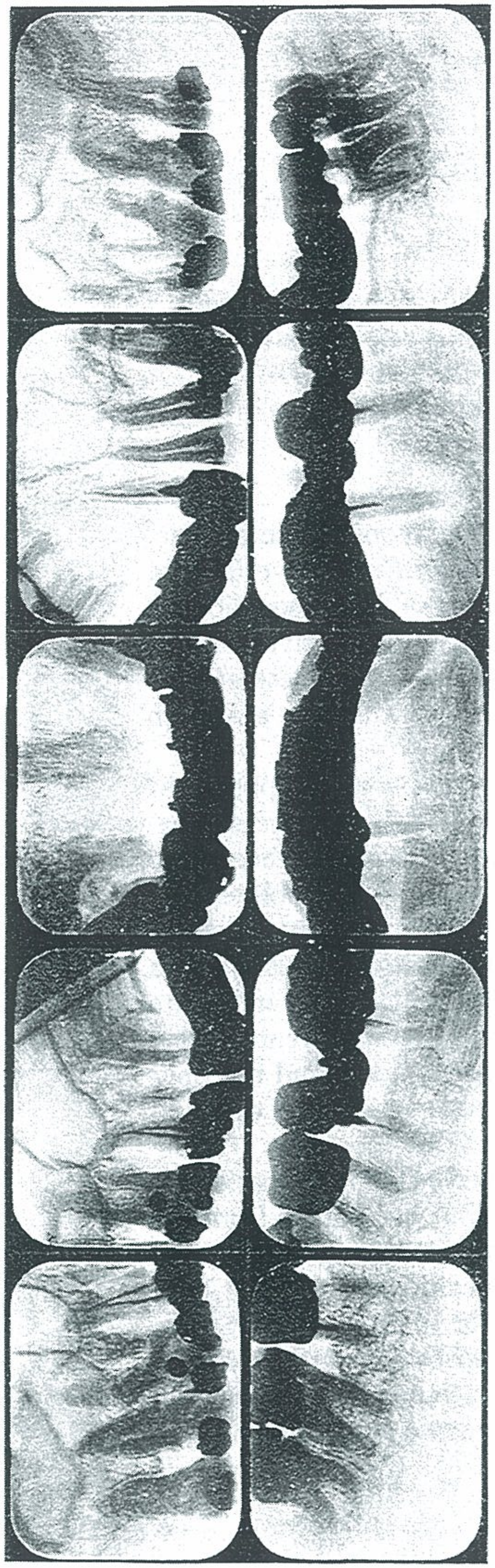


FIGURE 90. EXTENSIVE ALVEOLAR ABSORPTIONS IN BOTH GINGIVAL AND APICAL TISSUES, ASSOCIATED IN THE SAME INDIVIDUAL.

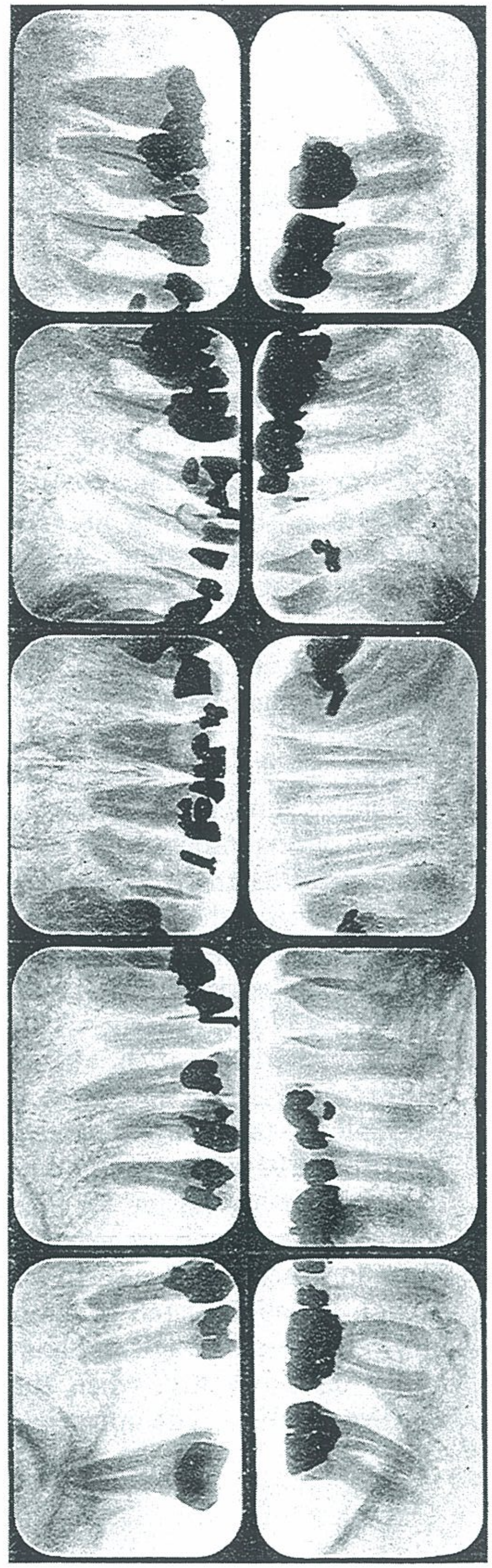


FIGURE 91. LONG CONTINUED GINGIVAL AND APICAL INFECTION, WITH THE RESISTANCE BREAKING.

zone of condensing osteitis about the rarefying from one view. This is also shown mildly about the extensive apical infections of the molars. This patient has reached the time when she can no longer defend herself against this infection. This patient has had, as will be seen, evidence of very extensive absorption about the molar roots. She has been entirely free from all rheumatic group lesions during her entire lifetime, until the beginning of the recent disturbances. These, incidentally, appeared simultaneously with a very severe overload occasioned by the illness and death of her sister, whose fatal illness had been caused by a septicemia resulting from a dental infection which had remained in a chronic condition for many years, but which, when her resistance went down at seventy-two years of age proved fatal after an illness of about six weeks.

In Figure 33, we have again much cause for irritation and practically no absorption of alveolar bone, no apparent tendency to periodontoclasia, and again pulp chambers with much cause for irritation and exceedingly little disturbance about the root ends.

When we relate these to the patient's susceptibility to rheumatic group lesions, we find in Case 1, practically no susceptibility and very marked periodontoclasia and extensive periapical absorption; in Case 2, mild periodontoclasia and apical absorption, and mild susceptibility to rheumatic group lesions, a condition frequently seen in acquired susceptibility; and in Case 3, no tendency to periodontoclasia and slight periapical absorption, and a strong tendency to rheumatic group lesions.

These three selected illustrations are not presented as being sufficient for final conclusions, but simply as typical illustrations; and an analysis of the illustrations of any of the chapters will show the same general principle. It must be kept in mind, however, that in order to make a comparison of reactions we must know that both the gingival and apical irritants were operating at the same period in the patient's life; otherwise, there may be an apparent contradiction, as, for example, if periodontoclasia were progressive during a period of high resistance, which for any cause was reduced so that the same irritations did not produce a continued progression of the absorption process, a condition of acquired susceptibility to rheumatic group lesions may develop, during which the defense is definitely lowered and consequently the reaction much less acute, in which state, caries may develop with pulp involvement, and with the characteristic apical reaction of an individual, without susceptibility to periodontoclasia, signifying simply a diminished or lost capacity for normal reaction.

SUMMARY AND CONCLUSIONS.

There is apparently a very definite relationship between these various affections and reactions, which we have discussed in later chapters; and we would, accordingly, restate the premise as follows:

> **There is a direct relationship between tendency to absorption of alveolar bone in response to irritation, whether at the gingival border or at the root apex; and individuals with extensive periodontoclasia have much more extensive areas of absorption at the apices of infected roots than do patients without a tendency to periodontoclasia.**

CHAPTER XII.

THE RELATION OF THE EXTENT OF ABSORPTION TO THE DANGER.

PROBLEM: Is the danger proportional to the evidence of absorption of bone about the apex of a suspected tooth?

EXPERIMENTAL AND DISCUSSION.

We perhaps should be expected to apologize for the presumption that there may be any question as to the truth of this so generally accepted premise. Probably in every city of any size in the country there are to be found would-be dental diagnosticians whose preparation presumably makes them competent to judge the difference in size of different areas with the naked eye, without a mathematical calculation, with considerable definiteness, but who have no qualification which would make it possible for them to interpret pathology in any other terms than dimensions. We must state frankly that this particular study was not inspired by any antagonism to these persons as a group, but solely in the interest of humanity. The fundamental basis for the procedure in the great majority of the laboratories of the country (and it is pathetically true that there is so great a majority of diagnostic institutions of the country undertaking to do dental diagnosis without the assistance of a trained dental pathologist) is to look in the roentgenogram for an area of radiolucency, and, if one be found, to judge its probable volume and extent, which factors alone determine whether that tooth is or is not potentially a possible source of danger to that patient; and in direct proportion to the volume will the danger be interpreted to be. We have, accordingly, classified fourteen hundred individuals in terms of their type and extent of absorption, both apparent and actual, in comparison with the presence and absence of systemic involvements.

In Chapter 5, when discussing the relationships between the characteristics of local structural change about infected teeth, and tendency to systemic involvement, I presented a series of cases as characteristic of the groups. While these did show those facts very strongly, and in accordance with the large number of cases involved in the various groups, they were not comparable

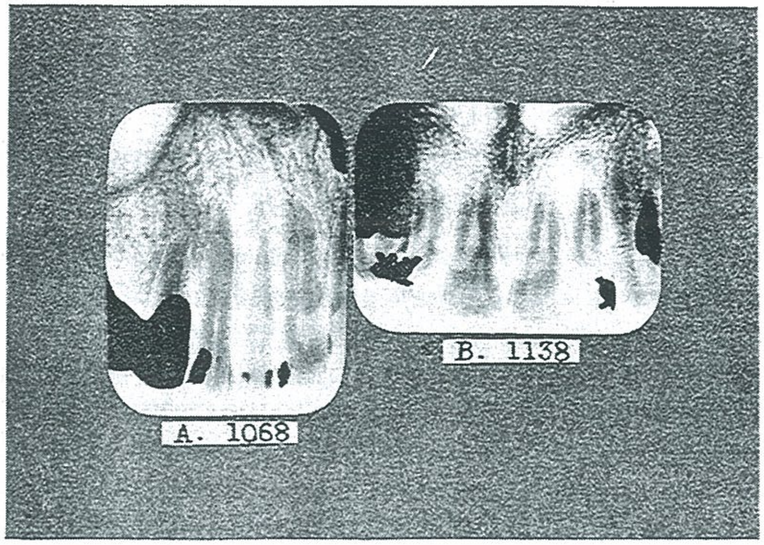


FIGURE 92. LATERALS WITH PUTRESCENT PULPS. ABSENT SUSCEPTIBILITY.

infections in the sense that they were either the same type of infection or the same tooth. For example, areas of disturbance about root-filled teeth represent not only different sizes of teeth and numbers of roots, but a different capacity for infection within the tooth structure. Accordingly, if we can take a definite quantity of infection, such as would obtain with a lateral tooth with a non-vital pulp, and study it comparatively in different individuals, it becomes immediately possible to make comparisons with greater safety. In Figures 92, 93, 94, and 95, we have selected a series of infected laterals, which teeth had evidence of being non-vital for a considerable time.

Since the problem involved in this special research is to determine the relationship between the systemic involvement and the local expression, and whether the latter in its extent is directly proportional as a danger with that extent, it becomes immediately necessary that our study include a very careful analysis of the patients having, or not having systemic involvement, and of the same having evidence or proof of the dental focus as being a principal cause by the patient's being definitely relieved of severe symptoms by the removal of that dental focus.

When, now, we make a careful study of the systemic reactions and check them against these tissue expressions, we find that those in Figure 92 are free, and have always been free, from systemic involvements of the rheumatic group; in other words, belong to our group which we spoke of as absent susceptibility. Those in Figure 93, we find, have both been free during their lifetime until

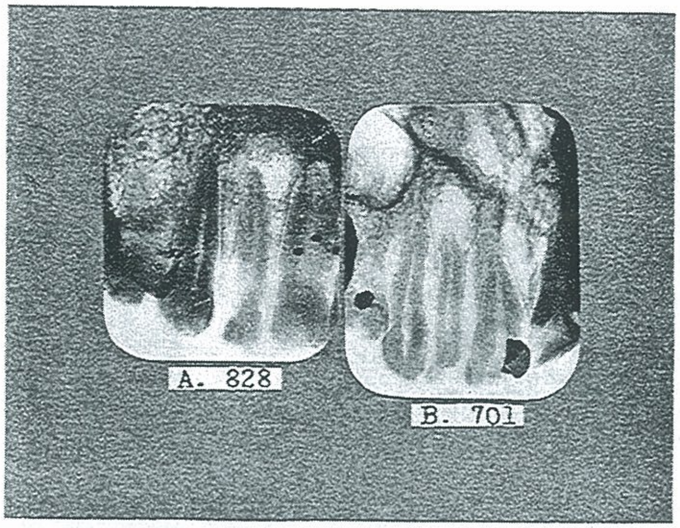


FIGURE 93. LATERALS WITH PUTRESCENT PULPS. ACQUIRED SUSCEPTIBILITY.

recently when they have had a sudden severe attack which has been entirely and permanently relieved by the removal of this dental focus. These patients belong to, and these illustrations are taken from, the group having an acquired susceptibility according to their systemic condition. Those in Figure 94, similarly belong to those with mildly inherited susceptibility; and those in Figure 95, with very little evidence of absorption about apices of infected teeth, all belong to the group which, according to their histories, have had serious involvements and have had a strong inherited susceptibility from both sides.

A clinical comparison of these individuals who represent different large groups reveals that those in Figure 92 have all the infection of a putrescent lateral pulp as have all the others, notwithstanding the fact, that they have never had any of the degenerative diseases which we speak of as rheumatic group. Fortunately these two individuals represent a large group of the total of society. During long periods of their lifetime their defense is ample to protect them against this and their various overloads and, as is so frequently seen, protects them against many such infected teeth. These two individuals are selected from the group of fifteen presented in Chapter 4, classed as those with absent susceptibility. The average number of severe rheumatic group lesions for the entire family represented by this group was found to be 1.07, and the severe and mild rheumatic group lesions 2.07. It will be remembered that, on an average, there are fifteen individuals included in the family circle of the ancestry.

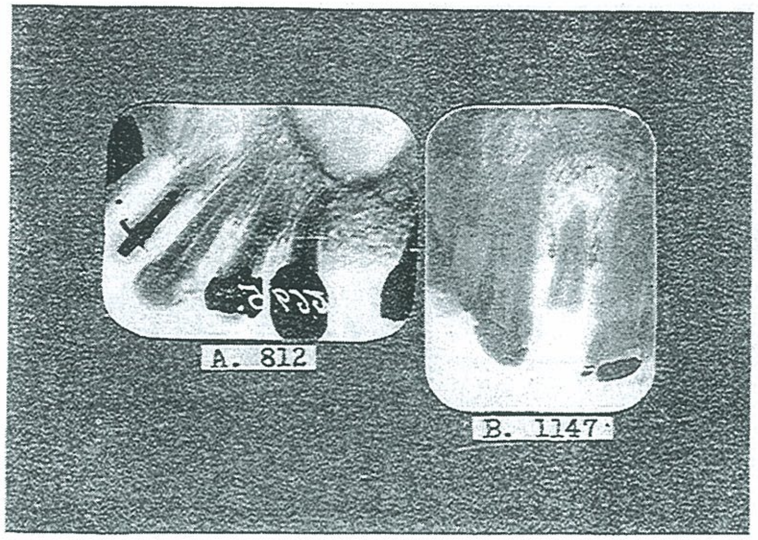


FIGURE 94. LATERALS WITH PUTRESCENT PULPS. MILDLY INHERITED SUSCEPTIBILITY.

The next two laterals, shown in Figure 93, are taken from the group with an acquired susceptibility. A is the lateral of a patient who has had a very unusually high defense all his lifetime until he was a victim of influenza. This lateral tooth had had a fistula for some time. With the onset of his Flu and following it, he suffered very severely from neuritis. This tooth was treated and root-filled and an apical curettement made, following which his neuritis promptly disappeared. Figure 93-B shows a different condition structurally in that there is a distinct tendency to the formation of limiting dense bone surrounding the rarefied area. This patient had been suffering for several weeks from rheumatism. This tooth was extracted and carried on a pinlay on the lingual surface of the cuspid. Her rheumatism seemed to be completely relieved by the removal of this tooth. These two individuals are typical representatives of the group which we have termed as having an acquired susceptibility. They, as a group, have had a high defense until an overload has broken them sufficiently so that they become a prey to their own infections, which previously they had carried without apparent injury. With the removal of their overloads or their dental infections, and particularly with the removal of both, they tend readily to come back to their own high normal. The individuals of this figure were selected from the group of fifteen, representing those with an acquired susceptibility, in which it was seen that the average total severe rheumatic group lesions of the entire immediate and ancestral relatives was 4.20, and the severe and mild 6.40.

Figure 94, A and B, shows two individuals with a mildly inherited susceptibility. They have tended more or less readily to have mild rheumatic group lesions with overload and infection. Whereas in the two former groups during the period of high defense, teeth tend readily to become tender and apical infections of this type nearly always have fistulae, in this group there is much less apical absorption for a given infection, less tendency to have a fistula, and less tendency to tenderness. With the removal of these dental infections which were of long standing, the results were as follows:

The patient represented in A, a married woman, age twenty-nine, had a severe nervousness following the death of her husband from influenza, from which she would cry on the slightest provocation, and had been incapacitated from her work, since she had to earn to support their four year old child. With the removal of her dental infections there was a rapid marked improvement in all of these nervous symptoms. She was able to take up her duties again and the world looked entirely different. However, just as grief is one of the greatest depressants and overloads, joys are among the most potential stimulants; and it is possible that one of the contributing factors to her vivacity and rapid return to health (although it is my belief that it came after this change in her physical condition) may have been the fact of a change in her life's responsibilities by a new engagement and a remarriage. Indeed, it is entirely probable that this happy change in her life could not have come for a long time, if at all, if it had not been by the remarkable change that was wrought by the removal of her dental infections. In these cases we have a combination of acquired factors superimposed upon the normal inherited tendencies. From her roentgenograms we would readily see that this woman's condition is indicated to be one of good reacting power and probably high defense. While this patient suffered from nervous breakdown, one of her sisters had a similar though less severe experience, and her mother and one of her mother's sisters had suffered very severely from nervous troubles. The health record, however, on the father's side was excellent, there having been no lesions of the rheumatic group recorded.

Figure 94-B is a case of a man forty-seven years of age, who has ordinarily had excellent health, but has recently suffered very severely from neuritis in his shoulders. With the removal of his dental infections, which were very extensive and of long stand-

ing, his neuritis entirely disappeared and has not returned, even in mild form for two years. This is a case where the factor of overloads was very important. This man had had very extensive dental infections. With the depression following the war, his business anxieties became very great. Rheumatism and neuritis had been mildly inherited from both his father and mother, and we should, accordingly, consider his case as being one of an acquired susceptibility superimposed upon a mildly inherited one. This is also indicated by the type of dental pathology revealed about his infected teeth, there being a zone of marked condensing osteitis surrounding the rarefying. The individuals of this group tend much more readily to have breaks from overloads than those in the two preceding. The susceptibility group to which they belong is mildly inherited, in which group of fifteen families we have found the average number of severe lesions per family group, inherited one side mild, to be 9.6, and the severe and mild, 13.4; and inherited two sides mild, 15.3 and 20.5.

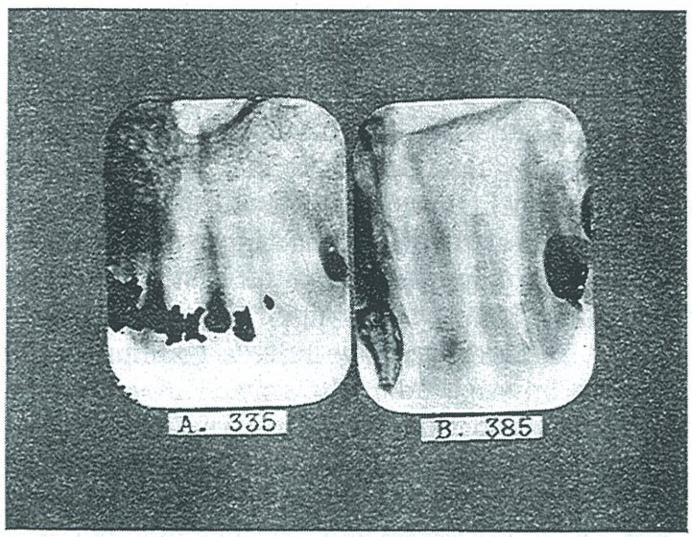


FIGURE 95. LATERALS WITH PUTRESCENT PULPS. STRONGLY INHERITED SUSCEPTIBILITY.

Figure 95, A and B, shows two cases from the strongly inherited susceptibility group of fifteen (in which group we have the average number of severe lesions per family group, inherited one side strong, to be 17.2, and the severe and mild, 22.5; and inherited two sides strong, 32.2 and 50.2) who are quite typical of that group. They are both young people. Each has a putrescent pulp in a lateral tooth. There is no fistula and never has been. The teeth are not sore and never have been, or but slightly. There is a very slight area of absorption about the apices of the roots. A has a type of nervous breakdown and rheumatism, and has been incapacitated from her work, or nearly so, approximately half the time for several years. This is only one of several dental infections that were removed, one at a time; and after complete removal of her dental infections, she has had very much better health and has scarcely lost a day from her work because of ill health for three years. B had a very severe heart involvement and rheumatism, which had completely incapacitated her from her work for several months. With the removal of this and other dental infections she very soon gained twenty pounds. Her heart is apparently normal in function again and her rheumatism entirely disappeared so that for several years she has not lost any time from this cause. A had previously had an operation for the removal of tonsils and appendix without beneficial result.

Each of these two individuals comes from a family with very marked susceptibility to the lesions from which they are suffering. Both of the parents of B died in the fifties of heart involvement after years of incapacity. A has some heart involvement. Her brother died of heart involvement as did her father, who also suffered severely from rheumatism. They are quite true to type for their class. The type of reaction about the apices of the roots shows progressively less destruction of alveolar tissue about the apices of the infected pulpless teeth; and with the decrease in apical absorption and the absence of fistulae, there is an increase in the susceptibility to systemic involvement, or a decrease in the patient's safety. This is precisely the opposite to what is taken for granted by the casual observations and as the general basis for the interpretation of dental roentgenograms.

And just at this point I wish to anticipate a misapprehension that is likely to occur. Individuals reading this must not quote this statement except in connection with the premises on which these observations are made and the comparisons given: namely, that with a given dental infection the apical reaction is progressively less with the decline of that individual's defense against that infection. In all these individuals, or more specifically stated, in any individual of any of these groups a larger quantity of infection will produce a greater local reaction of the type characteristic of that individual than a smaller quantity. Or otherwise stated, in any individual of any of these groups a tooth, with the quantity of infection so large as that involved with an entire infected pulp in addition to the infected dentin, will have a larger zone of disturbance about the apex of that tooth, than about some other tooth with a partial root-filling, and these two greater than some other tooth with an excellent root-filling, and all these three more than a tooth with a very recent suitable sterilization and root-filling. Accordingly, an individual with a very high defense and a very excellent reaction will show as much apical disturbance about a tooth with but slight infection, as an individual with a very low defense will about a tooth with a very large quantity of infection. We have, then, two important factors. A given amount of disturbance at a root apex will represent two entirely different quantities of infection with these different groups, and the danger to the individual having that given quantity of apical disturbance will be an entirely different matter, if she or he belong to the group having a very high defense, from that if they belong to a group with a very low defense, and these again will be directly related to their overloads.

We have made a careful study of the data disclosed in our records of case histories and find that in 681 selected cases having

RELATION OF LOCAL TO SYSTEMIC

Susceptibility Class	Local Expressions of Dental Infections						No. of Lesions per Group	
	Caries	Pyorrhea	Open	Locked	Rarefying	Condensing	Severe	Severe & Mild
Averages of Total No.	75	23	25	75	32	26	170	245
Groups of 15								
Absent	40	40	40	60	67	0	16	31
Acquired	80	33	33	87	33	20	63	96
Inherited 1 side mild	67	33	40	87	33	20	144	201
Inherited 2 sides mild	93	20	27	80	40	33	227	308
Inherited 1 side strong	80	20	20	80	27	33	258	338
Inherited 2 sides strong	93	0	0	87	7	67	483	754

FIGURE 96.

adequately complete histories, the average number of open dental infections, by which we mean infected pockets at the gingival margin of the teeth extending at least a third of the distance at the apex, or apical abscesses with fistulae, the average percentage of all types is 25, and the average percentage with apical infections without fistulae, termed locked infections, 75, the percentage with rarefying osteitis 32, and the percentage with condensing osteitis 26. (See Figure 96.)

When we select groups of fifteen from each of the six different degrees of susceptibility and compare the percentages for these four local conditions, we find for the absent group: open 40, locked 60, rarefying 67, condensing 0; the acquired susceptibility group: open 33, locked 87, rarefying 33, condensing 20; inherited susceptibility, one side mild: open 40, locked 87, rarefying 33, condensing 20; inherited susceptibility, two sides mild: open 27, locked 80, rarefying 40, condensing 33; inherited susceptibility, one side strong: open 20, locked 80, rarefying 27, condensing 33; inherited susceptibility, two sides strong: open 0, locked 87, rarefying 7, condensing 67.

From this it will be seen that there is a progressive change in the type of reaction directly in proportion as the susceptibility changes; the condensing osteitis progressing from 0 in the absent group to 67 per cent in the inherited two sides strong; and the rarefying osteitis decreasing from 67 per cent in the absent to 7 inherited two sides strong; and, similarly, the open decreases from 40 in the absent to 0 in the strongly inherited, and the locked increases from 60 in the absent to 87 per cent in the strongly inherited.

When we compare these figures with those in the next column —namely, the clinical expression of the rheumatic group lesions— called the Number of Lesions per Group, expressed as severe, and severe and mild, we have for the group with absent susceptibility as a total number of severe lesions in all or each one of fifteen individuals and all the members of their families—16, severe and mild—31; acquired susceptibility—63 and 96; inherited one side mild—144 and 201; inherited one side strong—258 and 338; inherited two sides mild—227 and 308; and inherited two sides strong—483 and 754.

SUMMARY AND CONCLUSIONS.

A study of the 681 selected cases from the 1400 here reported indicates to us that the danger is not in proportion to the area of apparent or actual absorption about root apices, and that it tends more nearly to be in a reverse order to the area of absorption produced by a given infection. Our interpretation of this is given in succeeding chapters.

This is a most important and striking discovery, and particularly so since the individuals recording these data and summarizing them had not the remotest conception of what was developing or its significance. It is a most singular fact that the presence of condensing osteitis in these groups should progress practically in direct proportion to the susceptibility to rheumatic group disturbances in the other members of the family as well as in direct proportion to the susceptibility in the individual. The former is a fact of profound significance. It is a very different thing that the type of local dental pathology should be related to these qualities in the other members of his or her family. We are therefore dealing with common effects of general causes.

We would therefore state the fundamental on which this research is based as follows:

> **Since different people react differently through a wide range to a given infection, the quantity of the absorption is not a measure of the danger but, on the contrary, it may be, and frequently is, true that the patient suffering severely from a systemic reaction caused by a dental infection, shows very little absorption compared with that which the same dental infection would produce in a patient with ample and high resistance.**

CHAPTER XIII

THE NATURE OF THE DISCHARGE FROM A DENTAL FISTULA.

PROBLEM: Is the discharge from a chronic dental fistula badly infected and very poisonous?

EXPERIMENTAL AND DISCUSSION.

Pus has been synonymous in the thinking and teaching of dental problems with concentrated virulent infective organisms, so much so that until recently it has been thought that teeth with fistulae draining apical abscesses were not only the most dangerous but, until the advent of the Roentgen-ray, constituted nearly the entire group of so-called abscessed teeth, and therefore the cure of the pathological lesion in question depended entirely upon the skill of the operator. The proof that the abscess was cured was the closing of the fistula.

Years of experience in oral surgery revealed that practically all of the teeth that were removed as border-line, proved to be seriously involved at the apex, though without a fistula. For years it was deemed that teeth were not bad enough to be border-line if they did not have a fistula; but if they were not bad enough to have a fistula, it was taken for granted that they were all right, that they were of necessity not badly infected; and those with fistulae were presumed by many operators to be incurable or not, depending solely upon the skill of the operator, and particularly on the magic secret formula which he possessed, most of which teeth required many and long treatments according to the ideals of those days. This phase of the problem, namely medication, is discussed in the next chapter. It is possible that even today it is well nigh universally believed by the members of both the medical and dental professions, and for that matter the laity also, that teeth with flowing fistulae have the same only because they are more severely and extensively infected than the teeth that do not have fistulae. In other words, the presence or absence of a fistula is a measure of infection.

This problem has to do with the nature of the material flowing from chronic dental fistulae. The method of procedure has been

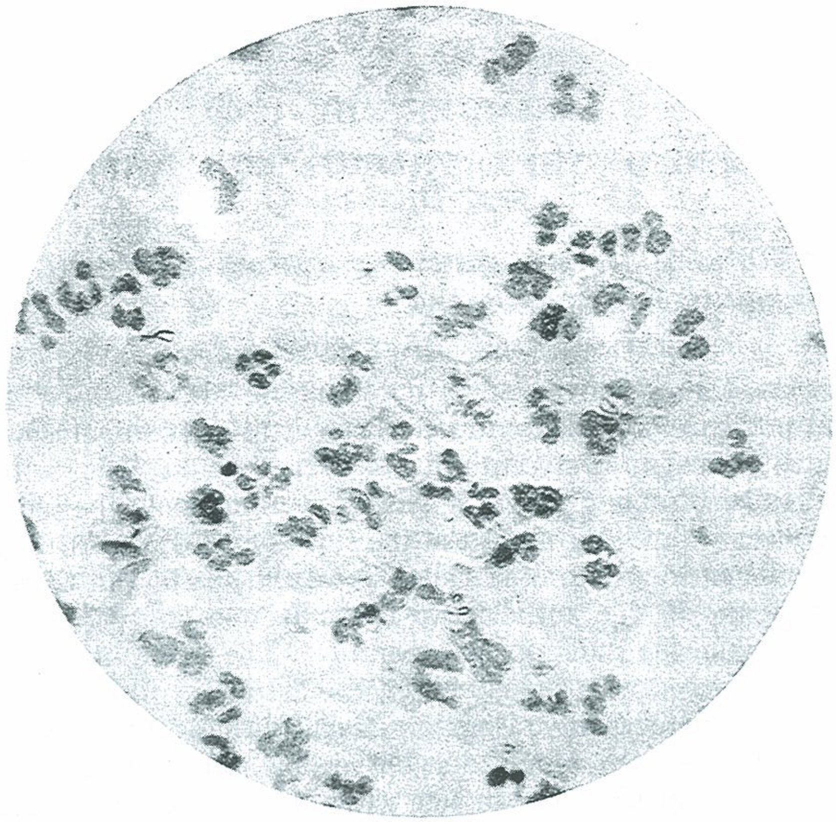


FIGURE 97. DISCHARGE FROM A FISTULA OF A PATIENT WH AS A HIGH DEFENSE.

to take by aspiration, material from the fistulae and study its contents both in living and stained mounts. Since this material is supposed to be made up, in addition to the leucocytes, with living and dead organisms, stains were expected to show large numbers of organisms free in the fluid. Figure 97 is typical, being a Wright's stain, and it will be noted that while there are many polynuclear leucocytes, only a small portion of them contain phagocyted organisms, and there are practically no organisms outside the leucocytes. Many phagocyting leucocytes have only 1 or 2, a few have 5 to 10, but the great majority do not have any. Plating of aspirated contents from agar media frequently reveals only scattered colonies, and not infrequently aspirated material from dental fistulae, cultured in suitable media, proves sterile.

Let us now study the patient from whom the pus was taken from this flowing fistula. The roentgenograms of the teeth are shown in Figure 98. She is forty-six years of age, with a very unusual physique, in that she has never had any of the rheumatic group lesions, nor have any of the members of her family. She has several poorly filled root canals, which if they had been in the mouth of an individual with low defense and marked rheumatic group susceptibility, would have had very little apical disturbance and no fistula. But in her case practically every imperfectly root-filled tooth shows extensive apical alveolar absorption. She has fistulae both in the hard palate and through the buccal mucous membrane, has frequent swelling, will feel very miserable for a day or two while fighting the acute attack, and with its subsidence rebounds quickly to her normal high defense and excellent health. She is relatively safe so long as she has this undisturbed high defense; and since her normal by inheritance is so high, only a severe overload will break it. Any of the severe overloads which we discuss later, such as influenza and pregnancy, would tend to make her splendid defense less complete and her factor of safety would be definitely reduced, perhaps to the danger line. This type of individual should live to be ninety or one hundred, with good health to the end. She may develop nephritis, hypertension, or other degenerative diseases as her defense goes down with advancing years; and since the chain is no stronger than its weakest link, her efficient and healthful life term may be reduced a decade or two or three by the presence of these very dental infections, for she will go into the class with an acquired susceptibility.

We will later discuss, but will anticipate here, the type of individual furnishing most favorable conditions for the various operations such as root-filling, apicoectomy, etc. It will, however, be readily seen that if such operations are to be made, they will be much more favorable of success in the individuals with the high defense, as we have outlined it, than those with the relatively low defense. In other words, such an individual as we have shown here, could doubtless have root fillings made in these roots, and root resections, and be relatively safe in carrying these teeth for a few years because of her normally high defense, not only because of the operation that might be made but, in fact, in spite of any dental operation that might be made. We will later discuss in further detail the many overloads that may come suddenly and make the individual suddenly susceptible to the infections to which he has previously been relatively immune.

This quality of the nature of the discharge from a fistula varies greatly with the stage of the break or absence of break in the defense. We have very frequently seen, and this can be observed

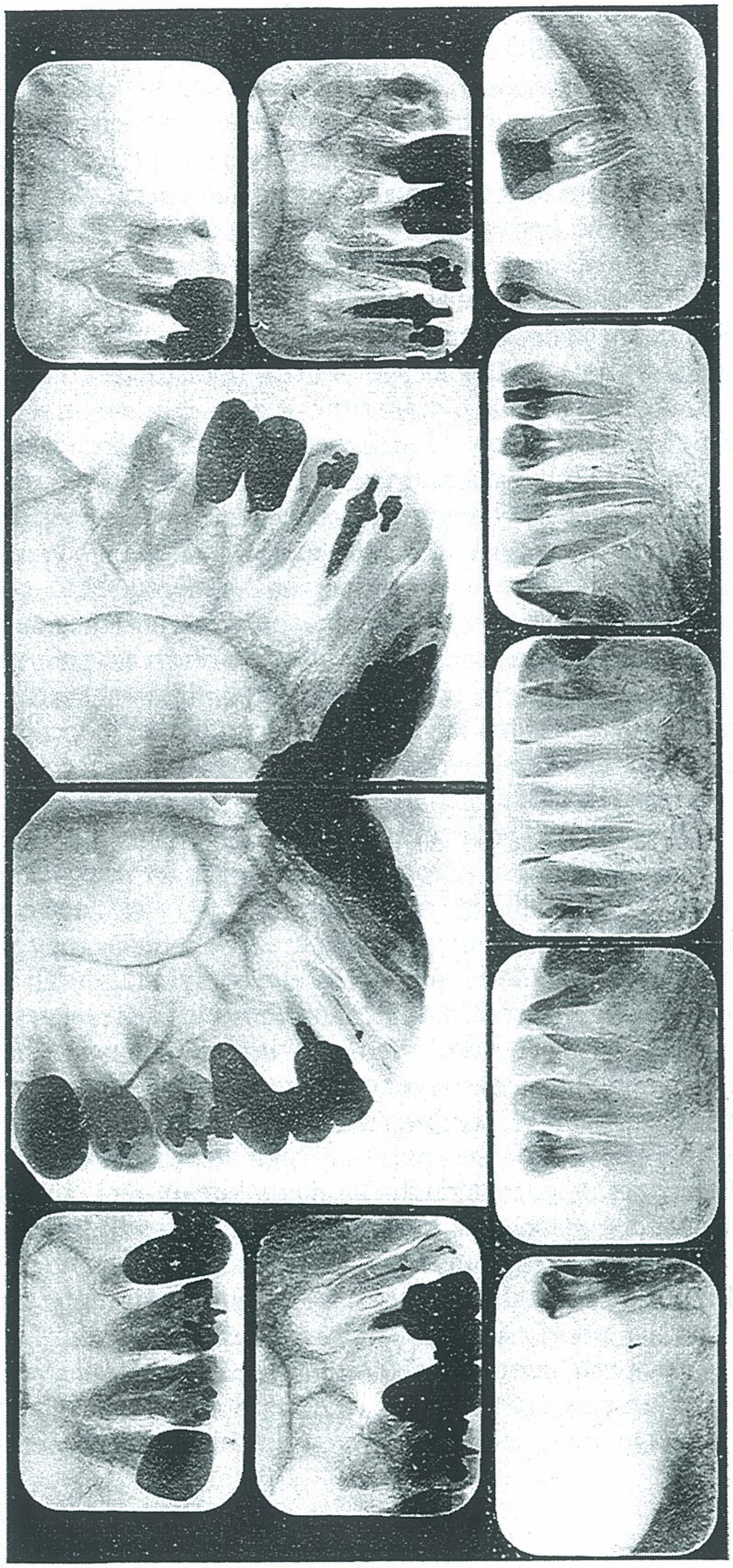


FIGURE 98. APPEARANCE OF PERIAPICAL REACTION OF PATIENT WHO HAS A HIGH DEFENSE, REFERRED TO IN FIGURE 97.

in many mouths, the scar of a fistula which has closed, not because the conditions have become better though they apparently have, since the flow has stopped, but because the conditions have become worse. Very many fistulae close, following an attack of influenza. In subsequent chapters I will discuss some qualities of the mechanisms involved. Incidentally, it has been because of the finding in the clinics, particularly the outpatient clinics of hospitals, of large numbers of individuals with flowing pus from fistulae about broken down and carious roots and no rheumatic group lesions, the patient having come because of a specific infection or traumatic injury, that large numbers of the medical and dental profession have concluded that if people with so much dental infection as these individuals apparently had, do not have rheumatic group lesions, then dental infections are not capable of producing so serious disturbance as some of us have claimed. In other words, these expressions of defense have been mistaken for evidences of quantity of infection. I would ask the reader to look again carefully at the root fillings in the teeth showing these large apical areas in Figure 98, and compare the quantity of infection possible in these teeth with the quantity available with an entire putrescent pulp, as shown in the various individuals in Figures 92, 93, 94, and 95, in the preceding chapter.

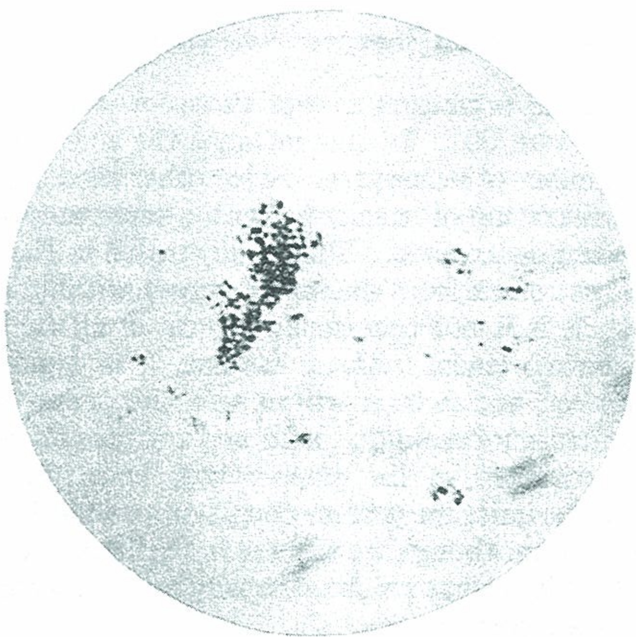


FIGURE 99. SMEAR FROM ROOT APEX OF TOOTH OF FLOWING FISTULA. ONLY PHAGOCYTED ORGANISMS ARE SEEN.

A smear from the apex of a root of a tooth with a flowing fistula

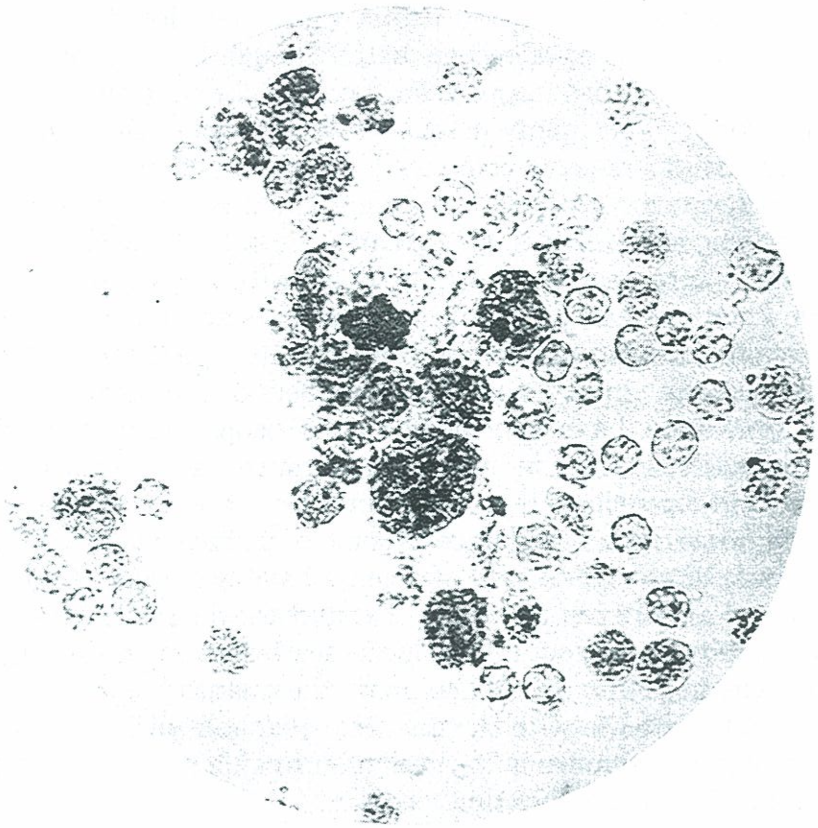


FIGURE 100. A PROFUSE PHAGOCYTOSIS FROM A FLOWING FISTULA.

shows practically no organisms except those few that are phagocyted. (See Figure 99.) In this instance the smear does not show a large number of leucocytes. Some other cases do show a very profuse infiltration of leucocytes with a large proportion of them phagocyting leucocytes. This is illustrated in Figure 100. Figure 101 shows a smear from the root of a tooth without a dental fistula, and while it is not necessarily typical of all the various types of apical involvement without fistulae, it is typical of a very large number; and in it, it will be seen that there are large numbers of organisms free in the fluid and not yet phagocyted, beside those contained in the phagocyting leucocytes. This would appear to be quite the reverse condition to what is generally expected to obtain, though we will see in the discussion of this phenomenon that it is what we should really have expected if our interpretations are correct.

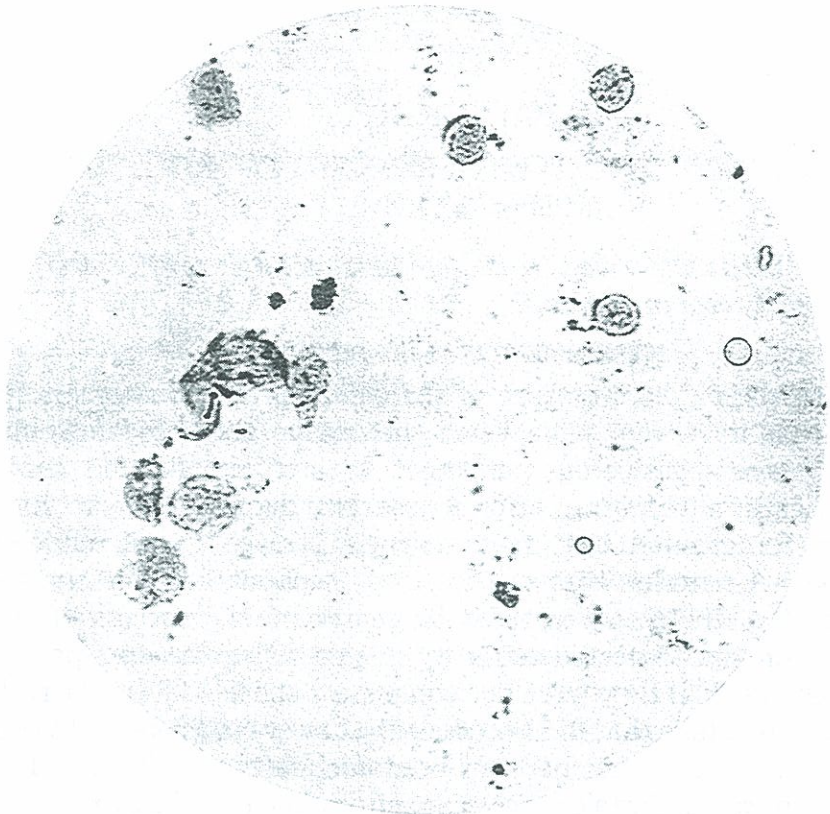


FIGURE 101. SMEAR FROM APEX OF A TOOTH WITHOUT A FISTULA.

We would, accordingly, restate the fundamental, which has generally been thought of about as follows: namely, *that flowing pus from a fistula is necessarily very dangerous to the patient,* to the following:

Since an adequately active defense against a dental infection produces a vigorous local reaction with attending extensive absorption, and the products of inflammatory reaction—namely, exudate and plasma in sufficient quantity to require an overflow, usually spoken of as pus from a fistula—this overflow may be, and usually is, evidence of an active defense and is constituted almost wholly of neutralized products, and is often sterile; and such a condition is much more safe than the same infected tooth without such an active local reaction.

CHAPTER XIV.

EFFICIENCY AND HARMLESSNESS OF ROOT CANAL MEDICATIONS.

PROBLEM: Can infected pulpless teeth be readily sterilized by root medication?

EXPERIMENTAL AND DISCUSSION.

A search of the literature of the dental profession reveals the fact that for seventy-five years there have abounded formulas which were considered competent to sterilize infected teeth. Practically all authors, prior to the last decade, seem to have taken for granted that teeth properly treated were rendered sterile. A criterion that seemed to be dependable—namely, the comfort of the teeth—seemed to guarantee the success of this operation since infected teeth were expected to become painful and abscessed if they were not completely sterile. The relatively small percentage that did become painful were interpreted to constitute the group that had not been adequately sterilized. This research was undertaken to establish, by means of experimental data, the soundness of that conclusion, and with the full confidence that the experiments would corroborate and establish the general presumption to be a fact.

Figure 102 shows a series of roentgenograms of the same tooth, treated by me in 1901, at which time I sterilized the tooth according to the regulation methods of the time; and in accordance with the roentgenograms, since the periapical area diminished and there was apparent deposition of bone with a condensing of the bone about the apex, I seemed justified in concluding that my operation had been a success, since I put in an up-to-date root filling extending to the apex. Owing to the patient's having a progressive type of deforming arthritis, I later, in 1916, sent for her and on the strength of our accumulating experience extracted this and some other teeth. Note that the bone, as shown in A, B, and C, had become continually more dense about this tooth. Upon its extraction, and at which time it was not in the least tender, nor had it been since its treatment fourteen years previously, cultures were made at the apex of the tooth, internally

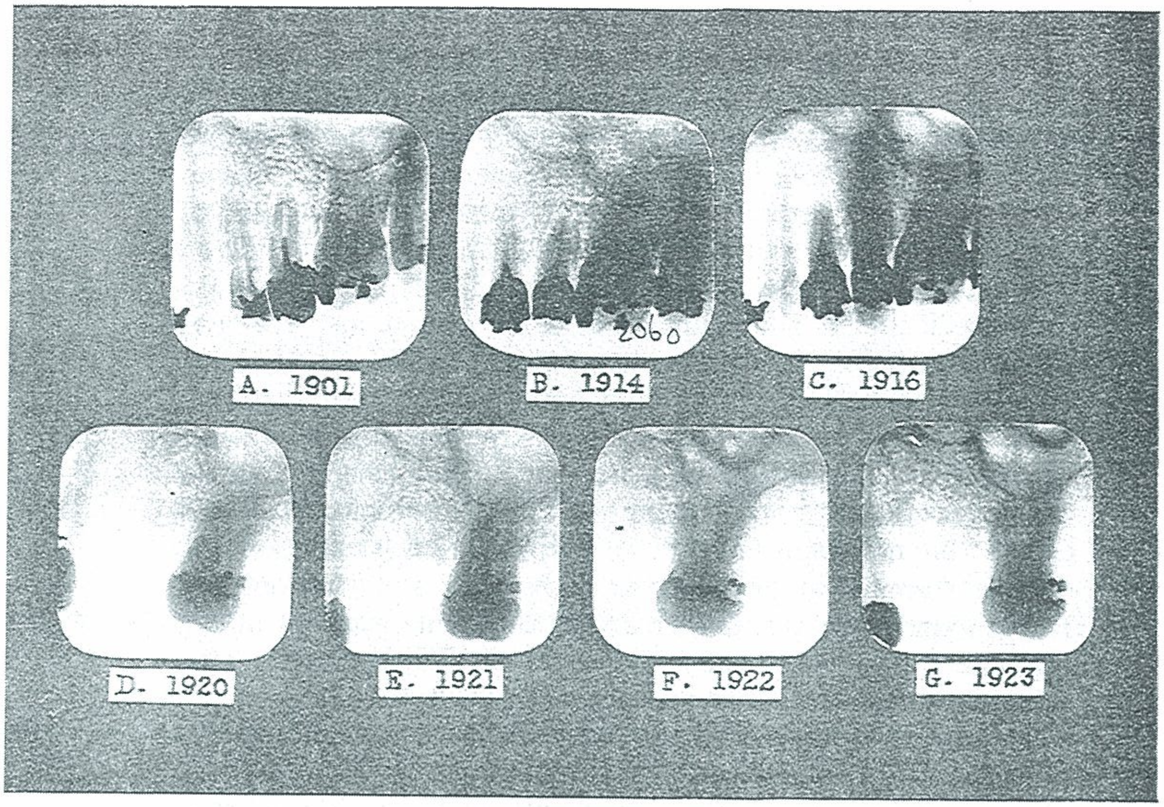


FIGURE 102. CHANGES IN RADIOLUCENCY ABOUT SAME TOOTH, SECOND BICUSPID, IN FIFTEEN YEARS. PATIENT HAS DEFORMING ARTHRITIS. A, TAKEN IN 1901, B IN 1914, C IN 1916. RETURN TOWARD NORMAL AFTER EXTRACTION SHOWN IN D, 1920, IN E, 1921, IN F, 1922 AND IN G, 1923.

and externally. Before extraction a curetting was made to the bone at the gingival margin with an actual cautery, and the tooth was found profusely infected with a strain with definite tissue affinity qualities; and more, not only was the root infected through and through, but the bone for considerable distance about the tooth, as reported in Chapter 3.

Because of many experiences corresponding to that just cited, we planned a series of experiments that were intended to demonstrate why we failed in such cases as the above. One of the earliest tests was to place in the roots of teeth sterile root dressings, carrying various medicaments which were left sealed in the root for various periods of time. This first series was all done on patients; and to our complete amazement, in practically all dressings that were left in roots forty-eight hours, and with most of them after twenty-four hours, the apical third of the dressing was found infected regardless of what the medicament was that was placed on that dressing, *provided there was an apical involve-*

ment about the root. These studies suggested that there was a quantity effect that was very important: namely, that we were not able to put into the root of a tooth, sufficient medicament to overcome the nutrient effect of the fluid within the tooth, and surrounding the tooth, regardless of the medicament. We accordingly tested the relation of tooth mass to medicament volume, by placing blocks of infected tooth substance in relatively large quantities of medicament, and found that, in over a hundred medicaments and dilutions of same even under the very unusual conditions in which a large mass of medicament was in contact with both the inner and outer portion of a section of tooth root, and where medicaments of strengths were used which clearly would be prohibitive in the mouth, because of their destructive action on the adjoining vital tissues the cementum and dentin were not disinfected except with a very few medicaments: namely, formalin in all strengths, iodin 5%, and chlorophenol. It was found, however, that many medicaments exerted inhibiting effects where organisms often grew out days or weeks after being transferred from the medicament to culture media.

The next series of experiments was made to determine the ability of medicaments to maintain the sterility of the root-dressing when sealed in an infected root and without the perforated root-apex's being immersed in an infected culture medium. The medicaments found most promising in the preceding experiment were used for these tests: namely, formocresol, iodin-creosote, iodin 7%, formalin concentrated, phenol 5%, cloves 50%, sulphuric acid 50%, thymol, mercurophen, chloralhydrate, hydrogen dioxide, creosote, alcohol 70%, chlorophenol, phenol compound, chlorazene 4%, dichloramin 15%, and phenol saturated. In these it was found that the most radical disinfectant was dichloramin-T used in saturated form; but in this strength was found to be exceedingly irritating when used in conditions of the mouth, often producing great pain and irritation of the supporting tissues. At that time we made a note: "The members of the profession are warned never to place the 15% or even a 5% solution of the Dichloramin-T in oil of Eucalyptol, its solvent, in any tooth or on tissues."

Of the 18 medicaments used for this experiment, only 6 gave instances of negative results in cultures; or, in other words, were efficient; the remaining all gave positive cultures in every case for the complete series of hours run. The five best results are shown

	5 Hrs.	24 Hrs.	48 Hrs.
Dichloramin-T	100%	100%	100%
Iodin Creosote	81%	25%	0%
Phenol Compound	81%	50%	0%
Formalin	75%	94%	37%
Formocresol	56%	62%	6%

in Figure 103.

The next experiment was made to determine the ability of the medicaments to maintain the sterility of the root dressing when sealed in an infected root, when the perforated root apex is immersed in an infected culture medium. For this experiment, forty medicaments which showed the most encouraging results in experiment No. 1 were tested, and the efficiency of the various preparations determined by placing the dressing carrying the medicament in the root canal with its small perforated apex corresponding with the small open foramen, and the tooth so prepared, itself an infected root, was placed in a container in which was placed a small quantity of infected culture media which came in contact with the apex of the tooth, the tooth crown being covered to the cementum, after it was dried, with paraffin. Of the forty medicaments used, only eight gave negative results, the remainder being all positive in every trial and for all periods of time. The length of time the medicament was left in the tooth was found to make a very great difference. This test was not made to determine the ability of the medicament to sterilize tooth structure, but rather the ability of the medicament to maintain the sterility of the sterile J. & J. absorbent point on which it was placed in the tooth. The points were removed under sterile conditions and cut into sections 1, 2, 3, and 4, numbering from the root apex up.

	5 Hrs.	24 Hrs.	48 Hrs.
Phenol Compound	70%	15%	0%
Dichloramin-T 15%	35%	5%	0%
Formocresol	34%	0%	0%
Iodin-Creosote	33%	25%	0%
Iodin U. S. P.	10%	34%	0%
Formalin U. S. P.	0%	34%	0%
Phenol U. S. P.	0%	45%	0%
Oil of Cloves	0%	25%	0%

Figure No. 104 shows the result of the eight best of the forty: All were infected in forty-eight hours, nearly all in twenty-four hours, and a few had some efficiency, if tested within five hours.

Experiment No. 4 was made to determine the ability of medicaments to sterilize infected dentin and cementum when sealed in the root canal, and when the perforated apex is surrounded by infected culture medium, as in the conditions in Experiment 3. Note the difference that in this experiment we are determining the influence on the dentin and cementum; whereas, in the former, we are testing the ability of medicaments even to maintain the sterility of the root canal dressing. After the test was made on the teeth, as in the previous experiment, the teeth were divided longitudinally with precautions to prevent contamination, and cultures were taken with fine-pointed burrs, properly controlled against contamination, from various structures in various parts of the tooth. The results of these experiments were that with the exception of concentrated formalin which showed 93% sterility, all of the teeth showed a general infected condition of the various tooth structures, and even formalin 5% had a very low efficiency.

Tests were made similarly with double salt of ammonium silver and formalin. Four series were run. Series 1 gave an efficiency of 69%; Series 2, 78%; Series 3, 84%; and Series 4, 95%.

The general result of these studies was summed up under "General Summary and Conclusions" as follows:

"1. Infected dentin and cementum are not readily sterilized by medication, even when the amount of medicament is largely in excess of the mass of tooth structure.

"2. The medicament contained in a medicated root dressing very readily and rapidly loses its disinfecting power, for the dressing itself becomes infected in a few hours, even from the infected dentin and without being in contact with an infected solution.

"3. The medicament in the dressing dissipates more rapidly when one end of the dressing is in contact with an infected culture medium, such condition as obtains in a periapical infection.

"4. The efficiency of a root treatment is greater a few hours after it has been placed in the tooth than several days', or even one day's time.

"5. All areas of cementum and dentin are difficult to sterilize, as well as tend to reinfection, when the medicated root dressing is left more than a few hours.

"6. The medicaments that are most efficient, namely, silver nitrate and formalin, are very objectionable, the former by its discoloration and the latter by its destructing and irritating properties, except when used very dilute and for a short time and then quite efficiently.

"7. There is much encouragement in the use of Chlorazene (chloramin-T) and Dichloramin-T products. The latter, however, is exceedingly painful and destructive in over half of one per cent strength.

"8. We now understand why it has been, that if a root treatment is left in the tooth for an extended period of time, it becomes foul in odor. Also a verification of the observation previously reported by one of us, Price, to the effect that medicated root dressings will always show bacterial growth after they have been left in infected teeth for a few days.

"9. These determinations indicate to us that an exceedingly small per cent of the teeth, that have been treated by members of the dental profession in the past, have actually been sterilized.

"10. Nature is, and has always been, very tolerant, and much of the credit that has been taken by, and given to, the dental profession for the sterilization of infected roots, has really been due to Nature for her kindness in tolerating, at least without local irritation, a probably almost universal and permanent condition of infection, though in lessened amount, when tooth structures have once been infected and have been subjected to treatment."

These experiments were made under my direct supervision, partly by myself and partly by an assistant, Mrs. Matilda Moldenhauer Brooks[4] in the Research Institute of the National Dental Association, in which I was Research Director; and a full report will be found in detail, in the Journal of the National Dental Association, Volume V, No. 3, March 1918, pp. 273-303, which report was read before the National Dental Association at its Twenty-First Annual Session, New York City, October 1917.

We have run many controls and parallel experiments, and have found that the one that seemed to give most satisfying results in our hands has been chloramin-T (not dichloramin-T) which can be used in saturated aqueous solution in paste form, is soluble in water up to about 8 to 12% (Dichloramin-T is not soluble in water), does not discolor the teeth, and by being used as a paste, has so much reserve material for sterilizing that it retains its efficiency for a longer time than most of the preparations, and is not particularly irritating unless there be considerable vital pulp tissue within the root. The silver formalin, or the silver precip-

[4] See bibliography.

itated by other means, as for example eugenol, has high efficiency where it can be used, but produces marked discoloration.

In addition to the above studies, we have carefully cultured many teeth after we had thoroughly treated them, with the hope and expectation of retaining them in the mouth; but later decided to remove them; and, in practically all cases where teeth had been putrescent and had evidence of periapical involvement, though slight, we practically always failed to sterilize cementum at the apex, and frequently had positive growth from the dentin.

Another phase of this problem has been the checking of the more improved methods of sterilizing infected tooth structures within the mouth, since it has been claimed[5] that by testing the tooth which had been sterilized, it was possible to determine whether or not that process had been complete. It is advocated, for example, by some authorities, that if a canal point saturated with culture medium is placed in the canal after its sterilization, a determination can be made in one, two, or several days, to determine whether or not sterilization is complete. In the chapter on bacterial accommodation we discussed the quality which bacteria have of adapting themselves to environment. In our studies, reported previously, we found that the practise of discarding controls and tests at the end of one or two weeks was entirely misleading, for very frequently the medication had the effect of inhibiting growth completely for a period of several weeks. But this is not the only difficulty. The organisms may, under the stress of the medication, take on a quality which requires an entirely different type of medium, not only differing in nutriment but in oxygen tension, which qualities apparently are not adjusted to, nor is there a report of their having been taken into, consideration in the reports that have indicated that complete sterility might be depended upon, particularly if checked by this simple test.

Another argument that will be made will be that ionization, either by means of ionizing potassium iodide, sodium chloride, or other solutions, will disinfect infected cementum, when applied through the pulp canal. This problem was very thoroughly studied by Pond and Price[6].

We have found strains in treated teeth in the mouth, for which we could not find any medium, on which they would grow until

[5] See bibliography.
[6] See bibliography.

we would place them in a sealed Petri dish, which was connected with another Petri dish by an hermetically sealed tube, in which second dish was growing a culture of an organism which rapidly consumed oxygen. For days no growth would appear, when suddenly it would start, but cease to continue to grow as soon as that optimum of oxygen tension was passed. They would not grow in less or more than a limited range of partial tension, and all our efforts to grow them by regulation methods aerobically and anaerobically completely failed. We now believe that many of our early determinations, where we got negative results, were so only because we were incapable of furnishing the proper medium and environment. We are also sure that this applies equally to some of the negative work of others; and, accordingly, while a positive growth can only be read positive, a negative growth is not necessarily negative.

As we have shown, it is exceedingly difficult to neutralize infected dentin under any circumstances, without using medicaments which may in some degree endanger the supporting structures. Such studies made on extracted teeth are open to the criticism, that the conditions are not the same as in the mouth, and therefore cannot be considered comparable, since we do not have the original tooth in contact with vital tissue. To overcome this difficulty, and to throw further light upon the matter of tooth sterilization, I have planted many teeth under the skins of rabbits to determine their effects upon the animal, before the tooth was medicated, for comparison with the effect of placing within that tooth such medication as is placed in teeth in the mouth. There is a great difference in the effect of different teeth on the rabbits when planted in this way beneath the skin. Frequently they are incysted and the rabbit tissue proceeds to absorb the root. In some others, notwithstanding the incysting, a nephritis is slowly developed, as illustrated elsewhere. In still others, the dental infection or the toxic products from the tooth, or both, completely prevent Nature's effort to encapsulate the tooth, and in many instances the rabbit is dead in from one and one-half to a few days; as, for example, we reported in Chapter 17 a series of over twenty-five rabbits which were killed by a single tooth in from one and one-half to six days, one rabbit living ten days. For this study I have selected teeth, which killed rabbits in from two to four days, after they were extracted from the patient, where, incidentally, there was no roentgenographic evidence that they were particularly serious, though the patient was being systemically injured by them. Some of the results have been as follows:

A tooth which killed a rabbit in three days (R. 1110) was opened through the crown as it would be in the mouth, the pulp canals cleansed and a dressing placed in it of iodin and creosote. The tooth was placed under the skin of another rabbit which died in four days (R. 1115). The tooth was again opened and treated with the same medicament and placed under another rabbit's skin and it died in four days (R. 1119). Inasmuch as sterile teeth produce no effect whatever in rabbits, it is very important to note that this tooth not only killed the rabbits, but that they began losing in weight within a few hours after placing the medicament, and lost approximately twenty per cent of their weight in two days. This amount cannot be due to the dehydration or starvation of the animal, for it goes on progressively till sometimes at the time of the death of the rabbit, it will have lost forty per cent of its weight, and present a condition of marked marasmus.

Similarly, we have tested a number of medicaments by treating the teeth with them before planting them under the skin of a rabbit, as nearly as possible as they would be treated in the mouth. In this way we have tested chloramin-T, silver nitrate neutralized with formalin or eugenol, formocresol, two per cent formalin and hot air, with similar results to those obtained with iodin and creosote as above. I have now made several hundred of these determinations, results of which emphasize the need both for a new appreciation of danger from infected cementum, because of the apparent difficulty, if not impossibility, for sterilizing infected cementum by treating through a root canal. The results so far obtained also stress the great necessity for repeating these tests under as carefully controlled conditions as possible, which we are doing as this goes to press. I will publish further details later.

To determine the nature of radiations of a greater length but still in the ultraviolet range, we have studied the effect of placing a tooth that had proven to be fatal to several animals when placed under the skin, under the radiations from the water cooled mercury vapor quartz arc lamp, and have found a very marked change in the reactions of the rabbits when the tissues over the implanted tooth were so treated. Whereas, this tooth previously produced a very marked leucocytic infiltration about it, there were practically no leucocytes, or very few, the organisms being present in pure culture in large numbers in the exudate of the tooth at the time of the death of the rabbit, and the rabbit had developed a very violent appendicitis and colitis, and probably peritonitis. Whether this change in the effect of the infection after the ultraviolet radiation was due to a change in the attacking power of the organism as a result of this radiation, or to a greater susceptibility of the abdominal viscera because of the depressing effect of the radiation, we are not yet able to state.

But it may be stated, and justly so, that this does not yet produce an experiment which is closely comparable to those under which operations are made in the mouth. To test this still further, I have made the following experiment: A tooth, the right

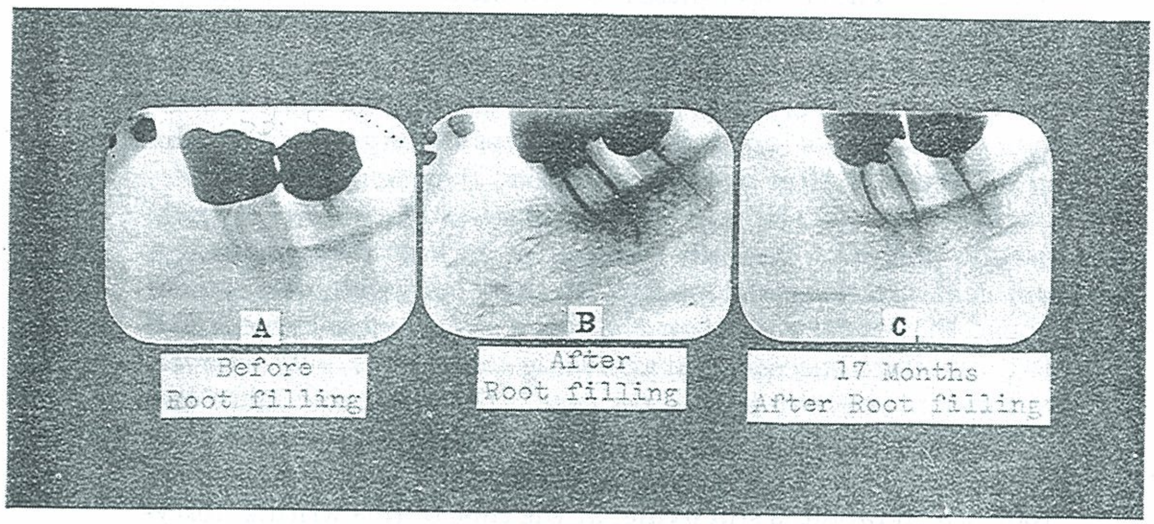


FIGURE 105. THREE SUCCESSIVE VIEWS OF A MANDIBULAR MOLAR. A, BEFORE REMOVAL OF DEGENERATING PULP, B IMMEDIATELY AFTER ROOT FILLING, C SEVENTEEN MONTHS AFTER ROOT FILLING.

second mandibular molar, with a hypersensitive and degenerating pulp, shown in Figure 105-A, which tooth was considered very valuable to the patient, and which showed slight, if any, alveolar absorption at its apex, was treated with dichloramin-T, put in with good access in the canals for twenty-four hours, and was then root-filled with the result shown in Figure 105-B. Two years later the tooth was extracted and also one adjoining it (the third mandibular molar, the root of which was treated and refilled at the same time as the second molar), as a matter of precaution, since the patient was considered sufficiently susceptible to streptococcal infection to require protection, and because there was still enough sensation to pressure in tapping in the tooth, to suggest that it was not entirely normal, notwithstanding the appearance in the roentgenogram, as shown in Figure 105-C. This tooth was extracted, thoroughly washed in sterile normal salt solution, the root divided, and each part planted beneath the skin of a rabbit. It will be noted that at the time of the root filling, there was a very slight protrusion of the chloropercha to make a so-called encapsulation, but there was every reason to believe that the roots were efficiently treated and well filled, not only because of the method used, but also because of the suggested improvement in the supporting structures of the tooth about the apices of the roots, as shown by the reduced area of alveolar absorption from a small but definite area in B, to no apparent area in C. The effect of these roots on the rabbits was as follows:

To determine whether or not there was infection within the structure of the root, I sterilized the apex of the tooth with a hot instrument, searing deeply, then drilled through the seared structure to the interior, and cultured the chips which grew out a strain of diplococci. After dividing the roots, they were washed thoroughly in normal salt solution, and one was placed with surgical aseptic procedure beneath the skin of each of two rabbits. A purulent infection developed about each, but much more rapidly about the mesial root than the distal. The mesial root killed four rabbits in succession in an average of six days, with an average loss in weight of twenty per cent. The distal root killed two rabbits in thirty days, with an average loss of thirty-one per cent. In another place I am discussing the result of boiling the mesial root, which rabbit is still living, at the time of this writing, forty-six days after implantation and has gained 228 grams, or 21 per cent.

Great care must be taken in interpreting these results; and it is only because of the large number of similar results that I consent to present such a striking case, for I realize fully that many men, who, because of their greater interest in exodontia than in dental pathology, will be in danger of jumping at the conclusion that this is positive proof that all root-filled teeth should be extracted. I do feel it my duty, however, to make this important information public, to aid in crystallizing an appreciation of the danger that may attend an over-confidence in the completeness of the sterilization of infected roots. I will state here what I have stressed in further detail in other chapters, that many parts of the body are exposed to infection, and we have reason to believe contain infective germs frequently or for extended periods; but the condition of safety or danger will be dependent quite largely on the defensive factors of the patient; and with a given tooth, some individuals will, I believe, be relatively safe, while others will be in definite danger. I am not ready to draw the line so rigidly as to state that all root-filled teeth should be extracted for every patient or for all patients in any given time, though I do believe there is a limit of safety for all such teeth for each and every patient. I do deem it absolutely essential that very exhaustive researches be continued on this subject in order that we may be able to draw our lines with greater exactness than we can with the limited knowledge, though very important, that is available. I think I should state here that I have not seen a single logical presentation of data that will justify the claims of any of the extremists, such as the so-called "hundred per centers." In Chapter 17 on Quantity, Systemic Effect, and Tooth Capacity, in Figure 122, I present a table showing the results of 237 implantations 209 of which were teeth, which should give much food for thought for those who believe that comfort and roentgenographic appearance are a guarantee of the absence of tooth infection.

I have made many tests, and have many in progress, to determine whether, and by what means, infected teeth may be completely sterilized by treating through the pulp canal where there had been definite evidence of destruction of the peridental membrane about the apex. In Figure 106 (R. 1149) there is shown a rabbit that died in two days with a loss in weight of seventeen per cent, from a tooth that had been treated with silver nitrate neutralized with twenty-five per cent formalin, by placing these within the pulp canal, but without allowing these medicaments to get in contact with the cementum. A shows the tooth under the skin and an abscess as big as several silver dollars, with pure culture of streptococci, as shown in B, and which I believe grew out from the infected cementum, which infected cementum was not sterilized by the silver nitrate neutralized with twenty-five per cent formalin. Similarly, I have passed fuming formalin (forty per cent formaldehyde) through the pulp canal of an infected tooth, where I had reason to believe the cementum was also infected, and in which I protected the external surface of the tooth by putting a rubber dam on both the neck and the apex. This material passed through the tooth with a gravity pressure

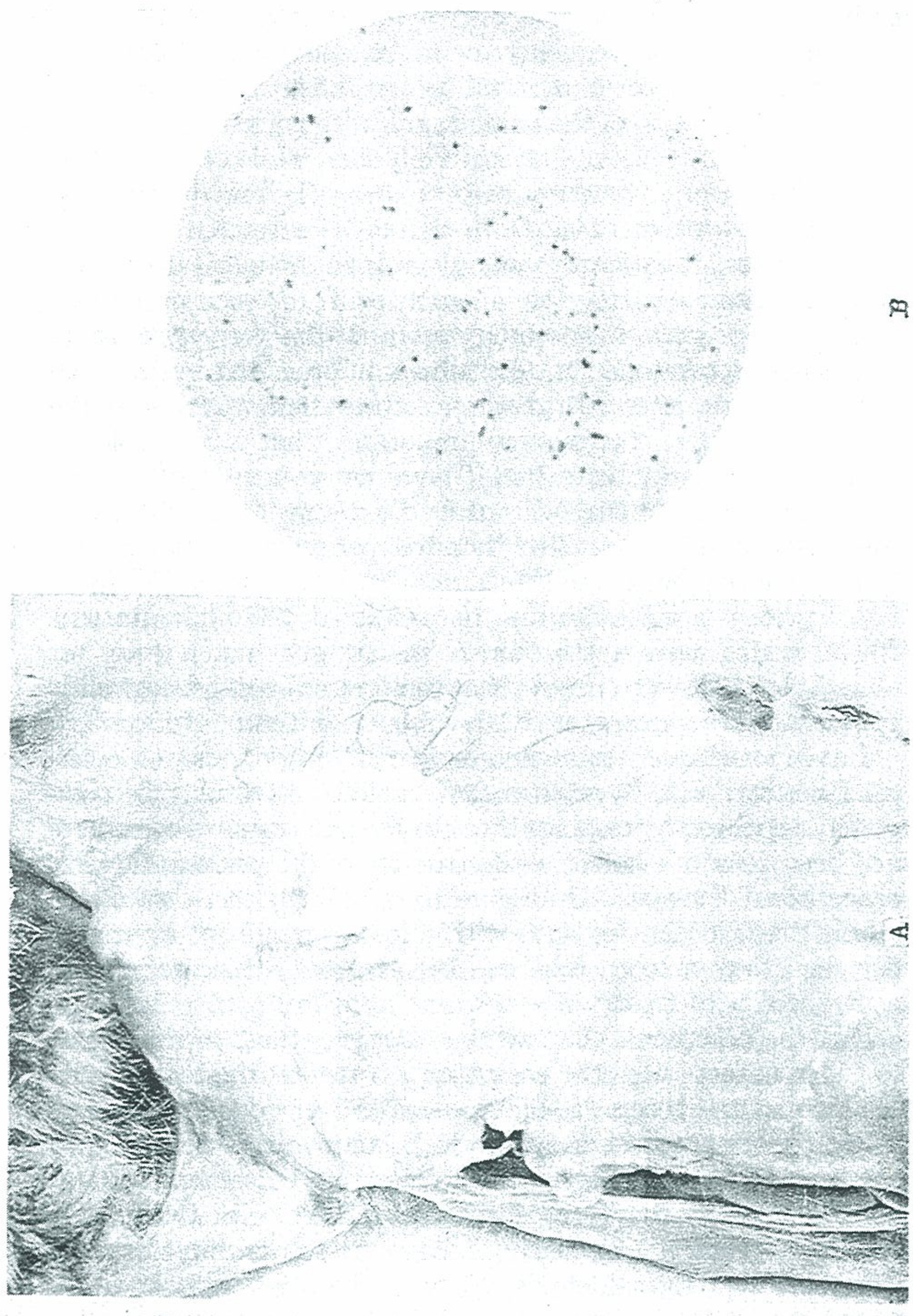


FIGURE 106. RABBIT APPARENTLY KILLED BY THE INFECTION IN THE CEMENTUM OF AN IMPLANTED TOOTH, AFTER DENTIN WAS TREATED WITH SILVER NITRATE AND FORMALIN.

of about one-tenth of an atmosphere for fifteen minutes. The tooth was then washed with normal salt solution, also irrigated through the pulp chamber. Ten grams of the concentrated formalin passed the tooth. After washing, an effort was made to remove all trace of formalin by passing a stream of air through the pulp canal, the apex of which was sufficiently large to pass a considerable stream. The tooth was then planted under surgical conditions beneath the skin of a rabbit, and an abscess developed containing pure culture of diplo- and strepto-cocci. It seems probable that the fire-wall existing between cementum and dentin was not penetrated by even concentrated formalin in fifteen minutes' time in sufficient quantity to destroy the organism within the cementum. Much light is thrown upon this problem in the subsequent chapters on Chemical Changes in the Blood and The Nature of the Defensive Factors in the Blood and the Forces which Injure Them.

In the physical structure of the tooth with the dentin connected chiefly, if not solely, with the pulp, and the cementum connected chiefly, if not solely, with the pericemental membrane, we are dealing with two structures, each sufficiently porous to give habitation to millions of organisms. We have shown elsewhere that the dentinal tubuli of a single rooted tooth comprise enclosed canals totaling approximately three miles of length, and it is probable that no dental canal exists too small for organisms to develop within it, but each of these two structures is formed on a practically continuous homogeneous base, the dentin and cementum being backed up to each other. Whether, as believed by some, there are communicating channels from the lacunae and canaliculi of the cementum through the dentino-cemental boundary into the dental tubuli of the dentin, or whether the only communication between these two structures will be through multiple foramina, is not yet definitely established, though several conditions suggest one or the other of these answers to be correct.

In order to throw light upon this important problem I have placed infected teeth, as they were extracted, in culture media sufficiently hardened with agar to localize the bacterial growths, and have found that the bacterial growth in the medium seems to be entirely limited to zones which are probably foramina of the tooth, chiefly apical with some lateral. I have similarly tested this same problem by placing within the teeth, solutions, the ions of which, when they would pass by osmosis from the tooth to the agar media surrounding the tooth, would react on the coloring medium by the change in the pH. These have also indicated that for all practical purposes a very large proportion of the total communication is limited to the foramina. In Chapter 23 we have shown that the structure forming the boundary between dentin and cementum, is in effect a dialyzing membrane, and that electrolytes may pass through that membrane quite freely while colloids cannot. It is, accordingly, very probable that certain crystalloid substances can pass through this boundary.

SUMMARY AND CONCLUSIONS.

It should be remembered that in the formation of the tooth, both the dentin and cementum are built upon a first zone of calcification which is in effect a continuous fire-wall or protecting zone between the two structures, with but relatively few direct connections or openings from the dentin to the cementum. In the structure of the dentin, with its long and narrow channels constituting the dental tubuli with their anastomosing branchings, we have a structure, particularly favorable, for the hiding away of the organisms because of the mechanical difficulty of getting medicament into the structure. We think it is probable that often a relatively high efficiency may be secured in sterilizing young dentin to the granular layer, and possibly to the dento-cemental junction. We think it is very improbable, from our many experiments, that the cementum can ever be sterilized by a medicament placed within the pulp chamber, without definitely and seriously embarrassing the surrounding supporting structures.

We would, therefore, change the accepted fundamental from *That infected teeth can be sterilized readily by medication, and that the usual medications do not injure the supporting structures*, to

> **That infected teeth can be sterilized in the mouth only with very great difficulty or by using over strong medicaments; and the usual medications (particularly those previously used) frequently, if not generally, injure to some extent the supporting structures.**

CHAPTER XV.

ROOT FILLINGS, THEIR CONSTANCY AND EFFICIENCY

PROBLEM: Do root fillings fill root canals, and do they continue to do so?

Practically all diagnosticians, whether dental, medical, or otherwise, who look at roentgenograms of the teeth, look to see whether there is evidence of more or less bone destruction at the apex, and if so, whether the tooth carries a root filling. If it does not, it is like a bottle filled with infection; if it carries a root filling, there is no opening in the bottle, provided the root filling goes to the apex. What could be more simple, complete, and fortunate?

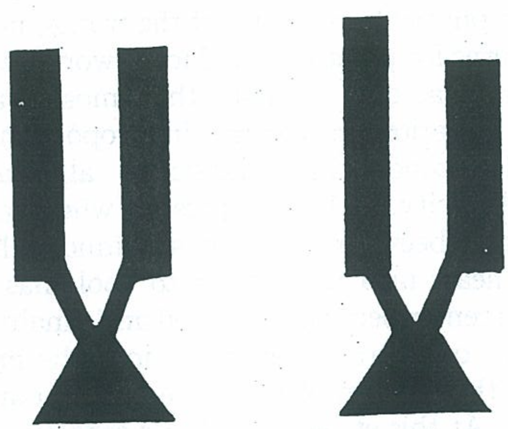
This particular study was undertaken to establish some of the mechanical problems involved: First, under what conditions is it mechanically possible to fill pulp chambers? Second, what is the constancy of the physical state of the mechanical plug we insert? And third, what are the physical properties of the materials used for root fillings?

My work on the physical properties of the waxes, used for impressions and patterns for prosthetic and inlay work, revealed the following important factors:[7] First, that most waxes have a wide range of variation in volume in proportion to their temperature; and second, that practically all waxes have a property of elasticity. Every person who has poured paraffin, tallow, lard, beeswax, or such substance when liquid and molten with heat, into receptacles to cool, has observed that, on cooling the center became depressed and, finally, with the last cooling, a great open crater ran down into the mass of the material, often to the bottom of the vessel, with branchings in various directions. At this stage, 5 to 10 per cent more material could be poured into the form filling these cracks before bringing it to the original level of the molten mass; and when this mass cooled, it again, in the same proportion, had its shrinkage, and so on indefinitely. Fundamentally, with a change of state, there was a definite change in mass.

[7] See bibliography

Since heat was formerly used a great deal in packing gutta-percha root canal points, the problem arises, "To what extent does the same factor obtain?" All who had experience in the use of chloropercha, as it was used so extensively years ago for root fillings preliminary to placing the points, had a practical demonstration of the effect of leaving the stopper out of the bottle, with the result that the bottle containing, say a half ounce of creamy chloropercha would, after evaporation of chloroform, leave a nugget that was always drawn away from the sides of the bottle, and which would rattle about with great freedom. In discussions and questionings regarding the possibilities of this condition's obtaining in the mouth, I was always assured that the two conditions were not comparable.

For references to some of the detailed work that I published on the relation of change of state to mass, of various substances including molten gold, I would give the following: Price[8]—The Laws Determining the Behavior of Gold in Fusing and Casting—Dental Cosmos March 1911; Special Researches in Physics—Journal of the National Dental Association, October, 1914; and the reference to my previous publication on this subject in the Journal of the National Dental Association, December, 1918.



A B

FIGURE 108. TWO SIMILAR WAX BARS PLACED IN A WARM INVESTMENT AND CAST. A, BEFORE HEATING, B AFTER HEATING AND WHILE INVESTMENT WAS STILL SOFT, SHOWING THE CHANGES IN THE WAX AND THE SHAPE OF THE CASTING.

From the latter I take the following illustration. Figure 108 shows similar wax bars placed in a warm investment and cast: A, in wax before heating by putting in the warm investment; and

[8] See bibliography.

B, after casting showing the change that has taken place as a result of the heat. This is neither expansion nor contraction; it is the releasing of elasticity. A wax bar was warmed, and while sufficiently warmed to compress, the pencil of wax was put under pressure on end, which shortened it. In changed state under pressure it was chilled. Another cylinder of the same wax was treated similarly, except that it was stretched instead of condensed. From each, sections one inch long were taken. These two wax bars were similar in every respect, except that one had a retained elasticity in one direction, and the other in the opposite direction. When the two pieces of wax were placed in the warm investment, the warmth released the retained elastic stress; and after the retained elasticity was released, the compressed piece became longer and the stretched piece became shorter. This took place in the investment which was heated just enough to follow the changing wax, with the effect that before the wax was melted from the investment, two entirely different sizes of molds had been formed. Since both were attached to the same sprue-gate, and both were cast together, the castings reproduced this physical change which took place in the wax. It also followed its own law of contraction, and all diameters were reduced in accordance with its laws of contraction.

In the former of the articles just referred to, I showed that pure gold changed approximately 2 per cent in mass, within a fraction of a degree, in changing from the solid to the liquid state, and approximately another 2 per cent in changing from its first crystalline form as it freezes to room temperature. These are cubic measurements, linear being one-third of this amount. Ice behaves differently by changing in the opposite direction, which is the reason pipes and bottles burst. Our waxes, gutta-percha, etc., when placed in root canals, behave in the opposite manner to that which water does when taking on the solid state, and change to a greater degree, ice being 11 per cent greater in volume than water at the freezing point. To test these dimension changes cylinders of glass were used, and the gutta-percha packed with all pressure the cylinders would stand at various temperatures, and these, when cooled, were tested for the filling of the chambers by placing ink in the cylinders, which invariably passed in between the gutta-percha and the cylinder walls; and, indeed, it was a physical impossibility so to pack the gutta-percha at a temperature at which it could be molded, that there would not be a space between the gutta-percha and glass when the gutta-percha had cooled. Several hundred pounds' pressure were used on the gutta-percha without avail, and, notwithstanding that the pressure was kept on the gutta-percha while it was cooling (which could not be done in a tooth), this shrinkage took place. Figure 109 shows a typical tube demonstrating this effect. The metal pistons are shown continuing the pressure of several hundred pounds on the gutta-percha, notwithstanding which, the gutta-percha left the glass sufficiently to let the ink flow between.

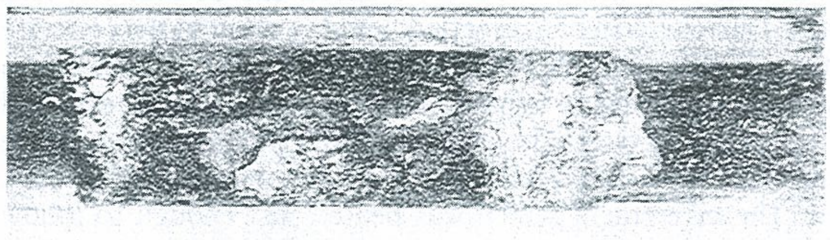


FIGURE 109. GUTTA-PERCHA UNDER A HIGH PRESSURE INSIDE OF GLASS TUBING, TO TEST THE SHRINKAGE OF COOLING GUTTA-PERCHA. NOTE THE INK FLOWED FREELY INTO THE SHRINKAGE SPACE.

However, it is not considered feasible to make gutta-percha moldable in root-filling procedures by means of increasing its temperature. This is done by placing it in a solvent, usually chloroform or chloroform carrying rosin, and the use of oil of eucalyptol as a solvent. Gutta-percha dissolved in chloroform to a consistency of a thick cream or paste, has a volume 300 per cent greater than that of the original material; or to express it otherwise, if the pulp chamber is filled with gutta-percha at about that consistency, when the chloroform has evaporated from it, the chamber would be approximately one-third full. If we put into the creamy mass a cone of dense gutta-percha, the usual gutta-percha point used in root filling, and select one which fills the chamber within 90 per cent of full, leaving only 10 per cent of the volume for the chloropercha, the chamber will still lack 6.6 per cent of being full, when the chloroform has volatilized. Doubtless, many operators have supposed that, merely softening the point with chloroform to make it flexible and moldable, would not change its volume, which is not correct. A sufficient quantity of chloroform to make the gutta-percha flexible is sufficient to change its mass very definitely as will be shown. Gutta-percha, when dissolved with chloroform, makes a sticky paint; but when the chloroform is gone, it peels from all smooth surfaces in its process of contraction and does not retain adhesiveness. It was doubtless for this reason that Dr. Callahan sought another substance, suggested and used rosin; and doubtless for this reason, and for the advantage of a non-irritating sterilizing medium that Dr. Buckley suggested eucalyptol as the solvent for gutta-percha. The ultimate contraction is very great for both of these substances, and corresponds ultimately, relatively with the amount of solvent used, whether the solvent be chloroform, eucalyptol, or chloroform carrying rosin.

The gutta-percha in oil of eucalyptol, the eucapercha of the market, has a volume 417% greater than the volume of the gutta-percha alone, which means that a pulp chamber, filled with eucapercha and submitted to long and complete drying, would ultimately have 24% of the total volume of the pulp chamber filled. When it solidifies, it does so into a lumpy, curdled mass which does not adhere to smooth surfaces. It, however, very fortunately, undergoes this drying process slowly. After weeks of spontaneous air drying at room temperature, it reduced to 87% of the original volume. During a similar period, gutta-percha and chloroform would have diminished to 76% of the original volume. However, by the addition of artificial heat equivalent to that furnished by the body, the rate is greatly hastened, as shown by the tables; but it is not hastened by moisture.

Rosin and chloroform mixture also undergoes a very great reduction in volume, starting with a fluid about the consistency of a light oil. Dr. Callahan suggested a consistency of sewing machine oil as best. In this condition it has a volume 406% of that of the volume of rosin. In other words, a pulp chamber filled with rosin and chloroform solution of that consistency would ultimately be about 24.6% filled. Again, using gutta-percha cones to occupy, say 90% of the total volume, would ultimately leave approximately 7.5% of the pulp chamber unfilled when the complete drying out process has occurred. However, the gutta-percha point will rapidly take up the chloroform and swell so that it will occupy more space, readily swelling to increase in volume ¼ to ½ in the softening process. Since the pulp chamber can contain only a given amount of material, whether fluid, semifluid, or solid, it is a physical impossibility to put in material to make up for the evaporation of the solvent, while the solvent is present. "It is, therefore, a physical impossibility completely to fill a pulp chamber with gutta-percha made plastic by any of the above solvents, except at the time the gutta-percha contains the solvent."

The chloroform and rosin compound has very distinct and favorable qualities, in that it is very adherent and tenacious in all stages, to both smooth and rough surfaces. In this respect, it is unlike each of the preceding compounds. The result of this quality is that, when a root filling is made of this material and gutta-percha cones, the contraction tends to express itself in a large part, by a shrinking towards the walls if dry, rather than as a continuous disc at the side of the mass, and between it and the wall, and herein lies the great virtue of this material as a means for filling pulp chambers.

It will be a matter of surprise, doubtless, to most of us to find that a skin is formed on the surface of each chloropercha, and rosin and chloroform, particularly the latter, which quite effectually delays the drying-out process. In a case of the chloroform and gutta-percha in a narrow chamber, this skin or film separates from the vessel or container, leaving a fault, which ultimately makes a series of caverns, each projecting from the last until the entire mass is honey-combed. In large chambers it contracts to a central free mass. Chloroform and rosin in a narrow chamber of the same form retains the continuity of the protecting surface skin by its adhesion to the wall, and underneath this film the liquid retains a fluidity nearly that of the original, due to the control of the vapor tension by this membrane. Consequently, it is not only a matter of weeks, but of many months, before our root fillings, inserted with any of these three compounds, will have attained their maximum contraction. After air drying in an open vessel for two weeks, rosin-chloroform had reduced to 65% of its original volume; in five weeks to 59%; eight weeks to 36.4%, and the last 12% of the total shrinkage requiring either a long period of time or the addition of artificial heat nearly to the boiling point of water for many hours. When we consider the great variety of forms of pulp canals and the variations in size, shape, and direction of the many foramina, it is evident that the root filling material must seal these foramina by a process of retreating while retaining a close adhesion to the surface of the foramen, instead of contracting to a common center of the mass, producing a fault or space. An illustration of some of the difficulties encountered in root canal fillings is shown in Figure 110. Many other cases are shown throughout this volume, as for example, in the following conditions:

It will clearly be seen that there are many conditions which would make impossible the placing of a large percentage of gutta-

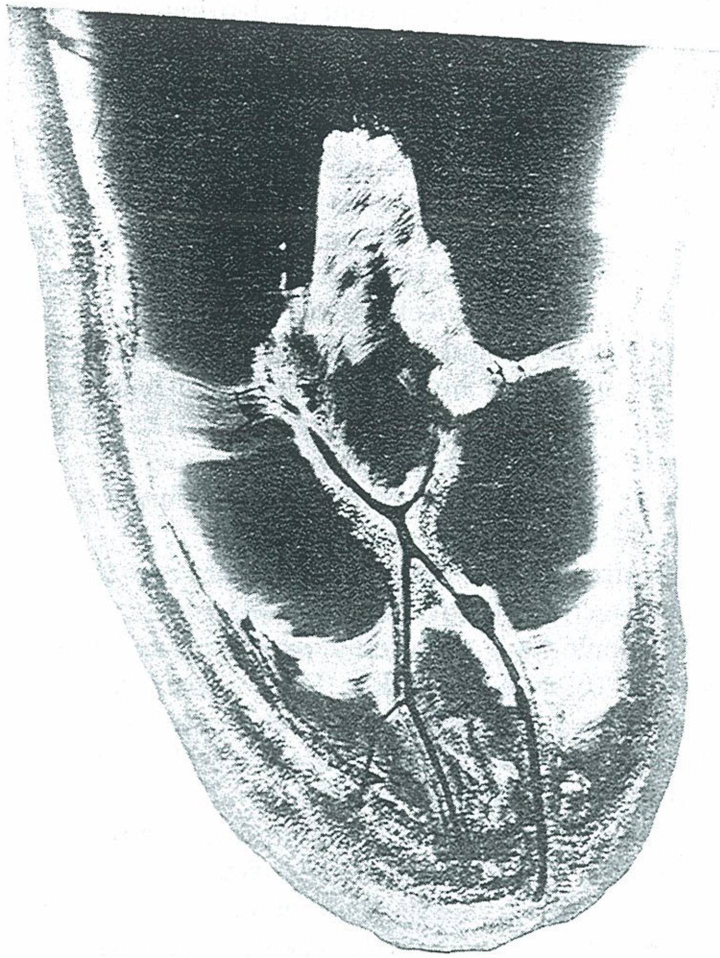


FIGURE 110. SHOWS THE MULTIPLE FORAMINA, BRANCHINGS, ENLARGEMENTS, AND CONSTRICTIONS IN THE ROOT APEX.

percha in the root filling, because of physical conditions: for example, in fan and wedge-shaped canals. No gutta-percha points are made fan-shaped. They are conical. Nor are they made wedge-shaped. The balance of the chamber, that cannot be filled with one or more of the conical points, will be filled with the solvent used; and in case of a lateral canal or several of them, it would clearly be impossible to work the hard gutta-percha into a cross canal, and particularly to make it fill the enlarged opening which many of the foramina have in the cementum.

In general, it may be stated that the amount of ultimate contraction will be the amount of solvent present, assuming that the pulp chamber was filled completely with the root filling and its solvent. The problem of the content of dentinal tubuli will be taken up in a subsequent chapter.

A large number of studies have been made of our own efforts at root-filling, to determine whether or not we had succeeded in filling the roots, and whether sterile teeth, when filled by us under ideal conditions outside the mouth, would remain sterile if placed in an infected culture medium; and in most instances, organisms were found in the tooth structure, in or beside, the root-filling material, or both, in a few weeks' time, and practically always after a period of a few months. Some of these studies are reported in detail in one of my articles referred to above. To assist me in this work, I engaged the service of Dr. Dayton C. Miller, Professor of Science, Case School of Applied Science, who made volumetric tests with a high degree of accuracy. The following are Dr. Miller's general remarks:

"The first noticeable fact in the experiments was the extreme slowness with which evaporation proceeded under spontaneous air drying. It would have required a year, or more, for the materials in the jar to have solidified. Even when heat of 70° C. (169° F.) was applied, the process was not much accelerated. A temperature of boiling water was required, and this was sufficient to melt the rosin and to soften the solid gutta-percha.

"It is not improbable that the rate of drying in a tooth cavity, the walls of which are more or less porous, would be different from that in a glass jar. It is possible that the body fluids may affect the rate; but experiments show that the placing of the material in water does not assist hardening, but rather prevents it.

"A conspicuous physical property of the materials is the great contraction in volume, the final volume in one instance being less than a fourth of the original volume.

"Both of the gutta-percha materials not only contract in volume, but in doing so they fail to adhere to the surface of the jar, leaving it clear of protecting covering, and the materials become porous or granular in structure. It would seem that these properties would render the materials useless for the filling of root cavities. As an illustration it may be assumed that root canals have diameters varying from 0.001 inch to 0.015 inch, that is from 0.025 millimeters to 0.375 millimeters, and that bacteria may have a size varying from 0.0005 mm. to 0.005 mm. The possible contraction of fluid gutta-percha root-filling material in the smallest root canal may be 0.005 mm. while for a large canal it

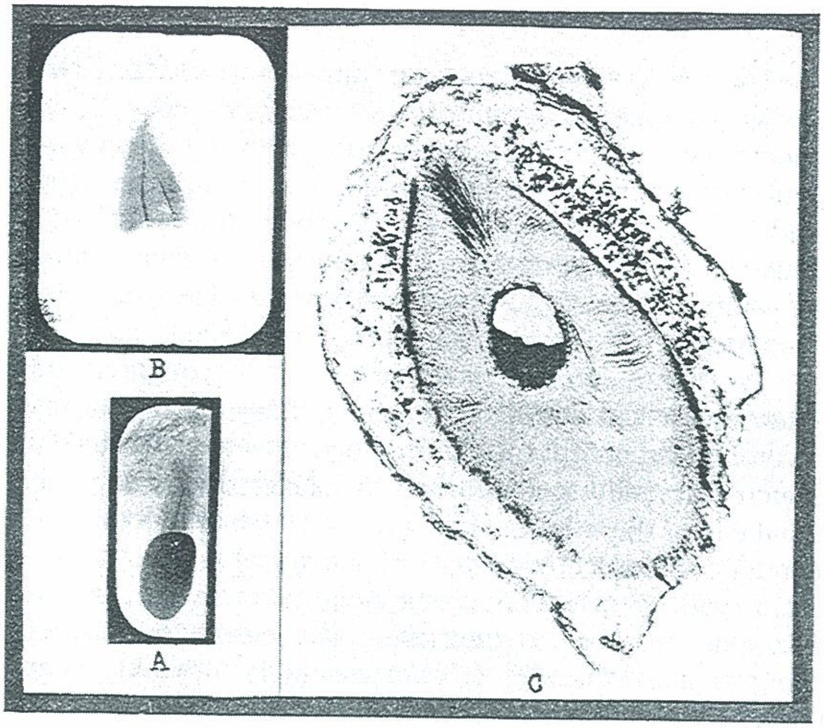


FIGURE 111. ROOT CANAL FILLING SHRINKAGE. A AS ROENTGENOGRAPHED IN THE MOUTH, B AFTER EXTRACTION, C A CROSS SECTION OF THE TOOTH AND ROOT FILLING.

may be over 0.075 mm.; these contractions are much larger than the sizes of bacteria. While these statements of the contractions are not exact because of the uncertainties in the manner of drying, yet it is certain that there will be little protection against the entrance of bacteria.

"The rosin solution when drying differs from the other in that a solid coating remains tenaciously adherent to the whole inner surface from which the fluid has receded, and the solid does not at any time part from the walls. Thus the whole inner surface of the cavity remains protected, and no cracks are left where bacteria may enter through the small canal. There is an inner hollow space, but it is wholly surrounded by the painted walls, and closed at the bottom by the solid.

"These conclusions, however, are not final in regard to the filling of teeth for the conditions in a living tooth are certainly different from those in the glass jar. But the facts of physical contractions, etc., are exactly the same in the two cases."

It is clearly evident also, from the clinical data available, that root canal fillings do not continue to fill the roots of teeth with as high a degree of efficiency, as is generally supposed. Figure 111 shows one of these root fillings, which in the original roentgenogram of the tooth in the mouth, gave the appearance of filling the canal. A shows a roentgenogram of the tooth in the mouth; B, a roentgenogram of the tooth after extraction, showing the mesio-distal appearance which, of course, could not be taken in the mouth; and C, a cross-section of the tooth above the branching.

These studies of the underlying fundamentals indicate how much more difficult it is to fill root canals mechanically than is generally supposed. If, however, a tooth is root-filled, which is surrounded by a non-infected vital membrane throughout its entire cementum, it will doubtless be very difficult for organisms to enter even unfilled dental tubuli, provided they are thoroughly sealed, if such is actually accomplished with chloroform and rosin at the pulpal end of the tubule. If, however, the dentin about the apex is denuded and infected as part of the apical and pulpal involvement, it will doubtless be more readily possible for that in-infection to gain entrance to, and infect, the dentin because of the continuous anastomosis. It is accordingly probable that favorable teeth, whose dentin is infected, but whose cementum is not infected, and has normal attachment, may have from ninety to ninety-nine per cent, possibly more, of the organisms destroyed by medication with Chloramin-T without considerable injury to the supporting structures, and that such teeth can, under most favorable conditions—namely, direct access, a round canal without islands, bays, or fan-shaped channels—be filled so that the great majority of the tubuli are sealed at their pulpal ends and the pulp chamber filled to within a few per cent of full the first few weeks, a larger per cent of unfilled space developing with the succeeding months with an ultimate space in even the most favorable root fillings adequate for the housing of many millions of bacteria, which, if they may find access to such an area, will be sufficient in quantity to do definite damage to the host, provided that individual's defensive activity against that organism is not sufficiently high, a matter which will be discussed in detail in subsequent chapters.

In the preceding chapter under the discussion of the efficiency of medication I have reported the result of planting the roots of a tooth beneath the skin of a rabbit, which had been as thoroughly root-filled as an unusually skilled operator was capable of doing after treating the teeth by a process which is proven to be as efficient as any we have available. The placing of these supposedly sterile teeth, which presumably could not because of their root fillings again become infected, underneath the skin of the rabbits, produced results which indicate that they were not sterile, and were not free from toxic irritation. That this was true, was further demonstrated by drilling into these roots and culturing after sterilizing the surface.

As a part of the daily routine of our research, extracted teeth, which are suspected of being related to systemic conditions, are cultured, some by crushing the root tip or the whole tooth, many by sterilizing the surface and drilling into the tooth structure with a sterile drill and culturing the chips, some by sterilizing the external surface and culturing the tooth. Of the last thousand teeth, less than one-tenth of one per cent of all of our root-filled teeth so tested, failed to grow a culture of streptococci.

SUMMARY AND CONCLUSIONS.

When we consider how many thousands of the extracted teeth we have cultured and found to be infected within the tooth structure, and the extremely low percentage, practically zero, in which infection was not demonstrated, together with the fact, that so many teeth with excellent root fillings, show structural changes of the supporting tissues after a few years have elapsed, we are led to believe that we are dealing in these cases with one of the clinical expressions of the physical facts we have just been studying.

We are, therefore, though very reluctantly, compelled to change our original premise which provided that, *good root fillings fill pulp chambers and continue to do so indefinitely*, to the following:

> **Root fillings rarely fill pulp canals sufficiently perfectly to shut out bacteria completely. Root fillings usually fill the pulp canal much less perfectly some time after the operation, than at the time of the operation, due to the contraction of the root-filling material. The ultimate volume contraction of the root filling is approximately the amount of solvent used where a solvent is used with gutta-percha as a root-filling material.**

(Note:) It is not proven that it is absolutely necessary that teeth be perfectly sterilized or that they be perfectly root-filled in order that an individual may not develop systemic involvement, since under favorable conditions the patient may provide an adequate defense or quarantine against these materials.

CHAPTER XVI.

COMFORT AND SERVICEABILITY AS SYMPTOMS.

PROBLEM: Are local comfort and efficiency of treated teeth an evidence and measure of the success of an operation?

EXPERIMENTAL AND DISCUSSION.

Clinical diagnosis as a science is based upon symptomatology. If we were to ask the question of the dentists, oral surgeons, and physicians of the country, the great majority would give the affirmative answer to the above question; as would also nearly 100 per cent of the laity. The special research on this problem has been undertaken to determine the relationships between symptomatology, clinical pathology, and tissue pathology. The approach has been made in three ways: First, by comparing the history and symptoms with the presence or absence of evidence of systemic involvement; second, by comparing the history with the local histopathology; and third, by tabulating a large number of cases to determine the type of condition which, when obliterated, produced a change in systemic reaction, including both the groups of disturbances with and without evidences of local discomfort.

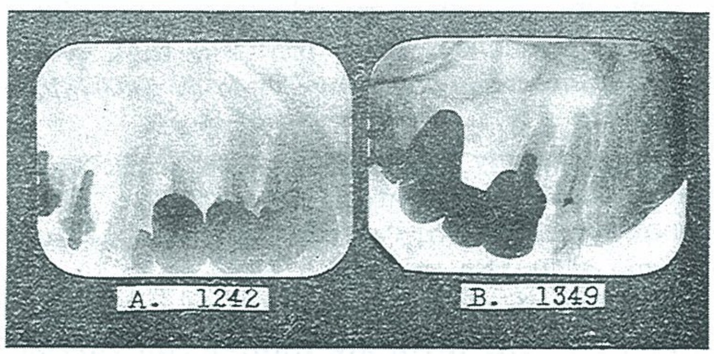


FIGURE 112. INFECTED LATERALS WHICH HAVE GIVEN FREQUENT AND SEVERE DISCOMFORT.

Figure 112 shows roentgenograms of teeth of individuals that reported either continual or frequently recurring tenderness of the teeth here shown. The lateral in A has been crowned for some years, with recurring acute tenderness and finally with a violent abscess which produced extreme discomfort and swelling.

B shows a similar lateral which has a partial root filling, as has also the cuspid. The lateral has had recurring apical abscesses developing for a long period, the tooth becoming very severely inflamed with each recurrence.

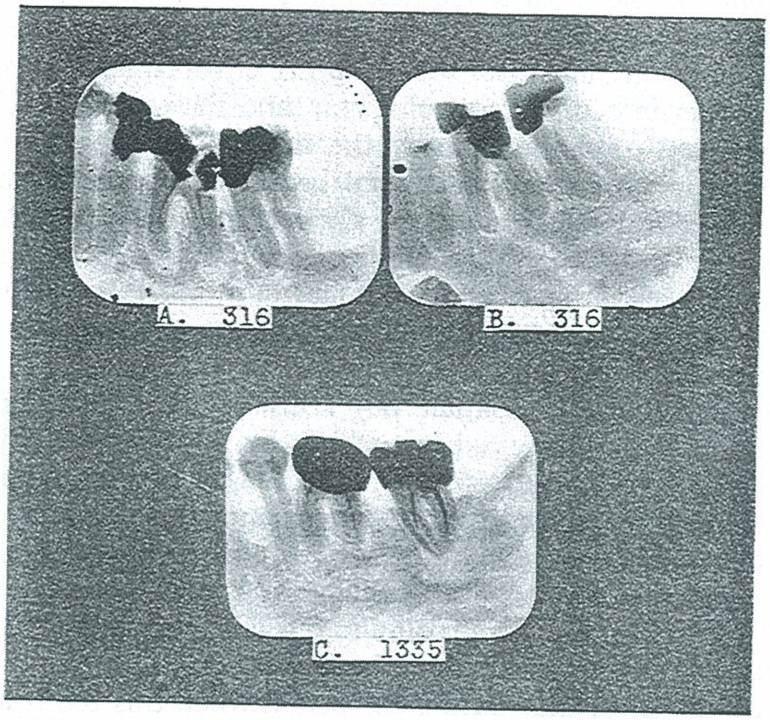


FIGURE 113. TEETH FORMERLY WITH FISTULAE AND RECURRING TENDERNESS, LATTERLY COMFORTABLE. ALL BADLY INFECTED.

In Figure 113, we have roentgenograms of the teeth of a group of cases with the following history: For a more or less extended period of time these teeth were, on recurring occasions, tender or had what the patients termed gum boils. Sometimes this changing condition extended over a period of years, and finally the teeth ceased to become tender. In A the tooth had a fistula which would occasionally close; the tooth would become tender; the fistula would open, establishing free discharge into the mouth; and the tenderness would subside. We insisted upon the removal of this tooth. The patient always delayed the operation and finally stayed away a couple of years for fear we would carry out the program. Finally the fistula closed; the tooth ceased to become tender; and the area of absorption became smaller as shown in B. The patient began to develop symptoms of rheumatism and consented to its removal. The rheumatic symptoms were relieved.

Figure 113-C shows a lower right second molar filled many years ago, and previous to about three years ago very frequently gave the patient trouble, with tenderness and some swelling. About that time she contracted "Flu" which was complicated with a lung involvement and also with a heart involvement, which latter kept her in bed for most of a year, and she was an invalid for a year and one-half. During the time of her attack with "Flu" and the subsequent lung and heart complications, and also since these latter had subsided, since she had had no tenderness whatever in this tooth, she had come to the conclusion that there was no need for its extraction, a matter in which her dentist concurred notwithstanding he had a roentgenogram and knew the history.

In another chapter (Chapter 60) I discuss the relationship between the susceptibility to heart which this lady inherited, five of the relatives on the father's side, including her father, having died in middle life with heart involvement; and in the chapter on the relation of condensing and rarefying osteitis, I have discussed the significance of the zone of condensation, which appears in this roentgenogram, and its relation to this clinical history. (Local Structural Changes. Condensing around Rarefying). The facts are very clear, that during the period when this tooth was giving serious and frequent local disturbance, the patient was relatively more safe, than in the later period during which it gave no response and was for all intents and purposes a normal tooth. The reason for this is discussed in the chapter on the nature and significance of local reaction.

In Figure 114 we have a group of crowned centrals showing even greater areas of pulp canal unfilled, which, according to the history, have never been uncomfortable, and the patients were able to eat with them without thought or consideration. A presents the roentgenographic appearance of the teeth of a middle aged man who was suffering so acutely from rheumatism that for months he had walked with great difficulty, besides which he was suffering severely and was almost incapacitated from his work. With the removal of these teeth, his symptoms were completely relieved and have not returned for five years. He states that he would not have them back for a million dollars. (Incidentally,

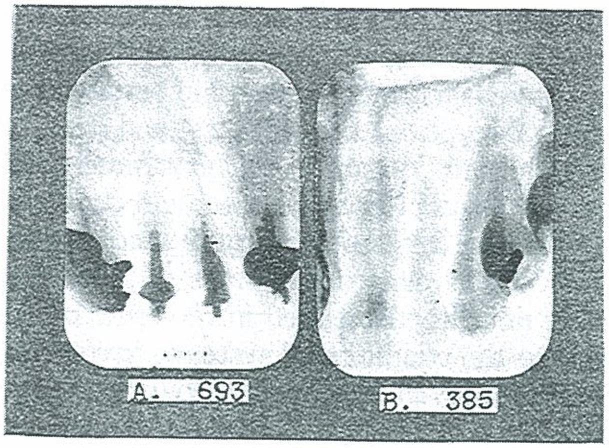


FIGURE 114. INFECTED TEETH WITH NO HISTORY OF DISCOMFORT.

it is of interest to note that the patient reported that his former dentist, following the regulation basis of interpretation, insisted that these teeth could not be related to his condition without showing more evidence of involvement. He also stated that he would eat his own hat if the man were any better after their removal. I have not learned whether he has yet done so but the patient insists that this is his treat.) The significant thing about this patient's teeth in connection with this present study is that they never were in the slightest tender and he had no suggestion or indication of an abnormality or infection.

Figure 114-B shows the teeth of a young lady suffering very severely from rheumatism and heart involvement with no local symptom of tenderness about these teeth, but which were infected, and after their removal with some others her symptoms so completely disappeared that she was able again to work, which she had not been able to do for many months, and in five years has had no recurrence. These two patients belong to the group of strongly inherited susceptibility. On the basis of comfort there was no suggestion whatever that these teeth were not in perfectly safe condition, notwithstanding the lateral contained a putrescent pulp of long standing.

These three groups of illustrations cannot be considered as proving anything since they represent such a few cases. However, they are selected simply as typical of large groups, and, therefore, are significant as typical illustrations. Those in Figure 112 are all classed in the non-susceptible group; those in Figure 113, the acquired group; and those in Figure 114, the strongly inherited group.

SUMMARY AND CONCLUSIONS.

An analysis of these four groups of studies suggests to us the following: That there is a direct relationship between the extent of rarefaction or absorption about roots and the condition of comfort; that this condition of absorption is directly related to the recurring acute inflammatory processes; and conversely, that the absence of extensive areas of absorption accompanies cases without a history of recurring acute processes, and that these two conditions are not directly related in the order of the extent of the absorption to the condition of systemic involvement, but inversely so; for with a given infection, the patients, with a large area of absorption and a history of recurring tenderness of the tooth, have been free from systemic involvements; and those with relatively small areas of absorption in proportion to the capacity of the tooth for infection, have proven to be the patients with systemic involvement. We shall give our interpretation of these phenomena in Chapters 45 to 56.

We would, therefore, reverse the accepted fundamental that comfort and serviceability are criteria of the success of an operation or the dangerousness of a tooth to the safety of a patient to the following:

> **Local comfort not only is not a certain index of success or safety but constitutes both what is probably the greatest paradox and the costliest mistake through loss of health and life that exists in both dental and medical practice, because it may only mean the absence of local reaction which would, if present, incidentally make the tooth sore and fundamentally destroy the infection at its source; whereas, the absence of this local reaction and its consequent destruction of the infection products permits them to pass throughout the body to irritate and break down that patient's most susceptible tissue, which tissue can be anticipated very frequently.**

CHAPTER XVII.

QUANTITY, SYSTEMIC EFFECT, AND TOOTH CAPACITY.

PROBLEM: What is the relationship of the quantity of dental infection to the type and extent of systemic involvement?

EXPERIMENTAL AND DISCUSSION.

That the problem of systemic involvement is essentially the problem of the overwhelming of the natural defenses by a large quantity of dental infection, is a fundamental that seems to be quite universal in its general acceptance. While it is recognized that there is a great difference in the virulence of organisms from various sources, since the capacity of a tooth is so small it is not deemed possible that a properly root-filled tooth will have capacity enough for sufficient dental infection to overwhelm the patient; and furthermore, the organisms constituting dental infections have been reported by so many writers to be of so low virulence, that the professional mind has come to accept that a fundamental requisite for a tooth to be a source of serious involvement will be that there is present, in addition to the infection in the tooth, a quantity of infection in what is considered an abscess or pus sac at the root apex, and which, because of its capacity, comes to be a menace and possible danger. Howe states in his article "The Focal Theory of Infection in its Application to the Teeth"[9] the following:

"In the experimental work that is used to support this theory, young animals, intravenous injections, and massive doses are used. It is replied to the criticisms that these doses are excessive and do not fairly represent what occurs in the human body, that, at times, it is possible to produce the desired pathological effect with smaller doses. These doses range from what in the case of man of 70 kilos. or 154 lbs. would be from ten quarts to a cupful. What is termed a small dose is one or two cupfuls of microörganisms in the case of a man of 154 lbs. Pure cultures are not

[9] See bibliography.

injected. The granuloma, the abscess, or pyorrheal pus, and the pulp are dropped bodily into ascetic-dextrose broth, allowed to grow, and the broth containing the conglomerate mixture of bacteria, altered and decomposing tissue and its morbid products are injected en masse into animals. . . . . . .Now we do not often deal with doses ranging from a cupful to five or six quarts from dental sources. Yet, to produce such results as are described, they must be used. In the small dose of a cupful of microörganisms, together with the other putrefactive products mentioned, possibly or occasionally an effect is obtained. . . . .Others have inoculated rabbits with ten to fifteen c.c. doses of streptococci of this type for three months and nothing has happened."

These researches have been conducted to ascertain what the quantity of dental infection may be in various cases, and how much of it may be sufficient to produce systemic involvement, and what may be the capacity of a tooth even with a good root filling as a reservoir or bacterial and toxin generator. Our first problem has been to determine the capacity of the tooth. To do this, we have taken freshly extracted, root-filled teeth, removed all excess moisture, and weighed them. We have extracted the moisture by placing them in a chamber in which the moisture of the air was being extracted by such chemicals as sulphuric acid, none of which came in contact with the tooth. The teeth were again weighed, re-soaked in water, weighed, and again re-dehydrated without carrying the dehydration to the point of removing water of crystallization. Figure 115 shows a group of such teeth; and it is found, in general, that approximately 5% of the volume of every root-filled tooth is a fluid which may be a culture medium and may be abundantly saturated with bacteria.

CHANGE IN WEIGHT OF TOOTH STRUCTURE

Fresh Tooth	Dehydration		$CaCl^2$	H^2O	Net Gain %	Net Loss %
	Weight Before	Weight After	M / 0.00706			
No. I	0.9940	0.8910	0.9188		3.02%	
	0.6124	0.5801		0.5770		1.56
No. II	1.0372	0.9280	0.9280		1.33	
	0.8635	0.7500		0.7424		1.01

FIGURE 115.

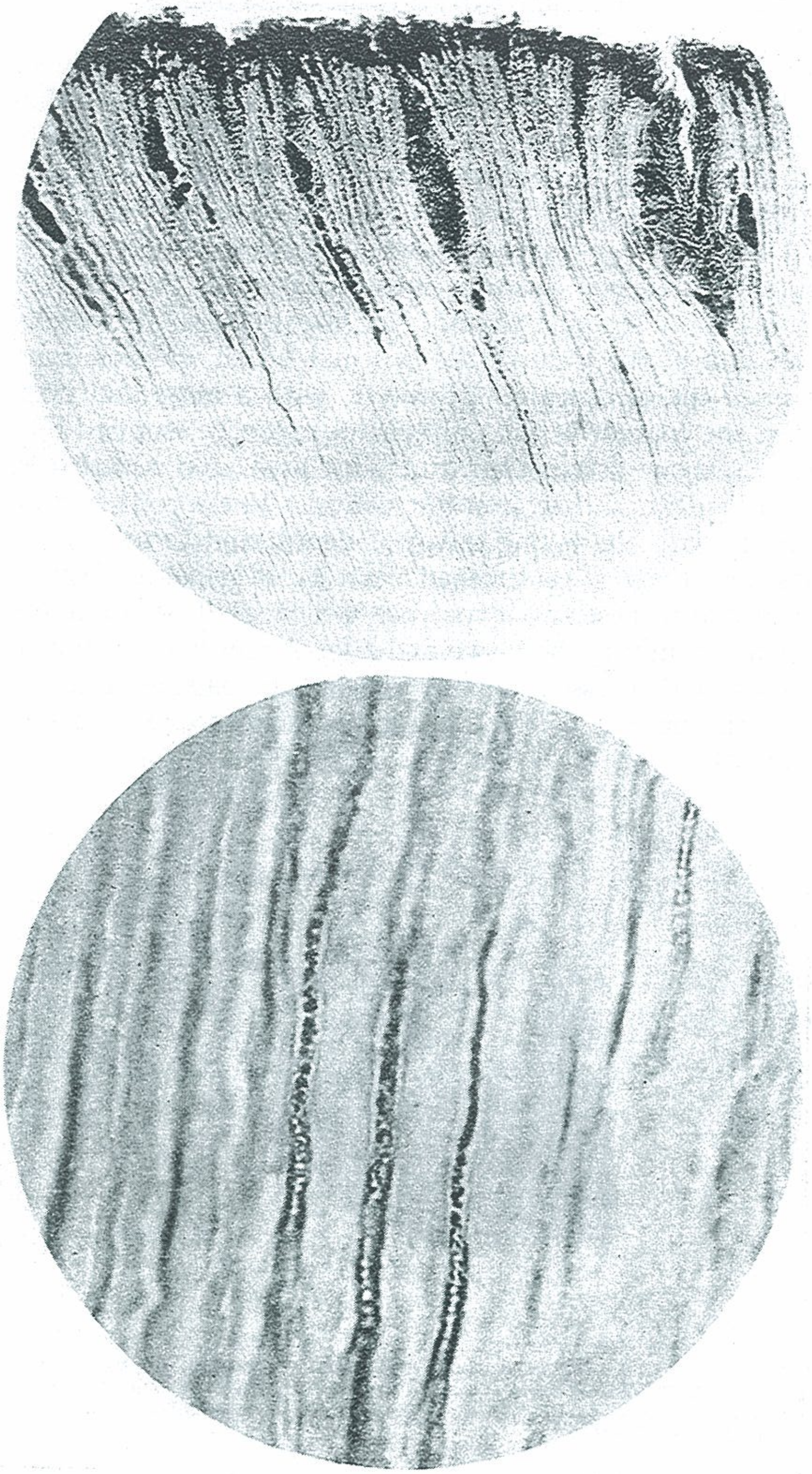


FIGURE 116. DENTAL CARIES EXTENDING ALONG THE TUBULI TOWARD THE PULP. UPPER IS CAVITY END OF TUBULI, LOWER NEAR PULP.

Figure 116 shows the organisms stained directly in the tubuli of the teeth. Many of the earliest efforts to stain bacteria within dental tubuli were confused by the photographing of the abrasive in the tubuli, which, with modern methods of staining, can be readily differentiated from bacteria; and besides, the tissue in Figure 116 was not ground but was decalcified, and there was no opportunity for foreign substances to contaminate the tissue.

The capacity of the pulp chamber for infection may be more or less than that of the total of the dentinal tubuli and acini and canaliculi of the cementum. However, with a large and open root apex, the content of the pulp chamber can be emptied into the tissue without destruction of a large area, and hence with greater concentration than from the dentin. In the construction of the tooth with the laying down of dentin and cementum in juxtaposition, there is established what is, in general effect, a fire-wall between these two structures, which, while it has many small openings uniting these two and a few larger ones, as multiple foramina and accessory canals, impedes the passage from the dentin to the patient of the infection and toxic substances in large amounts.

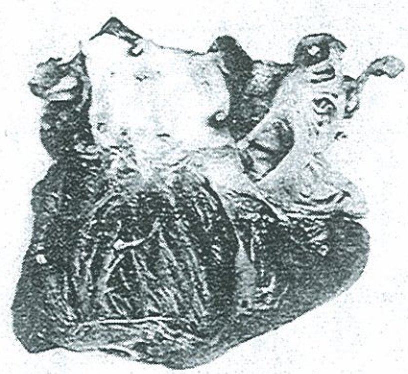


FIGURE 117. ACUTE ENDOCARDITIS. WITH EXTENSIVE VEGETATION ON VALVE CUSP, PRODUCED IN A RABBIT'S HEART BY THE INTRAVENOUS INJECTION OF THE ORGANISMS WASHED FROM A SINGLE ROOT-FILLED TOOTH. THESE ORGANISMS WEIGHED APPROXIMATELY ONE MILLIONTH PART OF A GRAM.

Another phase of this study has included the injection into experimental animals of the organisms that could be washed from a single root-filled tooth. Figure 117 is typical. It shows the heart of a rabbit which has developed acute endocarditis from the injection into the rabbit of the settled washings of the crushings of the tooth of a patient suffering from acute endocarditis. The weight of the organisms injected into this rabbit was determined by counting the number present in a known dilution and, by calculation, establishing the total number approximately, which was found to be a millionth part of a gram.

To answer the question whether there are possibilities of other substances than organisms having been taken from the teeth which produced these disturbances, we have made a large series of studies to ascertain whether toxic substances other than organisms are present in teeth. This we will study in the next chapter. We have also centrifuged the organisms, removed the fluid, and resuspended them in normal salt solution and inoculated them into rabbits, and have produced lesions and death of animals.

An analysis of several hundred individuals from whom teeth were removed having root fillings without evidence of periapical chambers containing infected granulomata, discloses that in a large number there has been a very marked, if not complete, relief of the systemic expression upon the removal of the tooth in question.

To determine whether or not a quantity of infection must be injected into a rabbit which would be equivalent to a "tea-cupful or several quarts per man" I made the following experiment: Into many dozens of rabbits we have planted beneath the skin a freshly extracted tooth to determine what the effect would be of the small amount of infection that a single tooth could contain. These have revealed a very great difference in the virulence of different strains and the type of reaction they would produce. In the chapter on kidney involvements we show several cases in which the planted tooth produced nephritis in the rabbit without the development of an extensive abscess about the implanted tooth, and in some cases the same tooth transferred to another rabbit again produced kidney involvements. In many cases the teeth become incysted. Many of these are discussed in subsequent chapters. Still others produce wasting diseases and the rabbit slowly fails in weight and finally dies. Others produce most violent toxic and bacterial reactions, causing the death of the rabbits in from a few days to a few weeks.

Figure 118 shows a roentgenogram and photograph of a tooth extracted from a patient suffering from a severe systemic involvement of the central nervous system, not suspected to be related to her teeth. This is discussed in further detail in Chapter 66.

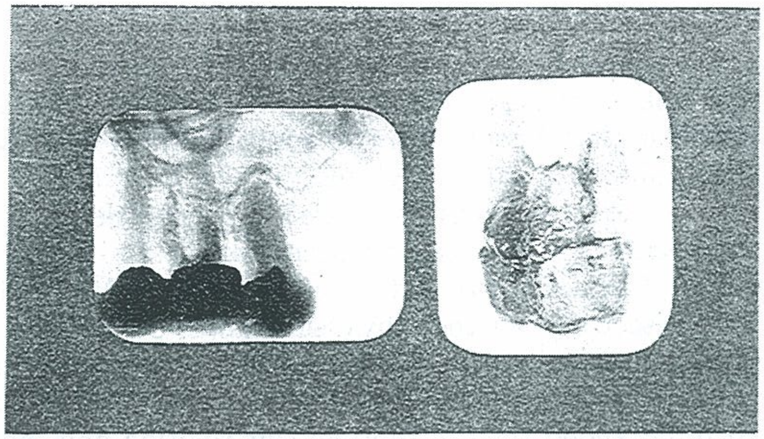


FIGURE 118. TWO VIEWS OF A MOLAR TOOTH PRODUCING NO LOCAL DISCOMFORT FOR THE PATIENT BUT WHICH KILLED MANY RABBITS IN SUCCESSION, IN AN AVERAGE OF FOUR DAYS.

This tooth was placed under the skins of thirty-one different rabbits, twenty-nine of which died spontaneously in from one and one-half to ten days, an average of four days. One of the rabbits which lived ten days, received special treatment to increase its defense. These all died in general of the same symptoms. Another rabbit (Rabbit 986) of this series was injected intravenously with 0.03 cc. of the fluid surrounding the tooth in one of the rabbits. This rabbit was dead in about one and one-half minutes. Another (Rabbit 989) was injected with the highly centrifuged material, which would not be entirely bacteria-free but nearly so, and it was dead in about seven hours. Another (Rabbit 991) was injected intradurally with the bacteria-free filtrate of this material and it died within twenty-one hours. The symptoms and reactions in the animals are discussed in the chapter on diseases of the nervous system, Chapter No. 66.

In order further to study the relation of quantity, very small glass tubes, approximately the size of a fine cambric needle and three-quarters of an inch long, were filled with this material. These open end tubes were placed inside the needle of a hypodermic needle and passed under the skin of a rabbit; and as the needle was withdrawn, the tubes were placed beneath the skin. The quantity of the culture fluid that these needles contained was approximately one five-hundredth of a gram each. From one to four of these small tubes were placed beneath the skins of rabbits and several were used for different types of tests. When placed in the peritoneum, Rabbit 1010 lost 585 grams, or 40 per cent of its weight. It died in 54 days with extreme muscle atrophy.

When placed subcutaneously, Rabbit 1011 lost 460 grams, or 33 per cent, in 38 days. It died of lobar pneumonia, with congestion of the myocardium. Another (Rabbit 1012) with a single tube placed beneath the skin, died in 5 days, with a loss of 75 grams, or 7 per cent, with a very large subcutaneous abscess, and hyperemia of the chest and abdominal viscera.

In order still further to determine this problem, two small round cover slips were sealed together with sodium silicate (liquid silex) and two very small openings left at opposite sides for the organisms to escape. The distance between these cover slips was approximately a fortieth of a millimeter. The area of the plane of fluid was approximately one square centimeter. Hence this quantity was approximately 2.5 milligrams of culture fluid. This exceedingly minute quantity of infection was sufficient to kill this rabbit in 44 days. (Rabbit 995).

A further study of this tooth was made to ascertain the relationship between the size and number of external openings to the danger, as evidenced by the length of time required for the tooth to kill a rabbit after it was planted beneath the skin. It was found, in general, that increasing the number of exits from the dentin increased the toxicity of the tooth apparently by giving a freer exit to the toxic substances. In this connection I think I should state a warning because the need of it has already been evidenced. Immature thinking or experience may suggest that the transferring of the tooth from one rabbit to another is synonymous and comparable with the animal passage of organisms injected intravenously or subcutaneously in lethal amounts, the result of which is to increase the virulence of the organism as it is successively killing the host. In that instance all organisms injected in subsequent animals are direct descendants of those that have lived in the environment of the animal with a lowered resistance, which animal has furnished the entire culture medium for the organisms, and which, because of the quality of adaptation expressed in Chapter 2, induces the organism to increase its aggressive factors. When, however, a tooth is planted beneath the skin of a rabbit and kills the rabbit in two days, and the tooth is then washed and placed under the skin of another rabbit, the organisms in the tooth which are planted in the second rabbit, did not grow in the free body fluids of the dying animal but in the incased moisture within the approximately three miles of closed channels constituting chiefly the dentin, and these organisms have been slightly, if at all, influenced by the presence of the tooth beneath the skin of the rabbit, and practically none of which are the descendants of those causing the death of the animal, or at least producing the infection within the animal's body. The passage of a tooth, therefore, from animal to animal is little more than changing its incubator and does not constitute animal passage in the sense that intravenous injection of a culture is understood.

I have, therefore, conducted experiments to obtain data, if possible, that would indicate whether it is possible for especially vigorous and healthy animals to destroy the virulence of the organisms within an infected tooth. There seems no indication that even the most vigorous animals have been able to render a tooth harmless which had been demonstrated to be capable of producing the death of an animal; and while an infected tooth when placed beneath the skin of a rabbit, kills small animals in proportionally less time than large ones, we have not been able to find a rabbit big enough to withstand the toxic substance of certain infected teeth. I have discussed elsewhere the circumstance of placing such a tooth under the skin of a very large and exceedingly vigorous buck rabbit, that was a most vicious fighter, seriously wounding and killing any other male rabbits he could reach. On placing such a tooth beneath his skin, in a few hours he was sulking in a corner of his cage, and was dead in five days.

This seems clearly to demonstrate that there is a direct relationship between the accessibility of the rabbit or host to the organisms confined within the tooth structure, for it seems very clear that the dentino-cemental junction seems a very considerable barrier. To determine this we have made the following experiment:

Various chemicals and infected culture media have been sealed in the pulp chambers of teeth to ascertain the nature of the substances that were capable of penetrating different structures and parts of the tooth. As discussed in the previous chapter, when a culture of acid producing bacteria is placed in the pulp chamber of a tooth, and the tooth planted in a jellied culture medium, carrying a disclosing die, the presence of the organisms can be read directly by the color change in the surrounding medium. These showed that the organisms leave the tooth only at foramina. When, however, chemicals are used containing electrolytes which are capable of passing through semipermeable membranes, these may pass through the dentin and cementum, or at least appear on all surfaces of the cementum as well as the foramina. This was also demonstrated by sealing the apex.

FIGURE 119. SHOWS A SMALL GLASS TUBE CARRYING CHIPS FROM AN INFECTED TOOTH. ITS SIZE CAN BE JUDGED BY COMPARISON WITH THE TOOTH.

One of these glass tubes containing approximately one milligram of the drillings of this tooth is shown beside the tooth in Figure 119; and one of the tubes which was planted beneath the rabbit's skin and open at both ends, containing some of the culture media from the organism growing in this tooth had about the same capacity, being longer but smaller in diameter.

When we compute the actual weight of the organisms, not dry but moist, which were actually found to be capable of killing some of these rabbits, we find it to be approximately one five-hundred-thousandth part of a gram. If we allow that a man is twenty times as large as a rabbit, which is the usual computation, we have a quantity which might be expected to be injurious to man, if not seriously so, of one twenty-five-thousandth part of a gram. Or when we compute the quantity of this culture medium which produced death in these rabbits, approximately one-thousandth part of a gram to kill a rabbit in one and one-half days, and extending it twenty times that might be expected to be mildly or seriously injurious to a man, we find this quantity to be: namely, 20 cubic millimeters (20 milligrams). This figure does not check very closely with Dr. Howe's findings, since a tea-cupful contains about 250 grams or 250,000 milligrams. In other words, the dose that we have found to be sufficient to kill a rabbit is about 1/250,000th part of the minimum amount Dr. Howe has suggested and 1/12,500th part of the minimum amount he has computed to be necessary to injure a person, using his minimum quantity of one tea-cupful instead of his maximum, which he states to be several quarts. As we have stated above, and as we show

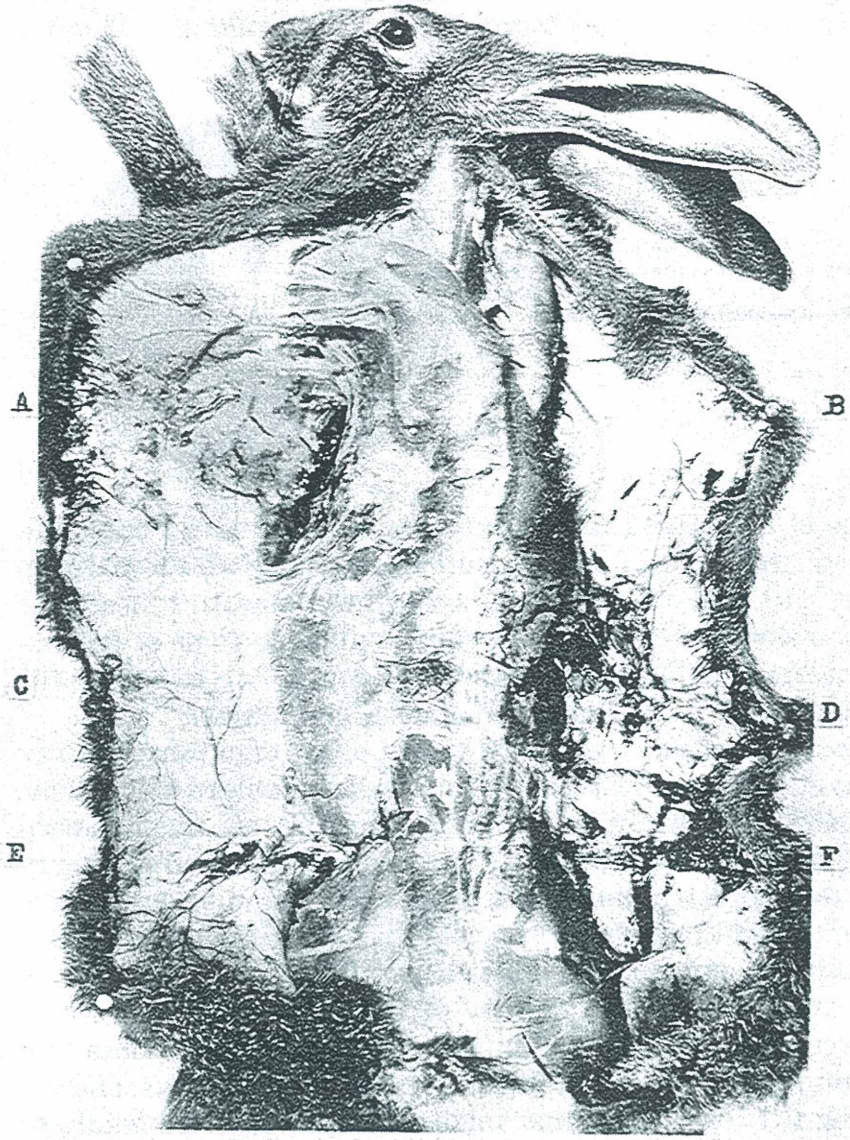


FIGURE 120. RABBIT REACTIONS TO OPEN END TUBES CARRYING DIFFERENT SUBSTANCES. A, VIRULENT CULTURE; B, SAME ORGANISMS WITH TWO PER CENT PHENOL; C, NORMAL SALT SOLUTION; D, ANOTHER DENTAL CULTURE; E, FILTRATE FROM CULTURE A; F, SUPERNATANT FLUID FROM CENTRIFUGED A.

in other chapters, many of the teeth that are planted beneath the skins of rabbits, produce very little disturbance, while others produce very violent disturbance locally and systemically; and still others apparently produce very little disturbance locally where planted, but systemically develop nephritis and heart involvements, as abundantly illustrated elsewhere.

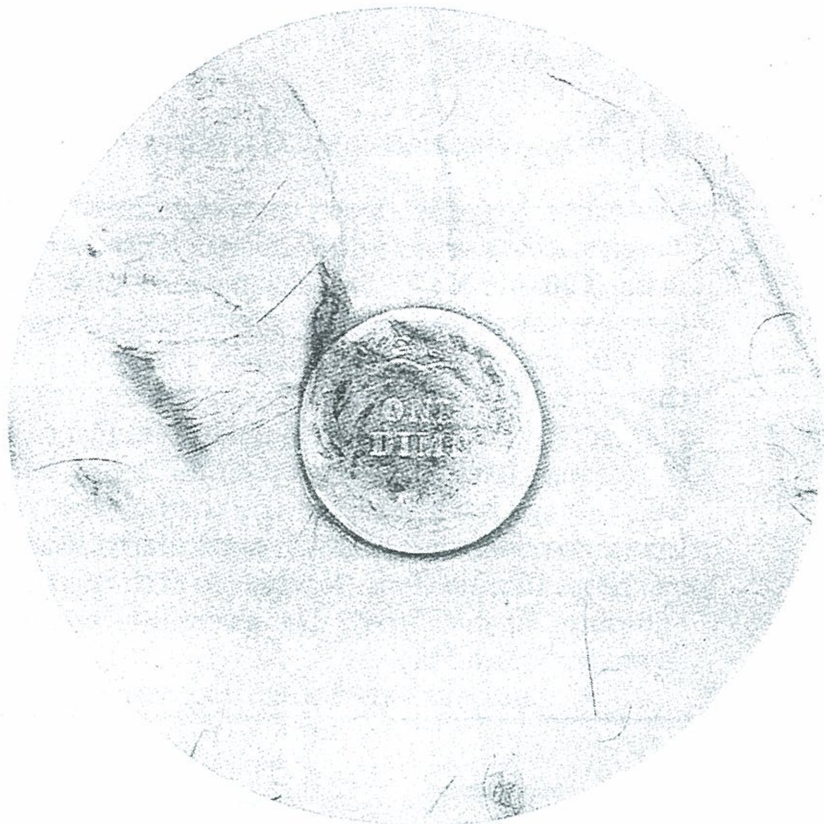


Figure 121. Appearance of a sterile coin two months after implanting beneath a rabbit's skin. No fibrous capsule formation or irritation. A slight etching of the coin.

It doubtless will be argued that these teeth planted under the skin of a rabbit, or the glass tubes or any foreign substance, would act as a serious irritant and produce local disturbance if not suppuration. To determine this, at the same time as the tubes shown in Figure 120 showing the abscess produced by the presence of the tube of this culture, similar tubes were planted beneath the skin on other parts of the back of this same rabbit. The large abscess referred to above is shown at A. At B were planted the same organisms but carrying a small quantity of phenol, approximately two per cent; at C, similar tubes filled with normal salt solution; at D, a similar quantity of the organisms grown from a tooth of another patient; at E, tubes containing filtrate of the same culture of A; at F, the supernatant fluid from the highly centrifuged culture used in A. Serious disturbance was produced in the rabbit by only the culture shown at A. (Figure 120).

As a further test of the result of a foreign irritant, and particularly that we might study the difference in the structure of the tissue which would be organizing about a sterile foreign substance and an infected tooth, we planted sterile coins beneath the skin of a rabbit and have sectioned the membranes that have developed. These are considered in the chapter on the structure of granulomata, (Chapter No. 38); and, incidentally, those experiments reveal that even after two months' time a sterile dime, placed beneath the skin, has so little tissue organized about it, that, except that there is no sign of the skin lesion through which the dime was planted, it would seem as though it were planted there but a day or two ago, for the inscription on the metal can very readily be read through the thickness of this tissue, which is an exceedingly thin transparent veil. This is particularly instructive since there has been sufficient local reaction to etch the surface of the dime and quite largely remove the inscription and lettering. This is shown in Figure 121.

RABBITS WITH SUBDERMAL IMPLANTATIONS

Implantations	No. In Group	Spontaneous Death		Chloroform		Still Living	
		No.	%	No.	%	No.	%
Subdermal implantations	237	181	77	30	12	26	11
Total implantations of teeth	209	165	79	26	13	18	8
Sterile coins	6	No local reactions					
Capillary tubes	13	7	53	2	15	4	32
Other substances	9	5	56	0	0	4	44
Maximum No. of implantations of teeth from 1 patient	60	58	97	2	3	0	0
Maximum No. of implantations of a single tooth	35	33	94		6	0	0

FIGURE 122.

In order further to test the relation of quantity of infection and systemic reaction, it has been desirable to check against the infected teeth, the general effect of foreign substances, sterile and otherwise. Accordingly, a number of substances have been tested. In Figure 122 we show a group of rabbits with subdermal implantations of various kinds. In the group there were two hundred thirty-seven rabbits, two hundred nine of which had teeth of various types implanted beneath the skin. Of this number one hundred sixty-five or 79 per cent, are dead at the time of this writing, having died spontaneously, and twenty-six or 13 per cent, have been chloroformed; eighteen, or 8 per cent are still living. Most of these teeth had been root-filled and were comfortable, so far as the patient was concerned, when they were extracted. Some were normal in health and structure, but malposed, such as third molars; some of these, as noted elsewhere, were removed impactions. Others were treated in various ways such as boiling, sterilizing with medicaments, etc. Even impacted teeth are not necessarily sterile though they probably generally are. In six cases sterile coins were used. In these there was no local reaction. Thirteen capillary tubes containing infected material were implanted, of which seven rabbits, or 53 per cent, died spontaneously. Miscellaneous substances were used in nine rabbits. In this chart, it will be noted that in order to compare the virulence of different teeth taken from the same patient, six different teeth were successively implanted in sixty rabbits. Fifty-eight, or 97 per cent, died spontaneously, and two, or 3 per cent, were chloroformed. The maximum number of implantations made with a single tooth was thirty-five of which thirty-three rabbits, or 94 per cent, died spontaneously, and two rabbits, or 6 per cent, were chloroformed.

It will naturally be argued that experiments made on animals may have little significance in interpreting human defense. In the chapter on bactericidal properties of the blood I have shown the relative defense of various animals, as compared to the human, for streptococcal infection as determined by the methods used; and it is important that few humans have as high a defense for the type of dental organism taken from infected teeth as do the rabbits. Rats and mice have a still higher defense.

SUMMARY AND CONCLUSIONS.

An analysis of these data suggests that pulpless teeth, whether root-filled or not, may, if the dental tubuli are not filled, be reservoirs of culture media, not only constituting approximately one-fifth of the total volume and weight of the tooth, but that this quantity is ample to produce very marked systemic disturbance; that the quantity of infection in such a tooth is sufficient to produce definite lesions in animals and even to produce their death. The clinical reaction by the removal of the systemic symptoms suggests that the quantity of infection was also enough, seriously to affect the patient.

We are, therefore, compelled to change the accepted fundamental that *infected teeth cannot, if the pulp chamber is properly filled, contain infection sufficient seriously to affect the patient,* to the following:

Since approximately 5% of every root-filled tooth is a fluid that can become toxic substance or culture media, and in single rooted teeth usually amounts

to about fifty milligrams, which space may be occupied by organisms which have access to it from either the blood or lymph stream or an exposed surface of dentin, and since death from typical heart lesions has been produced in animals inoculated with the relatively small quantity of bacteria, washed from a crushed tooth, taken from a patient suffering from acute heart involvement, constituting an amount of organisms which, by weight, is approximately a millionth part of a gram, and since the bacteria-free toxin washed from crushed teeth frequently produces very extreme systemic changes, I believe a single rooted tooth with a well filled pulp canal can, under certain conditions, be a source of systemic disturbance. I am also led to this conclusion by our clinical experience with patients.

When infected teeth produce disturbance in other parts of the body, it is not necessary that the quantity of infection be large, nor is it demonstrated that it is necessary that organisms pass throughout the body or to the special tissues involved, but the evidence at hand strongly suggests that soluble poisons may pass from the infected teeth to the lymph or blood circulation, or both, and produce systemic disturbance entirely out of proportion to the quantity of poison involved. The evidence indicates that this toxic substance may under certain circumstances sensitize the body or special tissues so that very small quantities of the organisms, which produce that toxin, may produce very marked reactions and disturbances.

CHAPTER XVIII.

STUDIES OF PULPLESS TEETH, WITH AND WITHOUT ROOT FILLINGS.

PROBLEM: Have pulpless teeth injurious contents other than microörganisms?

EXPERIMENTAL AND DISCUSSION.

A search of the literature has failed to reveal any new light on this question. We find no evidence that teeth contain other injurious substances than bacteria; nor do we find evidence that such a research has been made. A large number of studies has been made of the organisms which develop in infected teeth, both as regards their identity and their biologic and pathologic qualities. The general procedure has been to take the organisms from the suspected teeth by any one of several procedures, culture them in suitable media, and test their reactions on various sugars and on animals as well as determine their morphologic characteristics. In this way, a few organisms are increased into a large number, presumably of their kind, with the effect that the studies are made with new generations grown in new media in a new environment; and if many strains are present, those that can best adapt themselves to the new medium environment, will necessarily develop most rapidly and largely overgrow the less adaptable types.

One of our earliest studies in this connection has been the injection into animals of the washings from freshly extracted and finely crushed, individual teeth. Figure 123 is typical. It shows two rabbits, full brothers, weighing within a few grams of each other, kept continually under the same environment since birth and fed on the same nourishment. A was inoculated with the washing from the crushings of a tooth, which was centrifuged sufficiently to throw down all sediment. A few organisms that were washed from the teeth were left in suspension, but only the clear supernatant fluid was used. B is the control. It gained in weight continually, whereas A began to lose weight slowly. The amount of wash injected was 1 cc. of apparently clear water. In a few days A showed a loss in weight though there was no apparent loss of appetite. Both ate heartily and exercised freely in a roomy

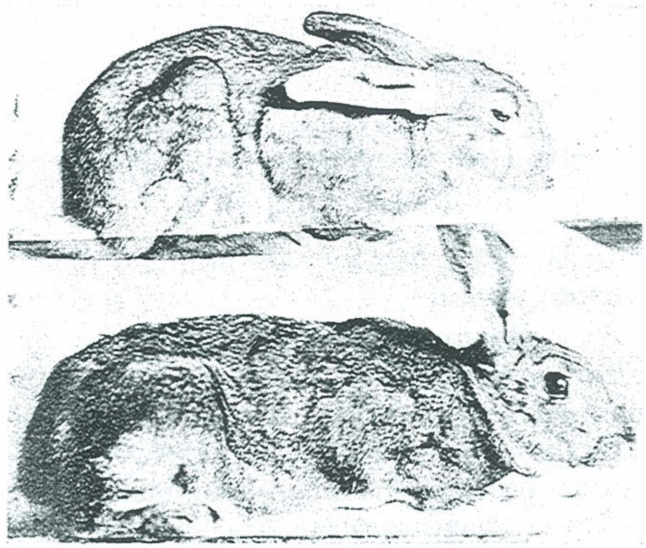


FIGURE 123. TWO BROTHER RABBITS OF CORRESPONDING SIZE AND WEIGHT. ALWAYS UNDER SAME ENVIRONMENT. A, THE UPPER, WAS INJECTED INTRAVENOUSLY WITH 1 CC. OF THE CLEAR CENTRIFUGED WASHINGS OF AN INFECTED TOOTH; B, A CONTROL. A LOST 37% AND DIED IN FIVE WEEKS AND B GAINED 12%.

cage. Week after week A was losing in weight and B was gaining in weight. At the end of four weeks, A had lost about 25 per cent in weight; B had gained about 10 per cent in weight. At the end of five weeks, A died, having lost about 37 per cent, in which time B had gained about 12 per cent. On post, A showed a general marasmus with marked atrophy of all muscle tissue, and development of changes in the digestive tract.

In Chapter 2, I have discussed the morphology and biological characteristics of the organisms involved in dental infections. In general, we have found that their most important characteristic is their power of adaptability. In Figure 24 of that chapter it was revealed in the spontaneous deaths the organisms, that were washed free from the culture medium and injected into animals, killed in an average of seven days, with a loss of weight of 2 per cent per day; whereas, when the whole culture was injected, they died on an average of six days, with an average loss of 3 per cent per day. This suggested that there was some toxic factor present in the culture medium that was injurious and was additive to the injury of the organisms.

Our problem in this chapter is to determine somewhat of the general nature of these toxic products. To ascertain this, seventeen rabbits were injected with the filtered washings from infected teeth, and it should be noted that this was not the washing of a quantity of bacterial growth but simply the accumulated toxic substance present in a single tooth in each case. Of these seventeen rabbits, thirteen died spontaneously in an average of five days, with a loss in weight per day of 3.8 per cent. Inasmuch as the organisms which produced the toxin in these particular teeth were not the same strains of organisms represented in the chart in Figure 24 of Chapter 2, we cannot, by this comparison, compare the organisms with their identical toxins. However, since the organisms in all these cases are diplococci and streptococci, they are, in general, comparable.

Another most striking result of this experiment was that of fourteen rabbits injected with the unfiltered washings, in which the toxic substance plus what bacteria would wash from the crushed tooth would be involved, eight died in an average of twelve days, with a loss in weight per day of 1.8 grams. We are at a loss to understand why, in these cases, the toxic substance alone washed from the tooth is more rapidly fatal than the toxic substance plus the organism, unless it be that it is the presence in the blood stream or body fluids of the dead or living organisms which furnish the necessary activity to call forth the antitoxins to combat this toxic factor. I am inclined to believe that this is the explanation. In this chart in Figure 124, it is interesting to note that only four of seventeen rabbits lived more than a few days, thirteen dying in an average of five days. One of the rabbits of

COMPARISON OF FILTERED AND UNFILTERED WASHINGS

No. in Group	Death	Days Lived	Loss			Gain		
			Actual	%	% per day	Actual	%	% per day
Filtered Washings								
3	Chloroformed	33	56	5	.1			
1	Chloroformed	44				176	18	.4
13	Spontaneous	5	191	19	3.8			
Unfiltered Washings								
5	Chloroformed	32				171	14	.4
1	Chloroformed	20	191	10	.5			
8	Spontaneous	12	221	22	1.8			

FIGURE 124.

this group received a washing which, apparently, was not at all toxic, or but slightly, for it gained 18 per cent in forty-four days; and, similarly, five of the group of fourteen that received the unfiltered washings, gained an average of 14 per cent; whereas, all the others in each group lost in weight. In the chapter on Sensitization, I have discussed the matter of the ability of these toxic substances to sensitize tissues of the host so that when later injected with the toxic substance or when the organisms producing it are injected into the host, the reactions are much more violent than otherwise. One rabbit died within a few minutes.

These data suggested to us that we are dealing with products in freshly extracted, infected teeth, which differ essentially from those developed by culturing the organisms from the same. We, accordingly, made determinations of the difference in the reactions when the organisms were removed from the washings, and found that in many instances that after passing the washings of the crushed tooth through a fine Berkefeld filter very similar disturbances were produced in animals even though the cultured filtrate developed no organisms. It was found that, whereas the organisms, when injected, tended definitely and regularly to produce lesions in various organs and tissues, the filtrate produced disturbances of metabolism and nutrition, with its principal effects upon the digestive tract and nervous system. It is evident that we are dealing in infected teeth with a substance which has very profound effect in very small quantities. The substance is a very complicated one which does not lend itself readily to concentration, as will be shown later.

We have next inoculated succeeding groups of animals with the filtered washings from teeth, and then tested these animals to determine whether they were more sensitive to the organisms grown from those teeth than our normal animals; and it has been found that whereas the controls—namely, the animals that had not received a preliminary injection of the filtrate—developed lesions in one or two weeks and usually recovered according to the sizes of the doses used in the tests, the animals that were inoculated with the filtrate usually developed their symptoms much more rapidly and severely. A much larger percentage died and often within a day or two; and in a few instances, the preliminary injection of the filtrate so prepared the animal for the bacterial injection that it died within thirty minutes to two hours, and in one instance in a few minutes; whereas the controls lived for weeks and often apparently recovered entirely. We believe this is one of the most important of the many new discoveries of our work. Its interpretation and significance are given in Chapters 45 to 56.

SUMMARY AND CONCLUSIONS.

We are, therefore, led from these studies to conclude:

That the organisms found in infected teeth are not the only product of that tooth which may disturb the host, that toxic substances are formed in the tooth which may pass from that tooth to the host, and in some instances (perhaps in many) tend to prepare the host for the invasion by that organism, and may, either in addition or separately from that process, produce definite disturbances of metabolism within the host.

We would, accordingly, change the old fundamental: namely, that *the only injurious substance which infected teeth may contain is a bacterium;* or *infected teeth contain no other injurious substances than microörganisms,* to:

Infected teeth may contain in addition to microörganisms toxic substances, which produce very profound effects upon experimental animals and, which tend to prepare the tissues of the host, at least in some cases, for a more ready invasion by the organisms growing in that tooth.

CHAPTER XIX.

HEMATOLOGICAL CHANGES IN THE BLOOD.

PROBLEM: What changes are produced in the blood and sera of the body by dental infections?

EXPERIMENTAL AND DISCUSSION.

While it has been known for a long time that many patients suffering from rheumatic group lesions, which have affected various of the harder tissues, such as the muscles and skeleton, nervous system, circulation, digestive tract, etc., there seems to have been very little work done to determine the effect of dental infections upon the various sera of the body. An analysis of over one thousand blood counts of animals discloses, that, whereas a given strain of organisms tends to produce, in general, the same changes in the blood picture, different strains may produce widely different results.

Figure 125 shows a group of rabbits which received their infection by having teeth planted beneath the skin. These were such teeth as were condemned because of suspected systemic involvements of the patients. We have selected for this group a series having from two to three counts and in which there has been an increase in the erythrocytes. It will be noted that in two instances, five are from the same patient.

Similarly, some cultures and implanted teeth tend to produce a decrease in the erythrocytes and, occasionally, very marked decrease. In Figure 126 we have a group of rabbits showing decreases in both the hemoglobin and erythrocytes, which are typical. The first decreased from 6,750,000 to 4,600,000 in twenty-four hours. The hemoglobin decreased 5 per cent, and incidentally, there was a decrease in the leucocytes. These, however, will be discussed in subsequent paragraphs. This rabbit was inoculated with culture. The second had a tooth implanted beneath the skin. The hemoglobin reduced 5 per cent, and the erythrocytes from 6,900,000 to 4,200,000 in eight days. The third shows a quite remarkable picture. This rabbit had a tooth planted beneath the skin; and while its hemoglobin remained constant, the erythrocytes reduced from 4,050,000 to 1,650,000,

ERYTHROCYTOSIS PRODUCED BY TOOTH IMPLANTATIONS

Case No.	Rabbit No.	Date	Erythrocytes
1236	813	2–15–22 2–23–22	3,900,000 4,600,000
1215	820	2–15–22 2–23–22	2,600,000 5,650,000
1215	821	2–15–22 2–19–22	2,056,000 3,050,000
527	822	2–16–22 2–23–22 3–29–22 3–30–22	1,050,000 2,750,000 5,200,000 8,500,000
	824	2–16–22 2–23–22	1,080,000 3,950,000
	825	2–17–22 2–23–22 2–28–22	5,700,000 7,700,000 6,750,000
	826	2–17–22 2–25–22 2–28–22	4,800,000 5,050,000 9,750,000
	851	3– 8–22 3–20–22 4–15–22	1,450,000 2,450,000 6,200,000
334	871	3–17–22 3–20–22 3–24–22 3–28–22 4– 4–22 4–15–22	3,050,000 1,300,000 5,900,000 6,300,000 7,050,000 6,900,000

Case No.	Rabbit No.	Date	Erythrocytes
1396	879	3–20–22 3–27–22	2,600,000 5,000,000
1222	828	2–18–22 2–20–22	3,500,000 4,500,000
1241	882	3–22–22 3–27–22	5,700,000 6,700,000
1241	894	3–22–22 4– 4–22	4,300,000 5,300,000
682	880	3–22–22 3–24–22 4–11–22	1,200,000 5,500,000 6,450,000
	838	2–22–22 3– 2–22	2,750,000 3,750,000
	839	2–22–22 3– 2–22	1,750,000 3,750,000
	840	2–22–22 3– 2–22	2,750,000 4,750,000
	843	2–22–22 2–25–22	2,400,000 3,400,000
1170	827	2–18–22 2–20–22 2–22–22	1,050,000 2,050,000 6,550,000
1205	831	2–20–22 2–22–22	3,080,000 5,050,000

FIGURE 125.

ERYTHROPENIA

Case No.	Rabbit No.	Hemo-globin	Erythrocytes
1322	960	80 75	6,750,000 4,600,000
1119	1057	85 85 80	6,900,000 5,800,000 4,200,000
692	849	80 80 80	4,050,000 5,050,000 1,650,000
692	868	80 75 75	3,600,000 2,900,000 750,000

FIGURE 126.

and the color index raised from 0.9 to 2 in twelve days. The fourth rabbit had a decrease in the hemoglobin from 80 to 75; the color index raised from 1 to 4; and the erythrocytes reduced from 3,600,000 to 750,000 in six days.

The most striking changes in blood morphology, however, have been produced in the various types of leucocytes, expressing themselves generally at first by leucocytosis, followed by a leucopenia. A group of these typical changes in leucocyte count is shown in

LEUCOCYTOSIS PRODUCED BY TOOTH IMPLANTATIONS

Case No	Rabbit No	Leucocytes
1367	1135	9,000 13,000
	1134	8,300 11,400
1370	1154	8,000 11,000 6,100
1390	1158	8,400 17,600
1353	1169	8,800 10,000 8,200
1363	1170	8,200 10,200 9,800 9,300 5,600
1317	1186	5,600 6,900 4,000 2,600
1388	1204	7,900 20,000
1394	1211	8,600 18,000
404	1153	8,200 6,200 10,800
355	1173	10,800 10,200 12,200 23,800
1385	1175	15,300 14,400 25,400

FIGURE 127.

Figure 127. The most striking effect, however, of the effect of the infection on the leucocyte count is expressed as a marked leucopenia, the reduction of leucocytes frequently being very great. This is shown in Figure 128.

For years I have been noticing a type of blood picture in patients suffering from dental infections, which is characterized by a low polymorphonuclear count and a high small lymphocyte count; and these studies seem to have thrown a new light upon this condition. I had noticed that it often changed, returning to or toward normal in the patients after removal of dental infections. In Figure 129 I show the successive counts of a series of eleven rabbits, each one of which had a tooth implanted beneath the skin. The first reading in each case was made before the

LEUCOPENIA PRODUCED BY TOOTH IMPLANTATIONS

Case No.	Rabbit No.	Leucocytes
1317	1167	11,500 7,200
	1186	5,600 6,900 4,000 2,600
1387	1178	10,800 9,600 5,000 6,000
1377	1208	25,000 7,000

FIGURE 128.

implantation and represents approximately the rabbit's normal. In this series, we have shown in heavier faced type, the polymorphonuclears and small lymphocytes, and it will be noted that in every instance in this group there was progressive depression of the polymorphonuclears, with a corresponding increase in the small lymphocytes. The average percentage decrease of polymorphonuclears is 33 and the average percentage increase of small lymphocytes 58. It is interesting to note that while the very serious changes in the different types of leucocytic cells have occured, there has been very little change in the hemoglobin of these animals, as shown in that column. It is also important to note the slight change that has been produced in the erythrocytes in these cases. In the third and fourth column we have shown the weight changes, total and per cent, and it is important that in those cases with very marked loss in weight (for all of these rabbits lost in weight,) almost in proportion with the loss there has been the depression of the polymorphonuclears and an increase in the lymphocytes. We have here an expression of the phases of blood morphology that are involved in the Walker Index, which we have discussed chiefly in the chapters on systemic involvements in patients, in which cases we have referred occasionally to the negative Walker Index.

An illustration would be shown in Case No. 1228, in which the patient was suffering from acute heart involvement and acute rheumatism with albuminuria, with evidence of direct relationship to focal dental infections. The polymorphonuclear count

EFFECT OF TOOTH IMPLANTATIONS IN DEPRESSING POLYMORPHONUCLEARS AND INCREASING LYMPHOCYTES

Case No.	Rabbit No.	Weight Loss Actual	Weight Loss %		Hemoglobin	Erythrocytes	Leucocytes	Polymorphonuclears %	Lymphocytes Small %	Lymphocytes Large %	Eosinophiles %	Basophiles %	Mononuclears %	Color Index
1317	1055	163	12	B	85	5,500,000	7,000	55	30	10	1		4	.7
				A	85	6,700,000	8,400	50	37	7		2	4	.6
				A	80	7,950,000	18,000	38	49	9		1	3	.5
				A	85	6,050,000	7,000	44	43	8		2		.7
1119	1057	100	8	B	85	6,900,000	12,200	39	44	9		3	5	.6
				A	85	5,800,000	12,800	32	52	8		4	4	.7
1353	1097	211	19	B				60	23	11		2	4	
				A	80	5,150,000	22,200	57	27	8	1	2	5	.7
				A	80	4,600,000	8,600	39	51	8		1	1	.8
1363	1123	114	10	B	85	5,200,000	12,600	42	43	8		1	6	.8
				A	80	5,600,000	6,800	40	37	19		1	3	.7
				A	80	4,300,000	7,200	26	58	14			2	.9
1363	1125	263	17	B	85	5,800,000	10,200	63	22	10		1	4	.7
				A	80	6,400,000	5,800	59	27	9		2	3	.6
				A	80	7,200,000	5,800	43	43	9			5	.5
404	1126	519	30	B	85	7,400,000	15,600	60	29	8		2	1	.6
				A	80	7,300,000	9,200	36	51	9			4	.5
				A	80	6,900,000	5,600	38	51	9		1	1	.6
				A	85	6,800,000	14,300	34	52	14				.6
1353	1128	440	28	B	85	7,350,000	10,200	44	42	12		1	2	.6
				A	85	7,100,000	9,600	46	34	10		6	4	.6
				A	85	6,250,000	15,600	31	49	14		1	5	.7
1346	1130	440	37	B	85	4,350,000	12,900	60	31	5		2	2	.9
				A	80	5,050,000	7,800	29	58	11			2	.7
				A	80	5,250,000	14,800	39	53	8				.7
1363	1131	354	36	B	85	6,450,000	8,000	45	44	9			2	.6
				A	80	6,000,000	6,800	43	47	10				.6
				A	80	6,800,000	5,500	26	61	13				.6

B. Before implantation
A. After implantation

Average percentage decrease of polymorphonuclears..........33
Average percentage increase of small lymphocytes........... 58

FIGURE 129.

was 83 per cent, which would call for a leucocyte count of at least 23,000 and she only had 10,500. She, accordingly, had a Walker Index of — 12. A blood culture was, accordingly, made, and the same strain of streptococci was found in the patient's blood as was found in the extracted tooth. With the removal of her dental infections, her condition improved very greatly. Her Walker Index reduced to — 4; and, when taken later when the patient was normal, her Walker Index had returned to approximately normal.

PATIENTS WITH DEPRESSED POLYMORPHONUCLEARS AND INCREASED SMALL LYMPHOCYTES

Case No.	Hemoglobin	Erythrocytes	Leucocytes	Polymorphonuclears	Lymphocytes		Mononuclears	Eosinophiles
					Small	Large		
1381	90%	4,600,000	7,200	50.0%	35.5%	7.0%	3.5%	1.5%
1267	85%	5,600,000	7,800	48.5%	42.0%	6.0%	2.0%	0.5%
1311	80%	5,800,000	5,200	56.3%	34.0%	6.2%	2.6%	0.9%
1405	85%	4,700,000	7,800	58.3%	36.3%	3.6%	0.8%	0.5%
1403	80%	5,750,000	6,800	50.0%	42.0%	4.5%	2.5%	
1404	85%	5,300,000	5,600	56.8%	34.1%	5.4%	2.7%	1.0%
955	85%	5,100,000	9,240	50.0%	40.0%	8.5%	1.0%	0.5%

FIGURE 130.

In Figure 130 we show a group of consecutive cases, being patients suffering from dental infections of long standing and all showing systemic involvements which I have interpreted to be influenced directly by the dental infections, if not largely produced by them. When we compare these with a case (Case No. 1385) of acute rheumatism which developed suddenly following a chilling of the patient, whose defense was lowered and who at the same time was carrying such chronically infected teeth, we find his blood count as follows:

Hemoglobin	90%
Erythrocytes	5,500,000
Leucocytes	16,000
Polymorphonuclears	78.6%
Small lymphocytes	13.8%
Large lymphocytes	4.1%
Mononuclears	3.0%
Eosinophiles	0.5%
Color Index	0.8

Note that his polymorphonuclears are 78.6; his small lymphocytes, 13.8. It must not be understood that I am presuming that all cases of low polymorphonuclear count have dental infections, nor that all patients with a high lymphocyte count, or both, have thereby an evidence of dental infection. I am submitting for your consideration the fact first that it occurs in the patients with chronic dental infections, and that it develops in our experimental animals in which we produce such states.

SUMMARY AND CONCLUSIONS.

While the data available are not sufficient for detailed deductions, they strongly indicate that there is much significance from both the pathological and diagnostic viewpoints in the blood morphology and its changes. The evidence strongly suggests that the toxic elements involved in the infection process have distinct and harmful effects; and while the reaction to infection of a normal defense is characterized by a leucocytosis (a fact which is quite universally recognized), it seems quite as universally true that certain types of infections, such as those produced by the planting of a tooth beneath the skin of a rabbit, have destructive effects, particularly on the polymorphonuclears, the depression of which decrease in the presence of the increasing infection, spells a very bad omen in the case of our animals, and practically always terminates in death. It, therefore, seems probable that our patients, who show a very marked leucopenia, and particularly with a markedly depressed polymorphonuclear count, are undergoing a degenerative process. Our application of this is discussed in Chapters 45 to 56.

We would, accordingly, condense the conclusions of this chapter to the following:

Dental infections may produce very serious changes in the blood and sera of the body, some of the most frequent of which are leucopenia, erythropenia, lymphocytosis, and hemophilia.

CHAPTER XX.

CHEMICAL CHANGES OF THE BLOOD.

PROBLEM: What are the chemical changes that are produced in the blood by acute and chronic dental focal infections?

EXPERIMENTAL AND DISCUSSION.

The preceding researches led us step by step through clinical expressions of dental infections, local and systemic, through a channel that has led us back into a mountain vastness, uninhabited and unexplored, and quite uncharted. I was led to make this mode of attack as a result of the study of the apparent causes of success and failure in the various lines of research that have been conducted, for apparently exceedingly few solutions to problems have been found by a direct attack on fundamental problems. The symptoms of dental infections are their clinical expressions. By association and exclusion we have been able to type the local structural expressions and also the physical manifestations. These have led directly to changes which occur in the hard structures of the body as increases or decreases in the density of a bone, and in the soft tissues as edema, atrophy, and disfunction. If rarefying osteitis is associated with an entirely different type of defense and reaction from that which obtains when condensing osteitis develops, it would seem most logical that those elements which are directly related to bone formation and tissue function must be most vitally involved. Accordingly, from nearly every research that has been herewith recorded, we have had evidence pointing directly to those factors which are most intimately related to metabolism and bone formation.

As a first approach we have made blood analyses of many hundreds of patients and animals to determine, if possible, what factors are most variable in the different clinical expressions and to what extent. In Figure 131, I show 146 successive blood chemical analyses for 92 successive patients, some individuals having had several determinations made. In these studies we have determined, in general, blood sugar, (Usually the first

COMPARISON OF BLOOD CHEMISTRY, DENTAL PATHOLOGY, AND SYSTEMIC INVOLVEMENTS

Case No.	Sugar	Non-protein Nitrogen	Uric Acid	Urea	Biological Determination				Chemical Determination		Dental Symptoms	Systemic Symptoms
					Calcium Ionic	Calcium Ionic plus Combined	Calcium in Combination	Thrombin	Calcium	Alkalinity Index		
1334	102	28.8				13.20		11.52	12.03		Locked. Rarefying.	Sensitization.
1337	118		4.41 3.12		9.40	11.12 11.84	2.44	14.44 21.60		39.40	Locked. Rarefying.	Neuritis.
1339	107	38.2			7.61 8.07	8.30 8.72	0.69 0.65	10.52 14.13		39.5	Locked. Rarefying.	Neuritis.
1338	109		2.0		12.28	12.52	0.24	7.73			Rarefying. Periodontoclasia.	Ear involvement.
1343					10.86	11.95	1.49	7.14			Locked apical.	Heart.
1342					9.77	11.95	2.18	5.43			Periodontoclasia. Extensive rarefying.	Normal.
812					9.84 10.76	10.76		12.16 9.24			Chronic periodontoclasia.	Lassitude.
410	109				12.47 11.95	12.47 12.03	0.08	11.53 20.05		34.4	Periodontoclasia.	None.
1348	102			7.50	6.84 9.62	7.87 11.42	1.03 1.80	12.16 19.38			Locked apical. Chronic periodontoclasia.	Sensitization.
1346	108 83 93 130	49.0 36.0 37.4 27.3	3.95 3.18 3.34 3.87	12.54 12.07 13.22	9.62 6.09 10.31 9.38 10.34 10.067	11.31 7.66 11.73 12.65 12.16 11.60 10.600	1.69 0.57 2.34 2.78 1.26 0.533	20.38 23.91 10.77 7.69 21.62 16.66 5.333		47.2 39.2	Locked apical. Chronic periodontoclasia.	Neuritis. Heart involvement.
1347	103				11.21	11.52	0.31	22.79			Periodontoclasia.	Sensitization.
735	101				7.67	11.21	3.54	18.33			Locked.	Pregnancy.
1317	98	33.0	2.4		12.87	13.44	0.57	14.13			Locked. Chronic per-iodontoclasia	Lassitude. Mild ne-phritis

FIGURE 131 (A)

1365	103	39.0	2.08		11.536	12.34	0.804	7.624			Locked apical.	Neuritis of spine.
1367	119 103 93 90	26.0 46.0 24.0			11.804 11.536 11.402 11.67	14.22 13.66 13.222 13.124	2.416 2.124 1.82 1.454	6.996 6.264 13.598 11.33			Chronic periodontoclasia. Locked apical.	Brain disfunction spasmodic.
1371	105	26.5			11.67	12.33	0.74	5.67			Chronic periodontoclasia.	Skin lesions.
1307	131	26.4			10.667	12.732	2.055	6.133			Chronic periodontoclasia. Locked apical.	Eye involvements Lassitude.
1372	92				10.033	10.804	0.77	7.34			Locked apical.	Neuritis.
1370	102	26.0			10.867	12.47	1.603	10.133			Chronic periodontoclasia. Locked apical.	Lassitude.
1200	106 73	27.0 32.0			9.84	10.333	0.493	7.16			Chronic periodontoclasia. Apical.	Lassitude. Digestive disturbance.
1373	108	28.2	1.36		11.134	12.464	1.330	7.667			Locked apical.	Heart.
1016	62 77	25.6 27.0	1.16		11.670	11.670		10.33			Normal.	Nearly normal.
1374	122	26.2	2.12		8.897	9.680	0.783	8.733			Locked apical.	Underweight. Nerves.
1040	111				12.34	12.34		10.46			Periodontoclasia.	Normal.
1376	94	47.4			11.534	12.866	1.222	3.667			Apical. Periodontoclasia.	Skin sensitization.
1382	82 121 93	25.6 23.6 30.0			10.576 10.801 10.134	12.753 11.402 12.340	2.239 0.601 2.206	12.424 6.399 7.667			Locked apical. Mild periodontoclasia.	Xerostomia.
1381	128 92	33.0 35.0			8.256 10.801	10.55 12.608	2.294 1.807	7.144 12.392			Locked apical.	Lassitude. Rheumatism. Physical symptoms improved.
1383	98.6	28.8			10.267	11.402	1.135	8.733			Locked apical.	Rheumatism.
1385	99	27.6	3.3		9.840	10.801	0.961	13.16			Locked apical.	Acute rheumatism.

FIGURE 131 CONTINUED (B)

COMPARISON OF BLOOD CHEMISTRY, DENTAL PATHOLOGY, AND SYSTEMIC INVOLVEMENTS

Case No.	Sugar	Non-protein Nitrogen	Uric Acid	Urea	Biological Determination: Calcium Ionic	Calcium Ionic plus Combined	Calcium in Combination	Thrombin	Chemical Determination: Calcium	Alkalinity Index	Dental Symptoms	Systemic Symptoms
1410	119.3	32.2	3.1		9.44	9.84	0.4	9.56			Normal.	Acute cold.
1262	75				10.23 10.81	11.42 12.03	1.19 1.22	20.77 19.19			Locked apical.	Duodenal ulcer.
1349					9.54	12.05	2.51	8.56			Acute apical abscess.	Fever. Lassitude.
1409	98 93	22.0			12.26 10.04	12.26 11.33	1.29	10.74 3.51		31.8	Locked apical.	Lassitude.
355			2.56		12.27	12.79	0.42	16.73			Normal.	Chronic deforming arthritis, not acute.
1350					9.06	9.66	0.6	10.34			Normal.	Normal.
1353	98 83 88 96	25.0 25.0 24.0	1.44 2.26 2.06 2.72		11.71 11.92 9.25 9.45 10.267	12.82 11.92 13.23 11.61 11.536	1.11 3.70 2.16 1.269	11.29 16.08 15.73 20.00 8.333	10.504		Extensive chronic periodontoclasia.	Nervous breakdown.
987	97	38.0 35.6			9.32 9.61 9.004	10.07 10.53 9.40	0.75 0.92 0.396	17.68 20.39 13.996			Normal.	Multiple recurring rheumatic group lesions.
1120	113 80	29.3 45.0	1.7	13.0	10.67	11.53	0.86	7.33			Normal.	Normal.
1354	96	18.0	1.13		10.58	11.40	0.82	9.42	11.64		Locked apical.	Acute neuritis.
1355	112	28.5			10.77	11.87	1.10	12.23			Apical.	Disturbed vision.
1332	79	27.0			10.39	12.8	2.41	19.11		46.0	Normal.	Normal.
1407	110	28.0			11.13	12.61	1.48	6.87			Mild periodontoclasia.	Normal.

FIGURE 131 CONTINUED—C.

1359	98	36.0	2.56		11.06	13.22	2.16	7.54	10.50	41.0	Locked apical.	Hypertension. Nephritis.
1408	115	27.0			11.26	12.48	1.22	13.74	11.733		Locked apical. Periodontoclasia.	Lassitude.
1228	113 102	29.8 34.0	1.55	20.5	11.134	11.938	0.80	15.86	8.29		Locked apical. Normal.	Very acute rheumatism and heart involvement. Normal.
1363	127 116 112	55.0 45.0	3.16		12.598 11.904 12.474	15.04 15.40 13.66	2.442 3.496 1.186	7.96 12.016	9.873	36.14	Apical. Chronic periodontoclasia.	Mild nephritis.
955	113				11.134	13.00	1.866	6.467			Excessive caries.	Xerostomia.
1404	105.6	33.4	3.55		11.802	13.334	1.532	4.998			Chronic periodontoclasia.	Neuritis.
1403	76	20.0	2.98		10.734	11.134	0.40	8.266			Locked apical.	Pelvic surgical.
1311					6.67	9.81	2.14				Nearly normal.	Acute and chronic deforming arthritis.
1325	106 116	35.3			10.50 10.68 9.86	11.25 10.93 10.58	1.20 0.25 0.72				Chronic periodontoclasia.	Lassitude.
1317	96	28.0			7.88 11.13 8.72 8.68	9.43 11.15 9.83 10.18	1.55 0.02 1.11 1.50		10.13		Chronic apical.	Lethargic encephalitis.
1267					7.05	9.47	2.24				Locked apical.	Nervous breakdown.
701					9.36	11.36	2.00				Normal.	Mild rheumatism.
1269					9.66 10.36	11.71 13.65	1.05 3.29				Locked apical.	Syringomyelitis.
1319					8.62	10.05	1.43				Locked apical.	Rheumatism. Heart involvement.

FIGURE 131 CONTINUED—D.

COMPARISON OF BLOOD CHEMISTRY, DENTAL PATHOLOGY, AND SYSTEMIC INVOLVEMENTS

Case No.	Sugar	Non-protein Nitrogen	Uric Acid	Urea	Biological Determination: Calcium Ionic	Biological Determination: Calcium Ionic plus Combined	Biological Determination: Calcium in Combination	Biological Determination: Thrombin	Chemical Determination: Calcium	Chemical Determination: Alkalinity Index	Dental Symptoms	Systemic Symptoms
1321					9.75	11.02	1.27				Locked apical.	Neuritis. Nervous breakdown.
817					9.89	10.13	0.24				Normal.	Normal.
1326					8.42						Locked apical.	Rheumatism.
1312					9.53	10.52	0.99				Extensive apical. Rarefying osteitis.	Lassitude.
1268	295 410	47.0 31.0	5.4	15.2 16.6	9.33	11.67	2.24		12.32 12.18		Chronic periodontoclasia.	Diabetes.
1315					5.25 6.37 8.66	7.50 9.02 9.93 10.26	2.25 2.67 1.60				No involvements.	Neuritis of right side.
381					7.99 8.24 6.68	10.75 10.15	2.76 1.91				Locked apical.	Acute rheumatism and arthritis.
1322					8.17 8.39	9.21 9.65	1.04 1.26				Locked apical.	Rheumatism. Heart involvement.
1285	106	42.0	7.54	19.50					8.78		Locked apical.	Neuritis.
1272	116	26.0	4.6	12.0					11.4		Chronic periodontoclasia.	Normal.
1288	102	30.0	5.4						12.1		Chronic periodontoclasia.	Nervous irritability.
1295	96	28.0	3.6						11.54		Dental cyst.	Insomnia.
1406	100	36.9	3.63		11.67	12.205	0.535	13.33			Extensive locked apical.	Neuritis, recurring
1416	115	31.0	1.65		11.134	11.402	0.268	15.866			[illegible]	[illegible]

FIGURE 131 CONTINUED (E)

1413	161 125	58 40	3.14 2.35		10.40 10.935	13.222 12.608	2.822 1.673	14.60 16.065			Locked apical.	Rheumatism. Nephritis.
797	119	49	2.86		9.640	11.802	2.162	3.582			Chronic periodontoclasia.	Lassitude.
335	87	32	2.34		8.721	10.067	1.346	6.879		32.53	Normal.	Nervoussymptoms. Lassitude.
1414	100	29	2.17		9.600	9.960	0.360	4.067			Locked apical.	Heart. Lassitude.
1415	134	37.5	2.73								Periodontoclasia.	Normal.
1416	115	31.0	1.65		11.134	11.402	0.268	15.866		38.65	Locked and open apical.	Proliferative Arthritis.
1421	107	33.5	2.80		9.80	10.935	1.135	7.20		39.39	Periodontoclasia.	Lassitude.
1419	107	46.8			8.384	12.205	3.811	9.216		25.49	Locked apical.	Lassitude. Tension in head.
1425	147 115	27.0 30.6	2.08		8.87 9.152	9.44 10.678	0.57 1.516	8.73 9.848		26.32 27.16	Periodontoclasia.	Nervous tension. Albuminuria.
1424	127	22.6			8.285	9.585	1.300	16.715		37.84	Locked apical.	Nervousness. Acute digestive.
1423	154	28.5			10.315	12.727	2.412	10.685		35.56	Locked apical. Caries.	Goiter. Heart. Nervousness.
1311					11.670	13.22	1.550	7.76		29.75	Normal.	Deforming arthritis.
1324	102	23.0			8.428	10.672	2.244			20.36	Periodontoclasia.	Osteomalacia.
1381	76 95	29.0	2.29		10.600 8.790	12.360 10.263	1.760 1.473	16.400 7.210		23.00	Normal.	Rheumatism. Ulcers.
987	71	35.60	1.52		9.004	9.400	0.396	13.996		32.50	Normal.	Rheumatism. Heart. Eyes.
1417	127	33.7			9.045 9.84 10.935 10.142 10.314	10.270 11.171 12.340 11.564 11.278	1.235 1.336 1.405 1.422 0.964	15.995 6.429 12.340 8.058 9.686		34.7 30.15 34.68 30.16 30.50	Periodontoclasia. Acute apical abscess.	Maxillary osteomyelitis. Acidosis.
1424	125	29.1			9.173	10.986	1.813	17.827		29.90	Locked apical.	Acute digestive. Nervousness.

FIGURE 131 CONTINUED (F)

reading was not made after fasting as is required for a determination of hyperglycemia. When, however, such seemed indicated this was done.) non-protein nitrogen, uric acid, urea, acid-base balance, alkali reserve or a CO_2 combining power, also expressed as alkalinity index, ionic calcium, calcium pathologically combined, thrombin content, total calcium (including the ionic, pathologically combined, and the physiologically combined, such as calcium proteinates, etc.) It will be noted from reviewing this table that there is a very great variation in these different chemical constituents of the blood in the different individuals, and this very extensive study has involved the relating of these to the various pathological states, and particularly to the dental conditions and the changes in these and the physical symptoms or involvements, with the changes in the dental focal infection elements.

When, now, we relate the general clinical symptoms and physical conditions of these patients with these data, there are certain factors that we find quite universally associated. It will be noted, for example, that with few exceptions the high readings of blood sugar are found associated with high ionic calciums of the blood. While there are very few exceptions, this becomes an association which has a quite constant significance, as we will see. Another striking association is the practically universally high ionic calcium in cases of acute periodontoclasia. Similarly, certain types of susceptibility and involvement with rheumatism and neuritis, tend to be associated with a depressed ionic calcium of blood. We have, accordingly, divided these patients out into groups and studied them intensively in connection with these various factors, and have made a very large number of animal inoculations and subdermal tooth implantations, and checked these chemical changes of the blood against these established conditions. These will be reviewed in detail.

But these studies have tended to be quite indefinite with regard to the calcium, for some groups of individuals with approximately normal total calcium have included many cases with definite rheumatic group lesions, for while there was enough total calcium present it did not seem to be available for normal metabolic and catabolic processes. Another great difficulty has been the variation that has developed in the calcium determinations as made by different methods. It has been known for some time that calcium is present in the blood in two principal forms: ionic and combined, the latter including the various calcium proteinates. We have, accordingly, spent a great deal of effort to determine the amount of the ionic calcium as well as the total calcium and, where possible, the pathologically combined calcium for the various types of lesions. We have found that in a large percentage of cases of certain types of rheumatic group affections there is at the time of the active process a low ionic calcium; and, furthermore that as the ionic factor approaches normal, the symptoms disappear. In the coagulation of normal blood, calcium is a fundamental factor; but instead of ionic calcium's being taken up as might be anticipated in that process, it is, as a matter of fact, liberated, for the ionic calcium of the plasma of uncoagulated blood is lower than that of the serum of coagulated blood. (The fluid of the circulating blood is referred to as the plasma and the fluid which separates out from the coagulum of coagulated blood, as the serum.)

Normal blood should contain from 10 to 10.7 milligrams of calcium per 100 cubic centimeters. When the blood is in circulation, approximately four milligrams of the calcium are carried in combination with the thrombin, a little less than a milligram in the blood cells and about six milligrams as ionic calcium. In the clotting process the four milligrams in combination with the fibrinogen will be released in the process of the formation of fibrin and will appear as freshly ionic calcium in the serum. Accordingly, if, in any individual the total calcium be reduced four milligrams—namely, from ten milligrams to six milligrams—the four milligrams which are combined with the fibrinogen will not be available in the circulation as ionic calcium, and the total ionic will be reduced to two milligrams; in other words, in this case there is a reduction of two-thirds from normal, whereas the total calcium has only been reduced a little more than one-third.

From this it will be seen that determinations of the total calcium may be very misleading. No problem with which we have been engaged has compared in difficulty with this one of the making of dependable determinations of the quantity of calcium in different states in the blood. It will readily be seen that any incineration method can only give total quantities, since the ash can give no indication of chemical structure of destroyed compounds. Nor have we, as yet, dependable quantitative *chemical*[10] reactions that will differentiate with certainty the ionic calcium from the combined calcium though, no doubt, experimental methods will be improved very rapidly, to make this more easily possible. We have, however, *biological*[11] methods for making these determinations and we have, accordingly, depended on these.

Similarly, we have determined the amount of calcium pathologically combined in different stages of the treatment of a rheumatic group lesion. Figure 132 shows the progressive stages toward normal, of a case in which the pathologically combined is expressed. It will be noted from this chart that the ionic calcium began at 7.5, with a pathologically combined factor of 2.2. This patient was suffering from a recurring infective process for months after a mandibular extraction had been made under gas. No sequestra ever formed, and there was no direct evidence of an osteomyelitis. The condition came finally to involve the fascia and musculature of the neck and side of the face, with much enlargement of lymphatics, which ultimately required deep surgical drainage. Gauze packs were replaced daily for months, and the cellulitis tended to increase. The method of treatment will be discussed in detail in the chapters on the glands of internal secretion (Chapter No. 37) and methods for reinforcing a deficient defense (Chapter No. 42). The important thing in this connection is to note that with the progressive increase in his ionic calcium, whether as a cause or an effect, there was a marked improvement in his physical condition. From the 31st of August to the 13th of September (two weeks' time) there was an increase in

Important references. See bibliography for others.

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EFFECT OF TREATMENT ON IONIC CALCIUM OF BLOOD

Date	Hour A. M.	Treatment for Ionic Calcium	Ionic Normal Serum	Ionic Treated Serum	Comb. Patho-logical
8-31-22	9:00	Began	7.5	9.7	2.2
9- 6-22	10:00	Continued	7.9	9.9	1.9
9-13-22	11:00	Continued	8.1	9.5	1.4
9-26-22	11:00	Continued	8.2	9.4	1.2
1-24-23	11:00	Discontinued	10.6	11.0	0.3
2-16-23		Resumed	9.2	11.1	1.8
4-25-23		Continued	9.4	9.9	0.5

FIGURE 132.

the ionic calcium of the blood of 0.6 mg. per 100 cc., and a decrease in the pathologically combined of 0.8 mg. This patient had been in a process of decline for a couple of years and had been unable to carry on his work for many months, and with his age of fifty-seven and the seriousness of his disturbance, it seemed very probable that with his progressive and continuous decrease in defense with increase in the severity of his conditions, he was heading for a complete, and perhaps final break. With his low defense and the history of previous surgical procedures, which seemed to give only temporary benefit, hope for material assistance by that means was not indicated.

However, under treatment for the increase of his ionic calcium, his change was most rapid and remarkable. Not only did his state of impending doom give way to one of confidence and courage, but he gained in weight and physical endurance so rapidly that in a few weeks' time he was back to his office, carrying not only one man's work, but two or three, and notwithstanding his tremendous overload, went on gaining progressively as his ionic calcium increased; and in about four months it was up to normal, 10.6, and his pathologically combined had decreased to 0.3. His facial condition and neck involvement entirely disappeared spontaneously and never recurred. At this time the treatment was discontinued and in about three weeks' time his ionic calcium had decreased to 9.2 and his pathologically combined had increased to 1.8. The treatment was resumed and in nine weeks, notwithstanding an excessive overload and worry, his condition again improved, the ionic increasing to 9.4, and the pathologically combined (which is very important) decreased from 1.8 to 0.5.

It is a very common occurrence in connection with the study of these cases to find that accompanying the general physical depression there is a mental depression which I have termed "Mental Cloud", which varies in severity from a sense of impending doom to one of lack of courage. It is quite remarkable that this depressed mental state tends rapidly to disappear with the increase in ionic calcium toward normal. I have discussed this in further detail in the chapter on mental diseases.

Perhaps no phase of local and systemic involvement from dental infection is more frequently manifest than the disturbances of the circulation, both local and general, the most readily discerned of which, both by the patient and the operator, will be disturbances of coagulation; and it will be seen that these studies throw a flood of new light on secondary postoperative hemorrhages, bleeding gums, etc. This may either be a temporary expression or a quite general and extended one. It will also be possible for us to distinguish quite clearly between an hereditary hemophilia and a pathologically produced one. An extreme illustration of the latter will be seen in the following case.

Case No. 1084.—The patient was presented with a history of hemophilia so serious that he felt he was bleeding to death, and on several occasions his life had been despaired of. During the preceding two weeks he had had two transfusions to restore, if possible, the clotting ability of his blood. The hemorrhage was practically continuous from the gums, with occasional epistaxis. A tooth had been extracted three months previously and its socket was still bleeding. There was some hemorrhage from his gums practically every moment night and day. On a careful study of his case I observed that the hemorrhage was greatest around his non-vital teeth, none of which showed either extensive areas of absorption or were the least tender. (See Chapter 60) Inasmuch as I had several times had strains of organisms from dental sources that produced spontaneous hemorrhages in rabbits, I suspected that the teeth were providing some substance which was acting directly upon the blood, whatever other sources there might have been for his disturbance. He was barely able to walk; had to be assisted up all steps; was exceedingly weak and of ashen color. Notwithstanding the great danger attending an extraction, through the difficulty of controlling the hemorrhage, it seemed very desirable that his pulpless teeth be removed, both in order to relieve him from their injurious effect, if such existed, and to secure a culture for making a vaccine to reinforce his systemic defensive reactions. Great difficulty was experienced in controlling the hemorrhage following the extraction, a compress being required night and day for several days; and in spite of this, approximately 100 cubic centimeters of blood were lost in a few hours following the extraction. It is sufficient to state here that, with the use of a vaccine and the elimination of his dental infection his clotting time reduced progressively from eight and ten minutes at the time of the first extraction, to three and three and one-half minutes; and after the first extraction I had little trouble in controlling postoperative hemorrhage. In a week's time practically all spontaneous hemorrhage had ceased; and in four weeks' time he was carrying on his work approximately as normal.

But this is not the only important part of the history. Cultures grown from the interior of these teeth, and injected into a large number of rabbits, produced in many of them serious and early disturbances in the blood stream. Many had spontaneous hemorrhages and very marked change in the clotting time of the blood, even extending to ten minutes from a normal of from one-half minute to one minute and one-half. In the chapter on circulation disturbances there will be seen a series of cases, including this one, of spontaneous hemorrhages produced in rabbits when inoculated with the cultures from the teeth of patients suffering from hemophilia. One of the above rabbits inoculated with the culture from this case and shown in a later chapter referred to, died in twenty hours of spontaneous hemorrhage in the thigh and kidney, so profuse that although the effort was made promptly, we could not get enough blood from the heart and blood vessels to make a chemical analysis, a most unusual experience.

While the injurious effects of infections on the blood are by no means limited to the disturbance of the coagulation mechanism, this change is perhaps the most readily seen of any, and it is also very easily determined with instruments that are adapted for that purpose. It will be instructive, to note the progressive change in this patient. In the first three days after his first extraction, the clotting time decreased two minutes (from eight to six). He then had a slight reaction, and for three days it required seven minutes. In two weeks' time it had decreased two and one-half minutes to five and one-half minutes, and then decreased about a minute a week, reaching four minutes in about four weeks and three and one-half minutes in eight weeks. The spontaneous hemorrhages ceased the third day after the first extraction.

The chemical analysis of this patient's blood showed the CO_2 combining power to be 68, non-protein nitrogen 27, and blood sugar on one occasion 180, on another 158; the creatinin on one occasion 0.9, and another 1; blood urea on one occasion 8.4, on another 9.7; uric acid on one occasion 4.15, another 2.7; total calcium 8.16. At this time (April, 1921) we were not separating the calcium present in the blood in accordance with the amount present in ionic form, the amount pathologically combined and that physiologically combined, and total calcium. This figure would represent simply the total; and inasmuch as the total was reduced below the point of safety for the ionic alone, it is evident that this patient was in a state of very great calcium depression. Our later cases with our greatly improved method (though not as yet completely adequate and satisfactory) have thrown much additional light on these conditions.

A careful study of the data furnished in this case and several showing blood changes, which latter showed very marked improvement after the removal of infected teeth, led to the general conviction that the teeth in certain cases provided a substance which combined directly with the ionic calcium of the blood and removed it from availability, though remaining in the circulation. This is also very strikingly suggested by the very nature of both condensing and rarefying osteitis. In order to test this, we have made a series of studies of the effects of the extracted teeth on drawn blood of the patient from whom the tooth was extracted to determine its effect directly upon the available ionic calcium, and have found most important new data. Figure 133 shows a series of such teeth and the amount of depression of the ionic calcium

BLOOD CALCIUM CHANGES PRODUCED BY INFECTED TEETH

Case No.	Calcium			Tooth Placed in Serum			Decrease in Ionic		Total in Pathological Combination	
	Ionic	Ionic and combined	Patho-logically combined	Ionic	Ionic and combined	Patho-logically combined	Actual	%	Actual	%
1325	9.86	10.58	0.72	8.95	9.38	0.43	−0.91	−10	−1.63	15
817	9.89	10.13	0.24	8.66			−1.23	−13	1.47	15
1363	11.904	15.40	3.496	12.732	13.44	0.708	+0.828	+7	+2.66	17
1353	11.920	11.920		8.51	9.16		−3.41	−29	−3.41	28
1350	9.06	9.66	0.60	8.54	12.58	4.04	−0.52	− 6	−1.12	12
1404	11.802	13.334	1.532	7.258	11.802	4.544	−4.544	−38	−5.076	38
1267	8.10	9.53	1.43	6.05	8.13	2.08	−2.05	−25	−3.51	37
1315	5.25	7.50	2.25	1.25			−4.00	−76	−6.25	83

FIGURE 133.

produced in the blood of the patient from whom the tooth was extracted. It will be seen that some teeth produced practically no effect; most non-vital teeth of long standing, some effect; and some, very profound effect. For example in Case No. 1315, it will be seen that the ionic calcium of the blood was reduced from 5.25 mgs. per 100 cc. to 1.25. This patient was very ill, and the indications were very strong that her dental infections were seriously contributing to her illness. In this group we have several different types, and it is very significant that the teeth of the fourth, sixth, seventh, and eighth cases, which produced depressions in the ionic calcium of the blood of 25 per cent or more, the last 76 per cent, were taken from patients with a very marked evidence of physical injury, one expression of which was the symptoms of systemic involvements from dental infections. In only one case was there an increase in the ionic calcium (Case No. 1363), and in it the total ionic and pathologically combined was abnormally high, 15.4 mgs. per 100 cc., and it seems probable that the presence of the tooth in this patient's blood, carrying 3.49 mgs. of pathologically combined calcium, acted in some way on that element of the blood. In the last column we have added the original pathologically combined and the newly combined from the presence of the tooth, and it will be noted that in the last case of the original 7.5 mgs. of calcium, ionic and pathologically combined, in this patient's circulating blood after the patient's tooth was placed in the blood there was only 1.25 ionic available, or the total combined of this individual's blood after placing the tooth in it was 83 per cent. In the last column we have expressed in percentage that part of the calcium of the blood which should have been available but was in pathological combination: namely, the pathologically combined of the circulating blood plus the pathologically combined produced by placing the tooth in some of the freshly drawn circulating blood. It will be noted that these percentages run from 15 to 83, with four of the eight 28 per cent or over, with an average for the eight cases of 31 per cent.

It is, therefore, not surprising that if an infected tooth within one hour's time will seriously depress the ionic calcium, that the continued presence in the system of such a toxic substance must, of necessity, unless there be some powerful sterilizing agent, exert a very definite and serious influence on the blood. If, instead of a single tooth, the patient has several producing such a toxic substance and is not able to neutralize their products immediately about the tooth, grave systemic results should be expected to occur. It is, accordingly, just what should be expected, that so usually develops that when infected teeth are removed from the systems of patients suffering from depressed ionic calcium, that lesion automatically decreases or disappears. In another chapter on the glands of internal secretion I have discussed some phases of Nature's mechanism for neutralizing these poisons.

By studying the data in the next chart it is very readily seen that there is a constant relationship between the ionic calcium and metabolism, for continually with a decrease in ionic calcium there is a decrease in the weight. I have undertaken, therefore, to study this phase more exactly in order to determine, if possible, somewhat of the significance of the fact that patients suffering from dental infections tend so frequently to be underweight, and after the removal of these dental infections increase rapidly and materially. In the chapter on Marasmus and in the chapters of Part Two, I review cases with increases in weight ranging up to 50 per cent, with many of them increasing 10 to 25 per cent. This raises the question: To what extent is the depression of the weight below normal related to, or an expression of, the ionic calcium of the blood? To determine this and the associated factors we have made a series of implantations under carefully controlled conditions, where the weight has been checked carefully against the ionic calcium. A group of these is shown in Figure 134, entitled "Chemical Changes in the Blood, Produced by Implanting Infected Teeth Subdermally, and the Relation of the Changes of Ionic Calcium and Body Weight." In this chart we show six rabbits. In the first there was a decrease in the ionic calcium of 50 per cent, produced by the placing of the patient's tooth beneath the skin of the rabbit, and all this occurred in four days' time. There was, accordingly, not an opportunity for great wastage of the animal's body, death ensuing before the depletion of the tissues. The total loss in weight in this case was 17 per cent. In the second case the decrease in ionic calcium was 1.18 grams or 12 per cent. But this extended over a period of sixteen days, which gave greater opportunity for depletion of the body tissues, the actual loss of weight being four hundred seventy-one grams, or 34 per cent. In the third rabbit the period taken for the tooth to kill the rabbit was shorter—namely, three days—the ionic calcium loss being three

CHEMICAL CHANGES IN THE BLOOD, PRODUCED BY IMPLANTING INFECTED TEETH SUBDERMALLY, AND THE RELATION OF THE CHANGES OF IONIC CALCIUM AND BODY WEIGHT

Rabbit No.	Date	Weight	Weight Loss		Calcium plus Thrombin	Calcium Ionic	Calcium Ionic plus Combined	Calcium in Combination	Calcium Ionic Loss	
			Actual	%					Actual	%
1106	A. 2–16–23	1141			14.78	10.88	11.78	0.90		
1106	B. 2–20–23				11.27	7.00	7.00	0.0		
1106	C. 2–20–23	942	**199**	**17**	9.80	5.41	6.09	0.68	**5.47**	**50**
1145	A. 3–27–23	1381			14.50	10.13	10.13	0.00		
1145	B. 3–28–23				13.88	8.89	11.67	1.77		
1145	B. 3–30–23				16.80	8.95	11.23	2.28		
1145	C. 4–13–23	910	**471**	**34**					**1.18**	**12**
1099	A. 2–13–23	1822			14.80	11.00				
1099	B. 2–14–23	1483			13.2	9.92	10.36	0.44		
1099	B. 2–14–23	1385	**437**	**24**	18.5	11.22	10.82	0.40		
1099	B. 2–16–23				10.19	7.91	9.00	1.99	**3.09**	**28**
1108	A. 2–20–23	1265			17.00	10.59	12.61	2.02		
1108	B. 2–22–23	1185	**80**	**6**	18.40	8.84	13.00	4.16	**1.75**	**17**
1109	A. 2–20–23	1375			17.20	9.88	10.37	0.49		
1109	B. 2–22–23	1256	**119**	**9**	17.80	7.82	9.80	1.98	**2.06**	**21**
1080	A. 1–27–23	1478			16.50	8.90	8.90	0.00		
1080	B. 1–29–23	1360			10.17	7.12	7.12	0.00		
1080	B. 1–31–23	1321			7.74	7.25	8.69	1.44		
1080	B. 2– 1–23				11.80	7.99	8.56	0.57		
1080	B. 2– 3–23	1210	**268**	**19**	19.00		12.74		**0.91**	**10**

FIGURE 134.

A—Readings before tooth implantations.
B—Readings after tooth implantations and before death.
C—Readings after death.

grams, and the total loss in body weight four hundred thirty-seven grams, or 24 per cent. Similarly, the other three rabbits shown in this chart, reveal calcium losses of 17, 21, and 10 per cent respectively, with body weight losses of 6, 9, and 19 per cent in two days each for the first two and six days for the last. This shows clearly that there is a time factor involved, and seems to emphasize the profound effect on metabolism, in general, of introducing into the animal's body a toxic substance which directly disturbs the ionic calcium. The data available do not justify the conclusion, however, that no other important factors are directly involved, nor that the calcium decrease is the chief or only factor interrupting the metabolic process.

Studies were then made to determine the nature of this calcium compound. If it were an insoluble salt it could be removed by filtering or centrifugation or settling. It was found, however, not to be removed by an ordinary Berkefeld filter or by centrifugation. The chemical bond was evidently a very loose one, (though enough to take the ionic calcium out of service) since it could be again separated with sodium hydrate and the ionic factor of the blood serum restored approximately to normal.

In order further to study this factor, we have injected normal rabbits, to determine the effect of various cultures in reducing the ionic calcium. These are shown in Figure 135.

COMPARISON OF CHANGES IN IONIC CALCIUM AND BLOOD MORPHOLOGY, DUE TO CULTURE INOCULATIONS

Date 1923	Hemo-globin	Erythro-cytes	Leuco-cytes	Poly-morpho-nuclears	Lymphocytes		Baso-philes	Arneth Index	Ca and Thrombin	Calcium Ionic	Ca Ionic and Combined	Calcium in Combin-ation
					Large	Small						
6–1*	85	6,900,000	15,000	**57**	7	**34**	2	83	15.20	**11.53**	13.00	1.47
6–2	85	6,150,000	27,700	**70.1**	14.4	**15.4**		48	17.20	**11.80**	13.22	1.42
6–4	80	5,750,000	19,800	**35.5**	8.8	**53.3**	2.4		15.00	**9.45**	13.66	4.21
6–5	80	5,800,000	16,600	**64.7**	16.8	**17.9**			17.00	**8.46**	8.71	0.25
6–6	85	5,100,000	14,800	**31.2**	6.4	**62.4**			17.40	**8.05**	9.80	1.75

FIGURE 135.

*Before inoculation. (Case No. 1406. Rabbit No. 1221.)

Another research was established to determine further the effect of infected human teeth in reducing the normal ionic calcium of the blood stream, by placing the patient's extracted tooth beneath the skin of a rabbit. This has disclosed a very remarkable result, as shown in the table in Figure 136.

This chapter is supposed to deal with chemical changes of the blood in relation to dental infections. There are so many elements and compounds in the blood that this subject could be

DEPRESSION OF IONIC CALCIUM BY IMPLANTING INFECTED TEETH

Case No.	Rabbit No.	Date	Weight	Weight Loss		Ionic Calcium			Pathologically Combined	
				Actual	%		Actual Loss	% Loss		Increase
404	1118	3– 5–23 3– 7–23	1153 1140	13	1	**11.06** **9.84**	1.22	11	0.00 1.16	1.16
1353	1107	2–20–23 2–23–23 2–26–23	1069 1020 846	223	21	**10.76** **11.74** **9.52**	1.24	11	2.11 0.48 3.92	1.81
1371	1145	3–27–23 3–28–23 3–30–23	1381			**10.13** **8.89** **8.95**	1.18	12	0.00 1.77 2.28	2.28
1317	1099	2–13–23 2–14–23 2–14–23 2–16–23	1822 1483 1385	437	24	**11.00** **9.92** **11.22** **7.91**	3.09	28	0.44 0.40 1.99	1.55
1317	1108	2–20–23 2–22–23	1265 1185	80	6	**10.59** **8.84**	1.75	17	2.02 4.16	2.14
1317	1109	2–20–23 2–22–23	1375 1256	119	8	**9.88** **7.82**	2.06	21	0.49 1.98	1.49
1317	1074	1–17–23	1925 1552	373	20	**12.37** **6.83**	5.54	45	0.64 0.6	

FIGURE 136.

extended to cover an entire volume. Referring again to Figure 131 of this chapter, we find that just as calcium has been a very variable factor, similarly so have several other substances. It is particularly important in the study of these to note their relation to each other; and, accordingly, you will note the incidence of a hyperglycemia to the calcium factors, total and ionic. It is not an accident, in referring back to the composite table, that where calcium reads very high the sugar of the blood is very high. We have, accordingly, studied the effect of dental infections and the relation of the types of dental infection to the different types of diabetes. A very important new light has been thrown on the whole problem of carbohydrate metabolism and sugar retention in the blood by Banting, Best, Collip, Hepburn, and Macleod, working at the University of Toronto, to whose work there are many references in the current literature.

It is not necessary to review here that, glycosuria, or the presence of sugar in the urine, is not of itself an evidence of hyperglycemia. Formerly, distinction was made between a true and false diabetes, the latter being cases in which there was the presence of sugar in the urine without serious harm to the patient. It is now recognized, particularly due to the work of Hamburger, that an increase in ionic calcium in the blood makes it possible for the kidney cells to let through a quantity of sugar where little or no sugar would pass with the slightly lower ionic calcium.

While the pathology and etiology of diabetes are not completely established, it has been interesting to note that in many cases the quantity of sugar in the blood has decreased following the extraction of infected teeth, and this disregarding in large part the variations and uncertainty of the significance of decrease of the sugar in the urine, which is a very common sequence to extractions. Such a case is shown as follows:

The culture grown from the tooth of Case No. 1195, when inoculated into rabbits, raised the blood sugar in nineteen days from 97 to 149, which, seven days later, had subsided to 121, and in twenty-six days still later to 92, five below the resting normal of this rabbit before inoculation. The rabbit was given another inoculation of this same culture, which, however, had been retained in the artificial medium, and on this occasion the same dosage increased the blood sugar to 105. This patient's resting blood sugar was 130 mgs. per 100 cc. and the urine sugar 430, or 4.3 per cent. This patient from whom the tooth was extracted and whose urine sugar was 430 before the extraction of the tooth, had the same reduced so that on two subsequent determinations, (the first one in eleven days,) no sugar was found in the urine, and in two years' time, the reports that she has brought to me have been that her physician has made frequent determinations and has not found sugar.

One of these patients' blood sugar reduced from 285 to 235 in twenty-four hours (probably a temporary change) after the removal of dental infections. He had no suspicion that he had diabetes. When advised to change his mode of living, and particularly to put himself under a strict program prescribed by a good internist, he advised that his business affairs would not allow of any change in his program for some months. This patient's urine sugar was 542 mgs., or 5.4 per cent. That this disturbance was progressive and very serious was evidenced by the fact that twenty days later it was 1100, and at this time we again urged that he put himself in the care of a skilled internist. It is important and pathetic to note that he was buried before that time. The importance lies in the fact that people can be in so serious condition and not have any knowledge or suspicion of it, and again may take the matter with so little concern, depending entirely upon their feelings which may be so misleading.

However, some individuals are capable of carrying a surprisingly large excess of sugar in the blood for considerable length of time. For example, Case No. 1268 has had blood sugar ranging from 295 to 410 mgs. per 100 cc., and a urine sugar ranging from 306 mgs., or 3 per cent, to 446 mgs., or 4½ per cent, for over a year, and while it has materially improved since the removal of a number of infected teeth, the glycosuria and polyuria persist. He feels so much better that he refuses my urgent recommendation that he be placed in the hands of a first class internist for direction of his diet. Fortunately, many of these individuals are continuing into a period when there is more hope for their assistance through the use of insulin and other pancreatic preparations.

Accepting the threshold of probable danger for the human as 120 mgs. of sugar, after fasting, per 100 cc. of blood, it is quite striking to find some sugars in our list going to 400, and, yet, the patients were not aware that they had diabetes. These are discussed from the clinical standpoint in Chapter 63. We will also discuss in Chapter 53 (on theoretical discussion) the significance of the type of gingival pathology which accompanies diabetes.

There is a phase of the role of ionic calcium which must be stressed. While the evidence available strongly suggests that certain rheumatic susceptibilities and conditions tend to develop in the presence of a low ionic calcium of the blood, it is equally evident that certain types of rheumatism develop in the presence of a high ionic calcium. While these seem to be associated with a different type of rheumatic symptoms and bone changes, the data available do not yet justify an attempt at classification that will be expected to be final, though it may be very suggestive. I speak of this at this point to forestall hasty conclusions and unwarranted deductions. In general, we may associate divergences from normal in ionic calcium of the blood with general types of lesions somewhat as follows:

Normals tend to have a mean of approximately 10 to 11. Condensing osteitis and lowered defense to rheumatic group infections seem generally to be associated with a lowered ionic calcium (6.9). A good defense shows a high normal ionic calcium. Active periodontoclasias and pyorrhea show an abnormally high ionic calcium (10.7-12.5). Individuals with a low ionic calcium do not have, while in that state, a tendency to extensive alveolar absorption. Patients with large apical areas of absorption have a high or normal ionic calcium; patients with relatively small apical areas, a low ionic calcium. Individuals whose teeth are sore, or have fistulae with recurring inflammatory processes, have normal or high calcium; those with low calcium have little or no local reaction and evidence, as pain about teeth which are infected. There are certain associations too: Patients with glycosuria and hyperglycemia, or the latter without the sugar appearing in the urine, or even those with sugar appearing in the urine without its being above the threshold which would term it "True Diabetes" tend to have a calcium higher than normal. I have discussed these various associations of calcium with the various pathological states in succeeding chapters.

URIC ACID.

A study of Figure 131 shows a variation in the quantity of uric acid present in the blood of the various patients there recorded, ranging from 1 to 7 plus mgs. per 100 cc. Various textbooks give us a normal of 1 to 3. A study of cases before and after removal of dental infections has shown a reduction of uric acid following the removal of dental infections. For years an important theory as to the etiology of rheumatism has been the increased uric acid of the blood. It is, however, now claimed by eminent authorities that, while certain of the rheumatoid group of symptoms—namely, gout,—have definite etiological relation to uric acid content of the blood, the other types do not have this direct relationship and the uric acid factor may vary through considerable range either coincidentally or independently.

ACID-BASE RELATIONSHIPS IN THE BLOOD.

It has been known for some years that while the hydrogen ion concentration of the blood varies but slightly from normal, this fact is only possible because of the very efficient buffer system contained in the blood, which has made possible quite wide variations in acid and alkali production without considerably disturbing the actual hydrogen ion concentration. It has similarly been known that the CO_2 tension in the alveoli of the lung automatically controls the rate of respiration, a practical illustration of which has been the necessity for a certain amount of re-breathing with nitrous oxygen and oxygen in order that the presence of the CO_2 may normally control respiration. In the system carbohydrates are oxidized at a rate which is determined largely by this acid base (or alkali reserve) relationship, which rate is directly influenced by hyperfunction of the thyroid. This condition of unbalance constitutes an acidosis. This has been determined or measured quantitatively by determining the amount of oxygen consumed, and of carbon dioxide given off in respiration during periods of rest and of fasting for from six to twelve hours. We have made a number of determinations on rabbits to ascertain if dental infections seriously disturb this basal metabolic rate, the animals being placed within the chamber connected with the metabolism machine. We have developed important data by this method but have found it a very laborious and tedious one. Several factors enter into this determination which cannot be controlled in rabbits as they can in the human. If the particular animal under study has been used to being handled and watched, the problem of excitability and fear will be reduced to a minimum, whereas in other animals, either because of lack of handling or a normal instinct of alarm and fear, these elements will completely change its rate of breathing and therefore its metabolic rate. That this is true is evidenced by the considerable variation in the readings that may be gotten with a given rabbit under different conditions of excitement. Another method—namely, that of directly reading the Ph with a potentiometer—is also very tedious because of the involved difficulties in maintaining the same CO_2 tension as obtained in the drawn blood, which cannot be allowed to come in contact with air, or be breathed upon by the operator.

The discussion of the acid-base balance and alkali reserve will be found in Chapter 44, Calcium and Acid-Alkali Balance.

SUMMARY AND CONCLUSIONS.

From these data it is apparent that very important chemical changes are produced in the blood from dental infections, and such as are far-reaching in the processes of metabolism and function. One of the very conspicuous changes is the disturbance of ionic calcium and the presence in the blood of a pathologically combined calcium. Since cell activity, both metabolic and catabolic, is dependent largely upon a normal ionic calcium in the fluids at the cell boundaries, very minute changes may produce very important physical disturbances, as, for example, the kidneys' permitting sugar to pass through when the ionic calcium gets above the normal limits. We would summarize some of the important chemical changes briefly as follows:

(1) Dental focal infections tend in many instances to lower the ionic calcium of the blood. (The fact and significance of a high ionic calcium in periodontoclasia are discussed in Chapters 45 to 56.)

(2) The placing of certain infected teeth in the blood serum of patients suffering from certain rheumatic group disturbances tends markedly to lower the ionic calcium of that serum.

(3) There is frequently found in the blood of individuals suffering from rheumatic group lesions, a reduced ionic calcium state and also a measurable pathologically combined calcium, which progressively disappears after a patient returns to normal.

(4) The placing of a patient's infected tooth beneath the skin of a rabbit tends similarly to reduce the ionic calcium of its blood. (The relation of these to the endocrine system is discussed in that chapter, No. 37.)

(5) Injection into the circulation of animals of the organisms grown from the teeth which produce these changes in the blood, tends also to reduce the ionic calcium of the animal's blood.

(6) The presence of dental infections tends in many instances to change the alkali reserve of the blood of patients; and when these teeth are placed under the skins of rabbits, they tend also to change their alkali reserve.

(7) Dental infections in some instances change the blood sugar content of the patient, as evidenced by the return to normal after the removal of dental infections, and the increase in blood sugar in animals, by injecting them with the culture from such teeth.

(8) The uric acid content of the blood varies considerably with the presence or absence of dental infections, particularly in cases of gout. It may, however, be very slightly changed in many cases of definite rheumatic group lesions, and its variation in the blood is very closely paralleled by its presence in the saliva.

Dental focal infections tend to produce, in many instances, one or several chemical changes in the blood, which changes tend also to be produced in animals when an infected tooth is placed beneath its skin; and, similarly, with certain methods of inoculation with the culture grown from these teeth. Some of the changes most frequently found involve:

(a) The ionic calcium of the blood.

(b) The presence of a pathologically combined quantity of calcium in the blood.

(c) A reduction of the alkali reserve of the blood.

(d) The development of acidosis.

(e) An increase in the blood sugar.

(f) An increase in the uric acid.

(g) The development of nitrogen retention.

(h) The development of products of imperfect oxidation.

CHAPTER XXI.

CONTRIBUTING OVERLOADS WHICH MODIFY DEFENSIVE FACTORS.

PROBLEM:—What are the contributing factors causing a break in resistance?

EXPERIMENTAL AND DISCUSSION.

We have looked upon dental infections as being sufficient or insufficient to produce the disturbances under consideration. This series of studies has been made to ascertain what, if any, are the factors which may be associated with dental infections and produce, or aid in producing, the break in resistance which expresses itself in systemic, as well as local disturbance. In a previous chapter, No. 4, we studied the basis on which human beings are comparable and found that, in the main, they may be divided into three groups with regard to their susceptibility to rheumatic group infections: namely, absent susceptibility, acquired susceptibility, and inherited susceptibility. In this chapter we desire to study the contributing factors to susceptibility by modifying the defense of the individual. These, we find, naturally divide themselves into two groups: namely, those which tend to make people with a normally high defense become susceptible in some tissue; in other words, changing a person from an absent susceptibility to an acquired susceptibility classification; and second, the forces which control in a case of inherited susceptibility and tend to make some particular tissue susceptible in that family.

An analysis of our collected data reveals that a very large number of individuals develop their rheumatic group lesions either as a complication with influenza or quite early following it. This will include not only rheumatic group lesions, more strictly speaking, but also bacterial invasions of other types such as tuberculosis and pneumonia. This latter, however, probably should, under these circumstances, often be considered as a rheumatic group lesion since it is often a systemic expression of streptococcal involvement already focal in the body. To determine this more exactly, I made a very careful study of influenza patients in five hospitals, three in this city and two in Columbus, in the epidemic of 1918. This was an exceedingly difficult study to make for several reasons: First, the patients involved were frequently too ill to be questioned with sufficient care to bring out all the data; and second, it was not possible to make roentgenographic studies, and many cases of dental infections were undoubtedly overlooked since only those were included, which were sufficiently gross to be determined definitely by oral examination, palpation, etc. A study of two hundred sixty influenza patients in five different hospitals, Figure 137, disclosed that when the patients were divided into two groups—those with, and those without clearly demonstrable dental infections—the percentage of individuals developing serious complications (in which we included pneumonia, empyema, carditis, severe neuritis and severe rheumatism) was found to be in the group without dental infections 32 per cent, and in the group with serious dental infections 72 per cent. Several factors should be carefully noted: In the pneumonias, the tendency to strangulation following coughing spasms, as a result of the bronchial exudate, produced violent inspirations which draw into the lung, fluids and infections from the mouth. This makes gingival infections a very marked contributing factor to the development of pneumonia. In general, however, the so-called locked infections (by which we mean those at root apices without opportunity for drainage into the oral cavity, which therefore must drain into the system, into the lymphatic and hematogenous circulations) are more to be feared since the system must of necessity become invaded from this source, with a breaking down of the local defense which has tended to wall off and defend the patients in times of their normal defense.

In our studies of this series we have found that, in the patients who have kept their mouths in good condition, free from gingival and apical sources of infection, both the incident of influenza itself, and complications with the influenza, are much lower than in the patients with extensive dental infection. This fact has also been reported to me by other dentists.

These general observations, together with the observations made in private practice, seem to demonstrate that the individuals, who carry focal dental infections with apparent safety during the period of their normal health, immediately are endangered, and very seriously so, when they are attacked with influenza

ORAL INFECTIONS AND INFLUENZA COMPLICATIONS

Hospital	Date	No. of Flu Cases Studied	Flu Only	Flu with Various Compli-cations	Flu with Pneu-monia	With Oral Infection			Without Oral Infection		
						Total	Flu Only	Flu with Compli-cations	Total	Flu Only	Flu with Compli-cations
1 Lakeside, Cleveland Men's Ward	Nov. 30	20	13-65%	7-35%	7-35%	8-40%	2-25%	6-75%	12-60%	10-83%	2-17%
2 Lakeside, Cleveland Women's Ward	Dec. 1	6	1-17%	5-83%	5-83%	5-83%	1-20%	4-80%	1-17%	0	1-17%
3 St. Francis Columbus	Dec. 4-5	23	5-21%	18-78%	9-48%	18-22%	3-17%	15-83%	5-78%	2-40%	3-60%
4 Grant, Columbus Nurses	Dec. 5	50	41-82%	9-18%	2- 4%	0	Held certificates from dentists		50-100%	41-82%	9-18%
5 Grant, Columbus Private Patients	Dec. 5	51	38-74%	13-26%	8-16%	0	None known		51-100%	38-74%	13-26%
6 City Hospital Cleveland	Dec. 7	26	8-31%	18-69%	15-51%	23-88%	7-30%	16-70%	3-12%	1-33%	2-67%
7 Mt. Sinai Cleveland	Dec. 19	31	14-45%	17-54%	10-32%	21-68%	8-38%	13-62%	10-32%	6-60%	4-40%
8 Mt. Sinai Cleveland Nurses	Dec. 16	53	38-72%	15-28%	13-24%	0	Clean mouths		53-100%	38-72%	15-28%
Eight Sources		260	158-61%	102-39%	69-26%	75-29%	21-28%	54-**72%**	185-71%	136-68%	49-**32%**
Private Practice Patients		37	14-38%	23-62%	14-23%	37-100%	14-38%	23-62%	0		

FIGURE 137.

infection. In order further to test this problem, I have in the succeeding epidemics made inoculations into the tracheae of rabbits of the washings of the nasopharynges of the Flu patients, and have made comparative tests to determine whether or not these rabbits were more susceptible to focal infection. Figure 138, lower view, shows a reproduction in natural color of the lungs of a rabbit dying from pneumonia, as illustrating this condition. A rabbit was inoculated intratracheally with the washings from the nasopharynx of a Flu patient during the first twelve hours after the attack. This rabbit showed a depression in its mononuclear blood cells, typical of the influenza involvement, and was less active following the injection, with loss of appetite. (Rabbits so inoculated recover in a few days.) It was killed, and its lungs removed and macerated in normal salt solution, which washing was injected intratracheally into other rabbits. These developed the same general symptoms. The lungs of this rabbit are shown in Figure 138, upper view. Of the two injected intravenously with small quantities, ½ cc. of a 24-hour culture from dental infection, this one died of pneumonia (Figure 138, lower view). This experiment was made in the epidemic of February, 1923. A similar observation was made in the epidemic of 1922. A rabbit was injected intratracheally with the washings from the nasopharynx of a patient suffering from influenza, followed by the intravenous injection of a dental culture. This rabbit developed typical streptococcal pneumonia. A section of the lung is shown in Figure 139. Similarly, we have found that, when infected teeth are planted beneath the skins of the rabbits, they sometimes develop acute and terminal pneumonia. (See Chapter 61.)

PREGNANCY.

An analysis of the sex in Figures 42 and 47 in Chapter 4, reveals that the increase in the prevalence and severity of rheumatic group lesions shows a continued increase toward the female sex. A further analysis of these data reveals that in a great many instances, the acute attack of rheumatism dates directly to the time of pregnancy and lactation. The usual age of first rheumatic group involvement is between twenty and thirty-five in females.

Since so many young mothers, or expectant mothers, have developed acute rheumatism, heart, or other rheumatic group lesions, during the period of gestation or lactation, we have come to recognize this as one of the most important overloads which

FIGURE 138-A. INFLUENZA LUNG OF A RABBIT INOCULATED INTRATRACHEALLY WITH NASAL WASHING FROM A FLU PATIENT. THIS IS NOT FATAL TO RABBITS.

FIGURE 138-B. FATAL STREPTOCOCCAL PNEUMONIA FROM INJECTING DENTAL CULTURE INTRAVENOUSLY INTO RABBIT WITH INFLUENZA.

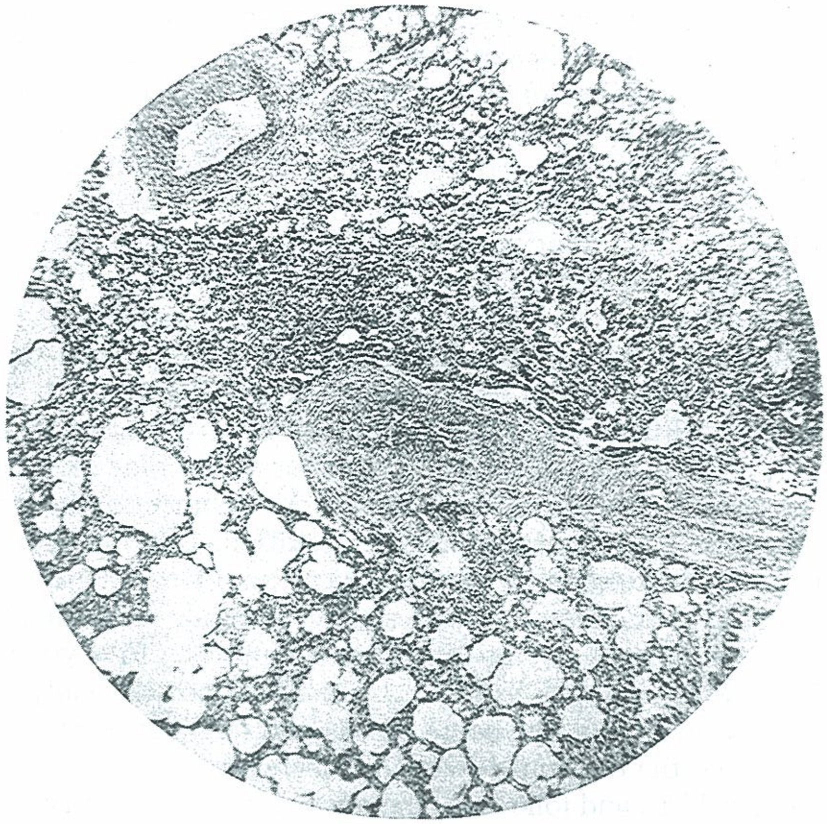

FIGURE 139. SHOWS A SECTION OF THE LUNG OF A RABBIT WHICH DIED OF STREPTOCOCCAL PNEUMONIA FROM A DENTAL CULTURE. THE BRONCHIOLES ARE OBSTRUCTED BY THE THICKENING OF THEIR LINING MEMBRANES AND BY THE EMPHYSEMA.

we have to consider. To illustrate:

A young married woman of twenty-two years undertook the responsibilities of motherhood with a physique and physical reserve which would be expected to be well above the average. In childhood she had had a mild endocarditis which left a mitral leakage. She also suffered from acute rheumatism, having had two quite acute attacks. After the birth of her babe, which was unusually robust, and during the early period of lactation, she developed such severe rheumatism that she could not wait on herself or the baby. A couple of infected teeth were removed and she immediately improved, though her relief was not complete. She ceased nursing her child in about six months, and immediately her remaining rheumatism disappeared. During the time of lactation she had been drinking large quantities of good milk.

We have seen this clinical picture over and over, and decided to make a test on rabbits. Accordingly, the following experiment was made. A rabbit referred to in Chapter No. 66, which had developed a complete paralysis posterior to the first lumbar vertebra, with loss of continence of urine and fæces, and complete loss of all sensory and motor control of the posterior region, and which had recovered so completely as to be apparently normal, except for a slight atrophy of a few groups of muscles, which caused a rotation of one hind leg, as shown in illustration, and which had gained in weight from 1025 grams to 1600 grams, was tested for the effect of maternity on its resistance to the residual infection. The roentgenograms showed both the location of the lesion and its structural change. The rabbit itself is shown in Figure 140, the roentgenogram of the spine in Figure 141, and a section of the repaired lesion about nine months after the original inoculation and the first development of the lesion of the spine. At the beginning of gestation, this rabbit was apparently in exceedingly good physical condition. It was fat, its coat sleek, and in every way seemed normal. It went through term with apparently no untoward symptoms. Five young were born, which seemed, at birth, to be developed normally. They all died in from a few hours to one and one-half days. She did not have the continued overload, therefore, of lactation. Prior to their birth and following, she was observed to be very nervous, easily frightened, and excitable. Her nervousness increased in severity and she began losing in weight, finally developing a very marked disturbance of the central nervous system, with choreic motions, rotation of the head with the least excitement, and she became so nervous that if the hands were clapped very hard near her, she would fall over on her side. She died in about five weeks' time with a terminal pneumonia which, on culturing, showed a streptococcus and diplococcus similar to that found in the lesions and similar to the organism injected, and originally secured from a tooth. Under another chapter, we will speak of pregnancy complications. It is my opinion that the lesion in the spine, though healed, retained the same type of infection which had originally been injected into the rabbit and which produced the spine lesion; that, at the time of lowered resistance, this organism about which Nature had been able to build a barrier, became rampant, again affecting first the overloaded nervous system, and later became focal as pneumonia.

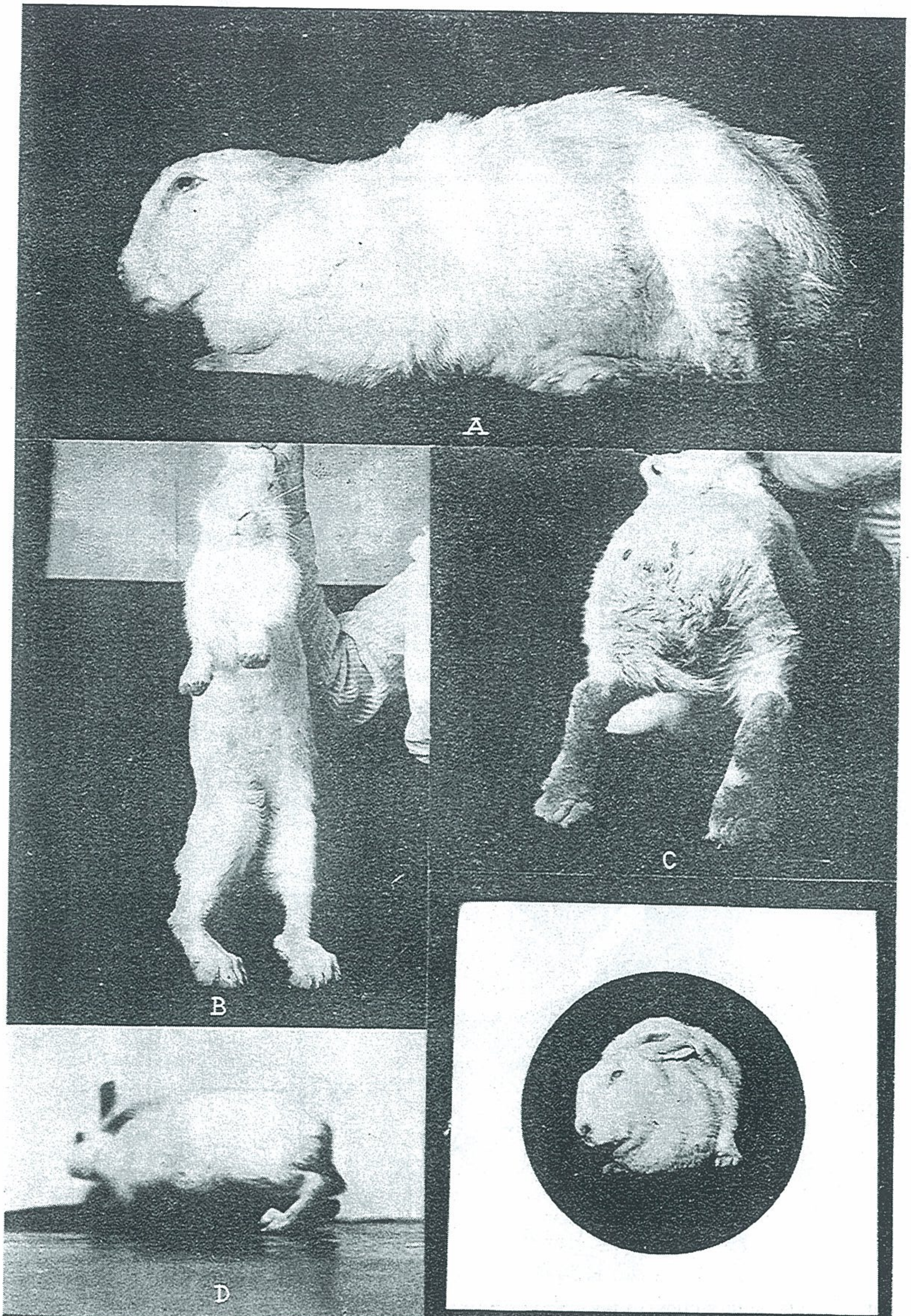

FIGURE 140. A RABBIT IN WHICH COMPLETE PARALYSIS WAS PRODUCED FROM THE CENTER OF THE SPINE BACKWARD, BY DENTAL CULTURE INFECTION. IT APPARENTLY RECOVERED ALMOST COMPLETELY EXCEPT FOR ATROPHY OF A FEW MUSCLES WHICH PRODUCED A TWISTED LEG.

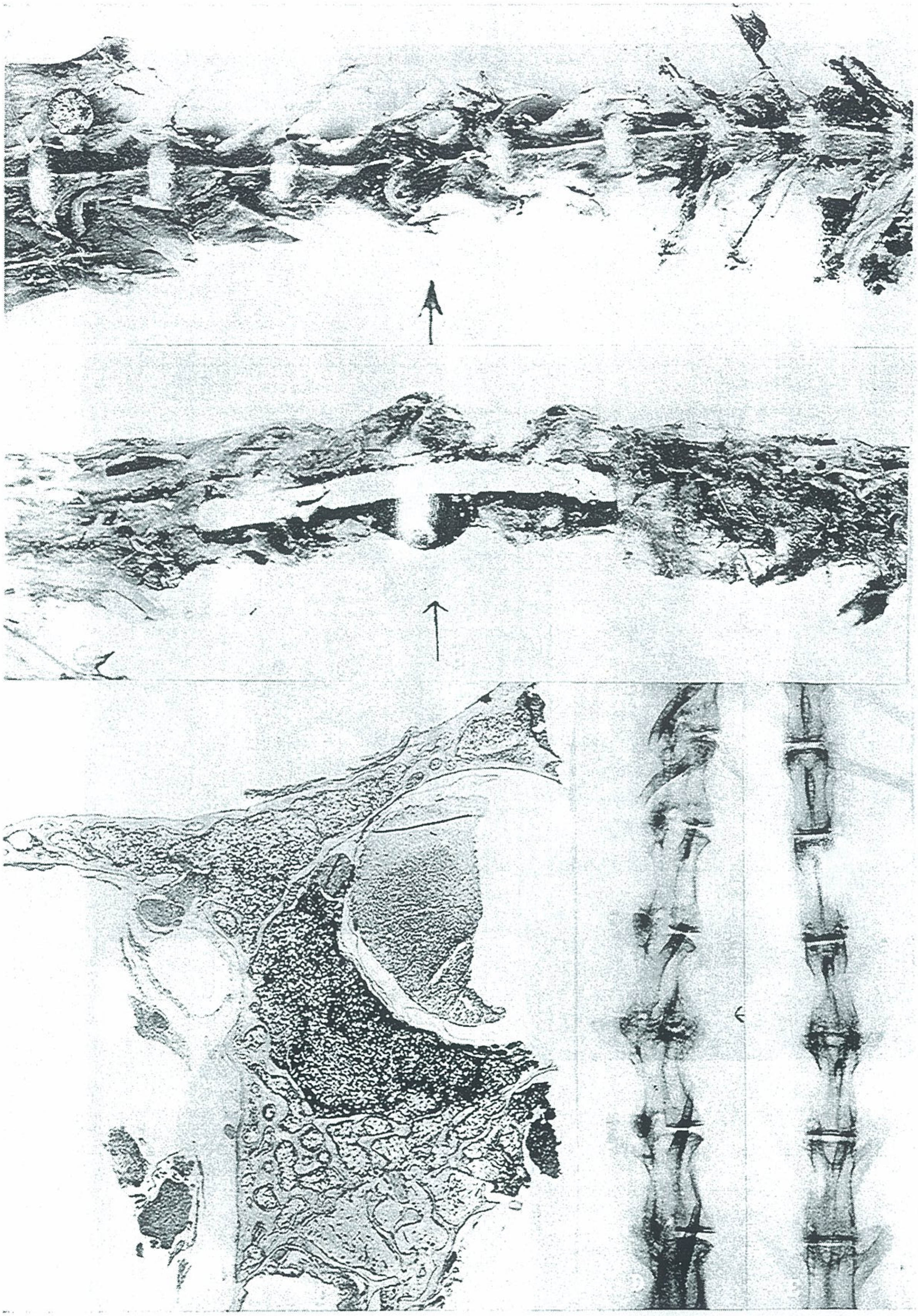

FIGURE 141. VIEWS AND SECTIONS OF THE SPINE OF RABBIT SHOWN IN FIGURE 140. NOTE DESTRUCTION OF INTRA-VERTEBRAL CARTILAGE IN A AND B; DESTRUCTION OF BODY OF VERTEBRA IN C; AND MILD CONDENSING OSTEITIS IN D AND E.

TRAUMA.

Acute irritations producing an active inflammatory process would seem to increase, or at least not reduce, the local defense of most tissues; whereas, a continued irritation tends to reduce the resistance of the tissue involved and make it more susceptible to infective invasion. To illustrate:

A man who had been on shipboard in passage from the Philippines to this country, following which he was on the train from San Francisco to Cleveland, all of the time without much exercise, secured a position as floor walker or night watchman in a manufacturing plant, having acres of cement floors, requiring several miles of walking for his beat to ring in the record of his rounds. He wore shoes without rubber heels. The irritation to the synovial membranes at first gave him a sense of discomfort, which was relieved by resting. After continuing the irritation for a few weeks, he was afflicted with acute rheumatism in the joints receiving the thrust. During all of this time he had had chronic dental infections. The removal of the dental infections, together with the use of rubber heels, relieved the synovitis and he was able to continue in the same occupation.

Similarly, eye strain makes the eyes much more susceptible to irritation from dental infection. Figure 6, Chapter 1, illustrates the dental condition of a patient who had such a disturbance. He had suffered for several years from the difficulty of not being able to read for long periods, a condition which was helped by periods of rest but not entirely relieved. It was also helped, but not relieved, by glasses. The removal of his dental infections made it possible for him to discard his glasses which he had worn for fifteen years, and made it possible for him to read without limit and without discomfort. We have had many cases where the patients have reported to us, after the removal of their dental infections, that they did not longer need to use their glasses.

GRIEF AND WORRY.

Similarly, an overload of mental strain makes individuals more susceptible to dental infection. Probably no contributing factor so greatly lowers the defense, unless it be an influenzal infection, as the mental condition in grief or anxiety. To illustrate:

A family of five girls nursed their father through a long illness, terminating in death from pernicious anemia, which was followed by a severe illness of the mother and final death from heart involvement. These girls were not physically exhausted from nursing duties, but were nearly prostrated by grief. All developed rheumatic group lesions such as neuritis, and two had heart involvements. The conditions were very markedly improved, and the neuritic symptoms relieved by the removal of dental infections.

An experiment was made to determine the effect of worry. Two ferrets were secured and put into a compartment within a rabbit's cage. At first the rabbits seemed very fearful of them but as soon as they learned that the ferrets could not hurt them, they entirely ignored their presence. Results of this test were negative.

EXPOSURE.

Physical exposure is probably one of the most serious and most common of the overloads which come upon the human body to lower its defense and make it a prey to the focal infections against which it has, under normal conditions, an ample defense. In Chapter 64 on acute rheumatism, I give the history of a case of a man who was a partial invalid from a form of neuritis for over twenty years, following exposure, repairing a burst water main in the winter, which threatened to do great damage to the factory in which it happened. This man worked in the nearly ice-cold water for about two hours repairing the break. At the time of this exposure, and for a year preceding, he was having dental work done, including the treating of several infected teeth. His neuritis was so severe that it drew his knees nearly to his chin for many weeks, and one arm was almost useless from atrophy.

To test this we have made cultures from the teeth of a patient suffering from rheumatism and have made inoculations into four rabbits, two of which were used as controls, kept in a warm cage, and received the same inoculations as the two being tested, which latter were submitted every day or two to exposure of cold, by having their hind legs immersed in water with broken ice in it. The dosage in these cases was purposely small. The control rabbits received twelve inoculations within a period of thirty-two days. The two exposed to the chilling received the same inoculations on the same days. All of the rabbits lost in weight following the inoculations. The average loss of weight of the two that were not exposed to the chilling was 10 per cent, and of the two that were exposed was 14 per cent. But this is not the most important effect of the exposure. The two that were not exposed did not develop any lesions from the inoculations. One is still living four

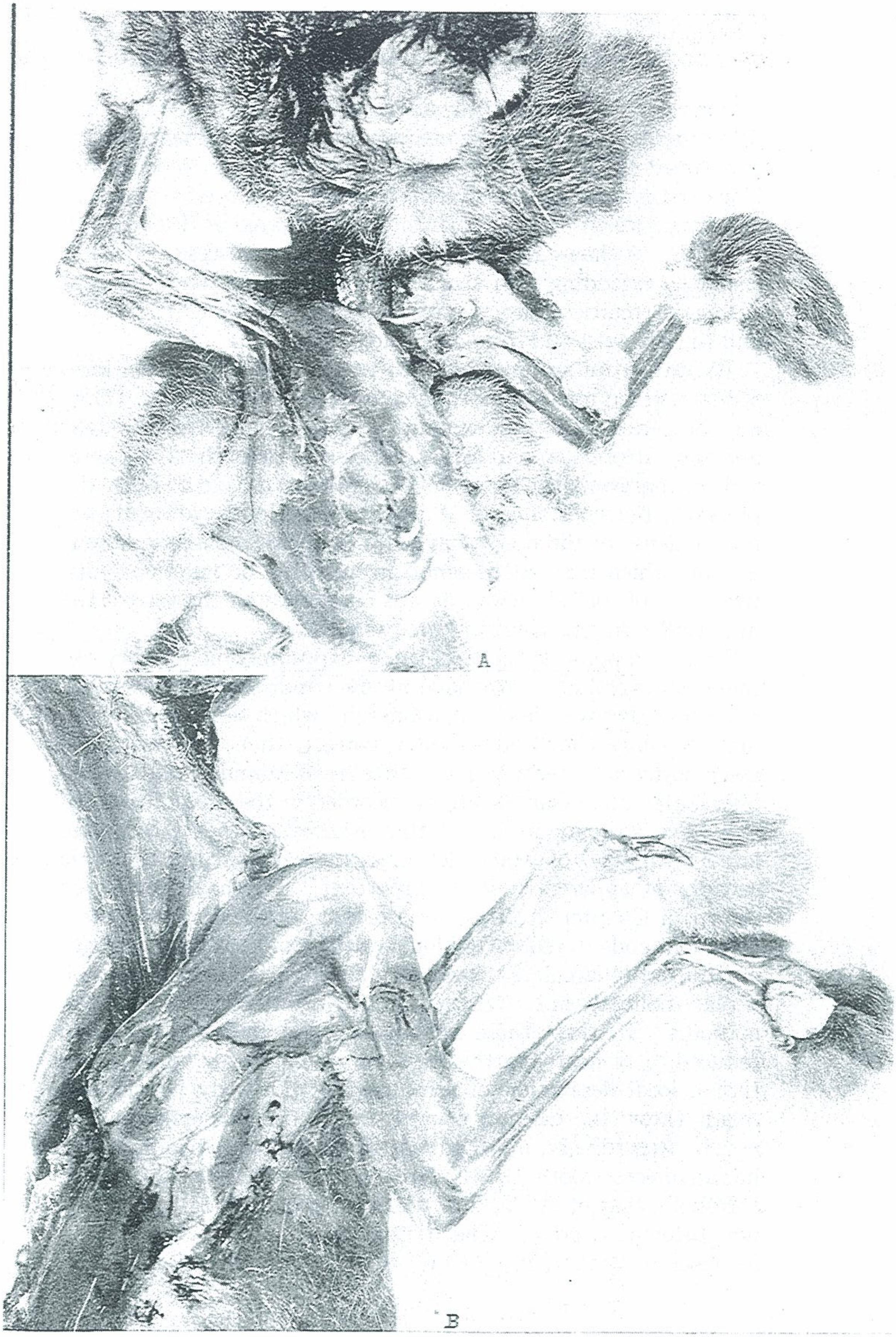

FIGURE 142. A AND B SHOW TWO RABBITS WHICH DEVELOPED ACUTE SUPPURATIVE ARTHRITIS FROM SMALL INJECTIONS OF DENTAL CULTURE, PLUS CHILLING IN COLD WATER. THE TWO CONTROLS RECEIVING THE SAME CULTURE BUT NOT CHILLED DEVELOPED NO LESIONS.

months after inoculation, has gained in weight to 241 grams. The other has recently been used for another experiment, it having gained considerably in weight following its last inoculation. The two that received the chilling, however, developed very severe rheumatic lesions, both of which are illustrated in Figure 142, A and B. A shows an extensive intracapsular abscess of the left shoulder, extending into the muscle sheath. B shows a subcutaneous multiple suppurative arthritis of the right shoulder and the left wrist and third and fourth digits.

We have here an illustration of what happens when an individual with an infected tooth is exposed to severe chilling. During the period of normal exposure he may have a loss in weight and some depression and injury, expressing itself chiefly as lassitude or marasmus, or both; but on exposure to cold or other depressant, the resistance of the tissue is seriously lowered, the total defense of the body goes down, and the rheumatic lesions appear, which may be rheumatism, neuritis, or functional disturbances of special organs, as was illustrated by the case of the man chilled in repairing the burst water main.

There is a phase of this problem of exposure which is very obscure and involved. We have all seen individuals who would remind us that their joints felt like rain, which seemed clearly to indicate, since they were so often correct, that their biological mechanisms were responding to changes of humidity and atmospheric pressure. Our researches recorded in the preceding chapters have strongly indicated that whatever other factors are largely variable, of which doubtless there are many, the ionic calcium is apparently very important. We have found, as shown in Chapter 20, that the presence of focal streptococcal infection tends to reduce the ionic calcium of the blood throughout the entire circulation, and very markedly so when the tooth is placed directly in the serum of freshly drawn blood for a couple of hours. If, then, these individuals have their ionic calcium reduced to, or nearly to, the threshold, we can readily understand that a local depression of some part of the body by chilling, might throw the calcium content below the threshold at that point. Accordingly, if the patient sits in a draft when he or she has an infected tooth, the circulation ionic calcium being near the threshold, that of the back of the neck where the draft strikes is sent below that point. That tissue, then, has lost, temporarily, its reserve defense; it is a prey to the toxic invasion passing through the system, and more or less severe symptoms from a mild myositis to a muscle spasm with torticollis are the result; or there may be neuritis of the cervical nerves. The problem of tissue temperature has also a very direct effect on susceptibility and defense.

Two things are most logical as a treatment: First, to remove the source of infection after it can be found, (not that all toxic irritations come from dental infections or even all from focal infections) and to raise the defense of this local tissue by both the application of heat and the liberation into the fluids of the involved tissue of ionic calcium through the process of massage. To test the first of these we have very many times, as recorded in subsequent chapters on the pathology of systemic dental infections, completely relieved these symptoms permanently or for years by the removal of dental foci, though they had been coming with very great frequency. To determine the form of different mechanisms that are operative in the process of massage of various types, we have made determinations of the ionic calcium of the blood in a given tissue, say a rabbit's ear, and then massaged the rabbit's ear first lightly and made determinations, then vigorously, and finally very vigorously, and have found that mechanical irritation of the tissue slightly changes the ionic calcium of the blood from that tissue.

It is a question whether the osteopaths will ever appreciate, and it is to be hoped the laity never will, how much that group is indebted to the dental profession for the preparation of a group of patients with an affection that will keep them coming; and we trust that the gratitude of the patients who have had these successive treatments made unnecessary, will adequately offset the curtailment of the visits to the physical therapists which will result from a final intelligent removal of dental foci.

In the chapter on sensitizations we discuss not only anaphylaxis as an antigen-antibody reaction, but also the phenomenon of local tissue sensitization, which it is probable are important factors in the development of this infinitely sensitive mechanism which makes it possible for the grandmothers and grandfathers and some not yet graduated into that class, to be such efficient weather prophets.

NUTRITION AND HUNGER.

Since every machine is dependent upon its supply of fuel if it is to maintain its output of energy, the human body is dependent upon the nature and quantity of its food supply. In the chapter on nutrition we discuss this definitely from that viewpoint, for few, if any, of the overloads of the human body will so often be found to be a contributing factor as will faulty nutrition. In this chapter we are discussing the relation of overloads to dental focal infections. There are, accordingly, two distinct methods by which this occurs. The individual who is hungry is more susceptible to infection of any kind. For years it has been the teaching, in medicine, that no man should go into the presence of contagion, hungry or with an empty stomach, because his defense would be lowered. The reasons for this are most apparent. Without an adequate fuel in the furnace, the generation of heat is diminished and the temperature of the dwelling decreases. The chemical processes of the body are practically all increased with an increase of temperature, with each increase of which, above normal, the reactions of the body are increased approximately ten per cent; and, similarly, with each decrease of which, the reactions are reduced approximately a similar amount. This is a fundamental factor in the problem of exposure to which we referred in a previous paragraph.

But there is another important method by which nutrition directly acts upon the process of defense. Metabolic processes, and particularly calcium metabolism, are dependent upon certain chemical substances, the nature of which we do not yet understand, which are derived from various glandular tissues of the body, generally referred to as the glands of internal secretion, and upon certain substances spoken of as accessory food factors or vitamines, a certain quantity of which will be necessary for metabolic and catabolic processes. The quantity factor of these is, incidentally, small and is discussed in the chapter on nutrition.

EXHAUSTION, PHYSICAL AND NERVOUS.

It has long been said that work never kills but worry does. It is exceedingly difficult to determine what are the chief factors involved in exhaustion, partial or complete; that is, whether the fatigue is chiefly a quality of the musculature or of the nervous system. Our clinical experience reveals many cases where partial exhaustion, or long continued overload of work, seems to contribute very directly to increasing the susceptibility to focal infections. It is exceedingly difficult in any clinical case to eliminate such factors as worry, lack of nutrition, exposure, etc. Because of this difficulty to exclude all other contributing factors, it seems unsafe to quote clinical cases.

ACUTE AND CHRONIC INFECTIONS.

It seems probable that any disturbance, whether physical or infectious, tends to disturb the normal defensive factors of the body. A very common overload of an infectious nature, but fortunately much less common than previously, is typhoid fever. We find in our case histories that many patients have their first attack of acute or chronic rheumatism accompanying or following typhoid fever. To what extent these patients would have been involved had they not had focal streptococcal infection is, of course, impossible to state. The following seems to be a typical history of some of these cases.

About thirty years ago this patient had typhoid fever at the age of about twenty. Immediately following her typhoid, she developed acute rheumatism with progressive arthritis deformans. I have watched her case for twenty-eight years, during most of which time it became progressively worse until she was entirely unable to walk. I sent for her six years ago in order that I might remove some teeth that I had crowned twenty years previously. The removal of these dental infections not only prevented the condition from becoming progressively worse, as it had been advancing, but since that time it has become progressively better. It seems very probable that the acute onset was largely due to her broken defense, and its attending disturbances of nutrition, etc., occasioned by her typhoid infection.

SYPHILIS.

In discussing the role of another infection as being contributory, the matter becomes much more complicated when we consider an organism which, of itself, is able to produce lesions which may be comparable to those produced by the injected streptococci. It is, however, a frequent observation that patients presenting with involvements of the nervous system and at the same time carrying a dental focal infection, seem to be more susceptible to their dental infections than normal.

The two types of nerve reactions are sufficiently different to be differentiated, even when present at the same time in the same patient. The streptococcal involvements are acute and recessive, the recurrences and exacerbations being characteristic in that they are relieved by salicylates, and are improved by massage. On the contrary, the syphilitic disturbances are more persistent and continuously progressive without the marked exacerbations and recessions that accompany streptococcal involvements. The removal of the dental foci will often relieve permanently these typical rheumatic type disturbances, while the less variable and progressive symptoms of the syphilitic infection will not be relieved by the removal of the dental foci. In a later communication I will discuss the means for differentiating, and the frequency of the presentation of lesions, which are thought to be rheumatic of streptococcal origin, but prove to be syphilitic processes. In a later communication I will discuss the need for careful differential diagnosis between streptococcal focal infection reactions as rheumatism, particularly of the knees, and rheumatic disturbances of very similar nature of gonorrheal origin.

ALCOHOL.

It is not strange that alcoholics are more susceptible to streptococcal infection from focal dental infection or other source than normal. Any substance which, when taken into the system, disturbs cell function would be expected to disturb defense. This is a very common clinical experience.

SUMMARY AND CONCLUSIONS.

The individuals constituting the members of the group we have designated as having an acquired susceptibility, have in large part come into that group as the result of overloads plus focal infection. It is probably not fair to speak of old age as an overload. It is, however, true that much less increase above normal constitutes a potential overload for the aged than the middle-aged; and, similarly, of the middle-aged than over the vigorous young.

The overload that we have most frequently found to have been the cause of transferring an individual from the group of absent susceptibility to acquired susceptibility is influenza. And in this connection it is significant that statistics that have been accumulated in England and Wales have shown that in the two years following the epidemic of Flu, approximately four times as many individuals died from the complications following and incidental to Flu as did from the Flu attacks. Our own studies have also shown that the prevalence of grave complications with influenza is two and one-third times as great in the group of individuals having dental infections, as in the group free from dental infections. It now seems evident from these and our inoculations on animals, that focal dental infections are a potential danger, because of the complications arising from them incidental to an attack of Flu, of far greater seriousness and importance than has been realized. Such an individual is carrying a potential charge of dynamite which may, when least expected, explode and involve his system and gravely endanger his life, for, of the individuals with complications whom we have found in the hospitals, thirty to fifty per cent died.

We have alluded to the fact that, whereas the percentage of males and females in the group which we designate as non-susceptible, is approximately equal, the percentage of females to males rapidly increases in direct proportion to the severity of the susceptibility, changing from fifty-fifty in the absent susceptibility to ninety-three per cent of females in the strongly inherited group to seven per cent of males. Our studies have shown that this change in ratio is largely the result of the increase of overloads induced by motherhood. Pregnancy is, therefore, one of the most important overloads which must be reckoned with, and prepared for, to which the human body can be subjected.

Similarly, overload of tissues such as physical injury, extreme anxiety, exposure, nutrition and hunger, exhaustion, physical and nervous, acute and chronic infections, syphilis, and alcohol, all contribute singly or collectively to breaking an otherwise ample defense. The individual without the dental infection, would suffer depression or exhaustion, from which he would readily recover, but with the presence of focal infection, finds himself the victim of an enemy he does not recognize and cannot reach. He breaks in some of the special tissues, frequently the one that has been overloaded or exposed. The tendency has been to treat the symptom, and it has often taken long periods of time for the patient with forced rest and every effort to reduce the overloads, to regain the mastery of his focal infection. Our clinical experience is that scores of these people go right on with their overloads, with complete relief from their physical symptoms due to the infections, when the dental infections or other focal infections are removed, showing clearly that the primary disturbance has been the infection. The individual has proved to have ample strength to carry his normal physical burden if relieved of his focal dental infection, which latter will often be sapping his system of more of his vitality and nerve energy than will his entire physical and nervous energy combined.

We would briefly summarize by saying that the individual with an absent susceptibility to rheumatic group lesions, is in a condition of only relative safety, for at any moment that his overloads of age, physical and nervous expenditure, exposure and grief, contributing infections, shall singly or in combination lower his total defense, he will become a prey to that dental infection which will attack his weakest link, which in these cases of an acquired susceptibility, whether in part or in combination with a mild heredity, will tend to be the most susceptible tissue, and most frequently the nervous system.

Dental infections, while potentially harmful, may not be causing apparent or serious injury until the individual is subjected to some other overload, at which time a serious break may come. The chief contributing overloads are influenza, pregnancy, lactation, malnutrition, exposure, grief, worry, fear, heredity, and age.

CHAPTER XXII.

ELECTIVE LOCALIZATION AND TISSUE AND ORGAN SUSCEPTIBILITY PHENOMENA.

PROBLEM: Do the organisms of dental infections possess or acquire tissue affinity and elective localization qualities?

EXPERIMENTAL AND DISCUSSION.

Seldom in the history of any problem in medicine has the storm centered around a single individual as it has, in this instance, around the pioneer worker and chief advocate of elective localization on the part of streptococci, Dr. E. C. Rosenow; and few, if any, of the contributions to medicine of the last two decades are likely to promise so great helpfulness to humanity, as the development of the relationships of focal infections to the degenerative diseases. The antagonism and opposition have grown out of a lack of knowledge of the mechanisms constituting the attacking forces of the invading organisms, and of the defensive forces of the hosts. (The bibliography of this discussion would cover hundreds of pages of references and would not be necessary or appropriate here.)

Probably no problem in connection with this most complicated group of pathological processes is so complicated and so certain to be misunderstood by many who read as will this phase. To many people there will be little difference at first thought between elective localization, and organ and tissue susceptibility, notwithstanding they are very different, since the former pertains to qualities which relate exclusively to the bacterium, and the latter to qualities which relate exclusively to the invaded special tissues. These are so distinct that they should be discussed in two separate chapters. Yet we have combined them in order to emphasize the need for their differentiation by specific reference made continually through this discussion.

There is another reason why the discussion of this subject is unusually difficult: namely, the prejudice amounting to almost unalterable convictions in two groups, one of which has maintained, that elective localizations should not be anticipated because of evidence developing from experimental data. Some of the disbelievers have tried and it did not work; therefore, it is not so.

A review of the elective localizations resulting from inoculation of a thousand rabbits with cultures taken from dental infections of various types, with and without acute processes on the part of the patient, but generally with, should, according to the claims of the extremists, prove or disprove whether it is possible for bacteria to carry into animals an elective localization quality corresponding with the lesion of the host from whom the tooth was extracted.

A study of any successive thousand inoculations would necessarily include so many different varieties of problems that the data would not all be directly related to this particular phase of elective localization. Instead, then, of making our specific studies of the quality of elective localization as an inherent property of the microbe, we have taken groups of inoculations from individuals with, and without acute processes in various tissues of their bodies, and in whose cases the organisms had been taken from the teeth, and after culturing by comparable methods had been inoculated in comparable doses into animals by comparable methods. Results of these studies and comparisons are more instructive than could be a miscellaneous group of inoculations made under dissimilar conditions and by methods that were not comparable.

In the first place, this group of experiments includes all types of studies, many of which were not even indirectly related to discovering the qualities implied in elective localization. In fact it includes, in many cases, experiments made to demonstrate that elective localization would not take place under the conditions of the experiment. There are so many factors involved which, as yet, even those best informed from intensive study do not understand, that no group of figures can be considered conclusive. To illustrate:

Few animals were considered more susceptible or more certain of death from inoculation of the anthrax bacillus than the guinea-pig; and, yet, it is now demonstrated by Besredka and Noetzel[12] that, if the guinea-pig could exist without its skin, it would be entirely immune to anthrax; that it can be injected with lethal doses into the blood stream or peritoneal cavity without serious

[12] See bibliography.

injury, but the most extreme care must be taken, not to permit one organism to reach the skin. If, then, two experimenters undertake to test whether or not the anthrax organism will kill guinea-pigs, without a knowledge of this fact, one individual may prove conclusively that the animal is immune because his technic is actually different, though apparently the same, in the matter of contamination of the skin with the organism in the process of inoculation.

The attitude of mind on this question can be illustrated by the following experience of the writer. I received a letter from a man in another state, stating that he was sending, under separate cover, some teeth that he had extracted from a patient, and that he desired to test whether or not there was any truth in the theory of elective localization by having me inoculate rabbits and develop in them the lesions, and report the lesions to him; and, if they were the same as those suffered by his patient, of which, of course, I was not informed, he would believe in the theory; and, if not, it would be proof to the profession that there was no truth in the theory. The teeth arrived after many days in a dry condition in a pasteboard box, overgrown with a mold, probably contamination. Of course, this is not comparable to that of the just critics of elective localization; and we have desired to keep ourselves free from opinion and prejudice for either side of the argument in conducting these studies.

The following experiment was made to throw light upon the question of the influence of the culture medium upon the quality of elective localization, if such resided in the organism. A culture was made from the live but infected pulp of a tooth of a boy nine years old who was suffering from an acute endocarditis and acute rheumatism with very severe symptoms. Thirty rabbits were inoculated in the first group with the culture grown out in ascites broth, 24 hour growth, centrifuged, washed, and inoculated in normal salt solution. One hundred per cent of the rabbits developed acute rheumatism, and 93 per cent acute heart involvements. The same organism was grown on artificial media for seventeen days, when another group of animals was inoculated, only 10 per cent of which developed heart lesions. All other conditions were as nearly constant as we could maintain them.

On inquiry of men who have stated that they had succeeded in developing evidence of elective localizations, and of others who have similarly experimented, but have not developed localiza-

SUMMARY OF ANIMAL REACTIONS AND PATIENTS' SYMPTOMS

Case Number	Patients' Chief Lesions	No. of Rabbits	Percentage with Major Lesions	Heart	Lungs	Liver	Gall-Bladder	Stomach	Intestines	Appendix	Kidneys	Urinary Bladder	Generative Organs	Joints	Muscles	ands	Spinal Cord	Brain	Eyes	Rheumatism Clinical	Chloroform
1124	Pelvic Inflammation and Discharge. Neur.	7	100	43	14	14			14		14		28	57	14	14				14	86
1087	Eyes. Nervous System	11	100	9	9	27	9		9	9	9			9	18		9		100		82
1094	Acute Rheumatism	12	42	33	8	33	8		33	8	33			25	16					16	75
1095	Heart. Rheumatism	2	100	100	100	50	50		50		100				50			50		100	0
306	Rheumatism	3	33	33									33		33					33	100
1098	Lassitude	6	67	67	33	33	17	17	33		50				50						50
381	Arthritis	2	50			50								50	50					50	50
1081	Neck and Shoulder Inv.	5	40	80		40			20	20	40		20		20	20	20	20		40	20
1085	Ovarian Pain	6	67T 100F	33		83	17		67	17	50	33	67T 100F								50
1050	Rheumatism. Heart. Uterine Pain and Discharge	13	55	23		31	8		8				8							8	85
1065	Lumbago. Myositis	3	100	67	33	100	33		67		67			33	33	33				100	33
581	Heart. Rheumatism	6	100	100	17	67	33	17	33	17	50			33	50					50	67
1049	Mild Rheumatism	4	0																		100
1057	Heart. Rheumatism. Emacia	6	83	50		33	33				17				17	17				33	67
1014	Neuritis. Myositis	10	20	30		80	50		60	20	30				10					20	30
1058	Heart	3	100	100	33	100		33	33		100			100	33					67	33
311	Heart. Rheumatism	2	100	100	50						50			50	50						50
1009	Heart. Arthritis	5	100	100		60			20		60				20				20	40	40
1048	Neuritis. Gall Stones	5	100		20	40	40	20	20	20	20				20	20	20	20	20	60	80
709	Arthritis	4	100	25		50	25		25		50			50	50	50		25	50	100	75
455	Rheumatism	9	78	22		11								22		11				78	100
1008	Eyes	13	62	38	8	54	31	23	69	15	54	8	15	23	31		8	8	62	8	23
1019	Diarrhea. Lameness in Shoulder and Arm	4	100			75	50	25	100			25			25	50				75	100
1005	Rheumatism	3	100																	100	100
987	Eyes. Rheumatism	12	83	58	25	42					33	33	25	33	25	17		8	58	50	58
955	Xerostomia. Rheumatism	12	83	33	42	33					33	8	8	17	8	83				8	67
1024	Rheumatism	10	60	30	30	30		20			40			20	30	10				50	60
433	Rheumatism	2	50	100	50	50		50			100			50	50	50				50	0
433	None	2	None																		100
938	None	4	None																		100
	None	1	None																		100

FIGURE 143.

tions, we have found many instances where it seemed that the explanation might be found in the methods of procedure, including the length of time the organisms had grown on artificial medium and the nature of that medium. For example, some men plate out the organisms on Petri dishes, pick off a colony in 24 or 48 hours, plate it on another Petri dish and when it has grown sufficiently to plant others, transfer again until finally enough dishes are covered, from which to wash the organisms for inoculation, which may take several days. In some cases, with which we are familiar, the organisms were grown in artificial media for five days, and in other cases as long as seven days. This is so important a factor, that it might well be used as an experiment to determine whether or not growing the organisms on artificial media, would efficiently eliminate elective localization qualities, if they existed.

In studying our large groups of inoculations, we find very distinct evidence that some types of lesions produce very large percentages of localizations whereas others do not. We have also found that different types of the same lesion will produce in some instances very large percentages of localizations, while others will produce a low percentage. In Figure 143 we show the result of inoculating 187 rabbits with cultures from the teeth of thirty different patients. This group represents a quite large variety of conditions, some acute, some chronic, some without lesions. The result of this study has shown that, in a large number of instances where the process was an acute one, the method of inoculation used showed evidence of elective localization in a high percentage of cases, often 100 per cent; that where the process was a subacute or chronic one, it was often a low percentage of localization; and when there was no lesion, there was often a very indefinite expression in the rabbits or no expression. (See the last three.)

There are many factors involved which modify the final results as, for example, the relation of the size of the dose to the weight of the animal. Many experiments are recorded in these various studies which illustrate this, though this mass action relation has not always been the purpose of the experiment. A splendid illustration of this phenomenon is shown in Figure 144 by inoculating three rabbits, Nos. 571, 572, and 573, with the same quantity of the same strain. All three were inoculated intravenously September 13 with 1 cc. of culture from four incisors, grown out 24 hrs. in plain broth; organisms washed and suspended in sodium

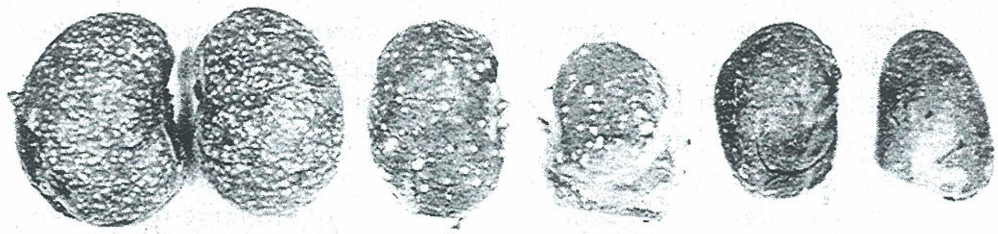

FIGURE 144. MULTIPLE KIDNEY ABSCESSES PRODUCED IN THREE RABBITS. ALL RECEIVED THE SAME CULTURE AND THE SAME TIME FACTORS. RESULTS ARE INVERSELY WITH THE WEIGHTS OF THE RABBITS.

chloride solution; three billion to 1 cc. All were chloroformed September 19. Since all were posted after the same length of time the results are comparable. This culture had a marked affinity for kidney tissue, and the figure shows the three pairs of kidneys from these rabbits, mottled with multiple abscesses; the smallest rabbit almost completely covered with abscesses; the next larger, many less; and the largest rabbit, relatively only a few visible on the surfaces; yet the elective localization was clear in them all.

This quality of elective localization is an exceedingly variable one. In some cases large percentages of the rabbits will have principally or exclusively one lesion, which may or may not be the lesion from which that individual is suffering at the time. This is illustrated in the chart in Figure No. 145 in an analysis of the lesions of the different rabbits inoculated with the cultures from a series of patients with involvements of joints and muscles. We could pick many dozens of cases, if we simply wished to present positive evidence, in which from 75 to 100 per cent of the rabbits had developed joint and muscle involvements. This group is selected to show that it is not always so.

The first patient was suffering from an arthritis, acute and chronic. Four rabbits were inoculated. All developed acute rheumatism; but in addition, one developed heart, two liver, one gall-bladder, one intestines, two kidneys, and one had a brain lesion.

The second patient suffered acutely from rheumatism. Nine rabbits were inoculated. Seven rabbits developed acute rheumatism; one developed liver involvement, and two heart. A striking thing about this series was, that every rabbit was chloroformed after it had developed acute lameness, such as carrying its leg,

JOINTS AND MUSCLES

R. No.	Material	Date Injected	Date of Death	Case No. and Lesions	Percentage with Major Lesions	Heart	Lungs	Liver	Gall-bladder	Stomach	Intestines	Appendix	Kidneys	Urinary bladder	Generative organs	Joints	Muscles	Rheumatism Clinical	Nerve trunks	Spinal cord	Brain	Eyes	Chloroform
194	C	2–18	2–22	709	100			x	x		x		x				x*	x*			x	?	x
196	C	2–18	3–2	Arthritis,				x					x			x*	x*	x*				?	
215	C	3–5	5–2	acute and		x												x*					x
217	C	3–5	5–2	chronic												x*		x*					x
				4 Rabbits		25	0	50	25	0	25	0	50	0	0	50	50	100	0	0	25		75
182	C	2–11	5–2	455	78			x										x*					x
183	C	2–11	3–15	Rheumatism												x*		x*					x
185	C	2–11	2–27															x*					x
186	C	2–11	3–9															x*					x
192	C	2–16	3–9															x*					x
195	C	2–16	2–18			x																	x
202	C	2–18	5–2																				x
345	C	5–9	6–17															x*					x
346	C	5–9	6–17			x										x*		x*					x
				9 Rabbits		22	0	11	0	0	.0	0	0	0	0	22	0	78	0	0	0	0	100
257	C	3–24	3–25	1065 Myositis.	100			x									x*	x*					x
260	C	3–24	4–17	Neuritis		x		x	x		x		x			x*		x*					
262	C	3–25	4–15	Lumbago		x	x	x			x		x					x*					
				3 Rabbits		67	33	100	33	0	67	0	67	0	0	33	33	100	0	0	0	0	33
416	W	6–14	7–13	1065 Myositis.	0	x		x															x
423	C	6–15	7–13	Neuritis				x															
424	C	6–15	6–23	Lumbago				x															x
				3 Rabbits		33	0	100	0		0	0	0	0	0	0	0	0	0	0	0	0	67
184	W	2–18	3–2	1024	60		x	x		x													
199	C	2–22	2–28	Rheumatism		x	x									x*	x*	x*					x
203	C	2–24	3–1			x	x	x		x							x*	x*					x
216	C	3–5	3–28																				x
218	C	3–5	3–28													x*		x*					x
219	W	3–7	3–21			x							x					x*					
221	C	3–8	3–28										x										x
222	C	3–8	3–28															x*					x
343	C	5–6	5–22					x					x				x*						
344	C	5–6	5–7										x										
				10 Rabbits		30	30	30	0	20	0	0	40	0	0	20	30	50	0	0	0	0	60
				Average	68	35	13	58	12	4	18	0	31	0	0	25	23	66	0	0	5	0	67

FIGURE 145.

which process subsided; and at the time of the postmortem, in only two of these rabbits was there still evidence of the lesion which had expressed itself so acutely, usually about two days after inoculation. In the previous case of the four rabbits developing acute rheumatism, only two showed involvement of the joints at the time of postmortem, and two involvements of the muscles. In the fourth one the involved tissue had apparently entirely recovered.

The next two cases are the same patient at two different times. Her disturbance was recorded as myositis, neuritis, and lumbago. At the time of the inoculation of the first group of rabbits, all three developed acute rheumatism; but, in addition, two developed heart, one lung, three liver, one gall-bladder, two intestines, and two kidney. Later, after her acute process had subsided, another group of rabbits was inoculated with the culture from another tooth, at which time none developed rheumatism, all three developed liver involvement, and that acutely; and with the exception of one rabbit which had a severe heart lesion, there were no other lesions in any of the three rabbits. Note that in both series of inoculations from this patient all of the rabbits developed lesions of the liver, which lesion appeared in only one rabbit out of nine in the preceding case.

In the last case the patient suffered from acute rheumatism. Ten rabbits were inoculated. Five of the ten developed clinical rheumatism and one myositis, making six rabbits with joint and muscle involvement. Three had heart involvement, three lungs, three liver, two with stomach, and four with kidney involvements. These rabbits were inoculated with the culture from the teeth at the time the patient was having acute rheumatism and from which she had been suffering severely for some time. Incidentally, she has not had a recurrence of the disturbance since the removal of the infected teeth three years ago.

In this case the first and sixth rabbit in the list were inoculated with washing from the teeth, followed by a culture from the teeth two weeks later. This will be discussed again under anaphylaxis. The effect of the previous injection with the filtered washing was to make these rabbits much more sensitive to the culture grown from those teeth. One of these rabbits died three hours following this injection, notwithstanding the fact that it was the first time organisms had been injected into this rabbit, and the dose was not large enough to produce serious or immediate disturbance. The other died on the second day.

EYES

Rabbit No.	Material	Date Injected	Date of Death	Case No. and Lesions	Case No.	Percentage With Major Lesions	Heart	Lungs	Liver	Gall Bladder	Stomach	Intestines	Appendix	Kidney	Urinary Bladder	Generative Organs	Joints	Muscles	Rheumatism Clinical	Nerve Trunks	Spinal Cord	Brain	Eyes	Chloroform
169	W	1–10	2–24	1008	1008	62						x			x								x*	
173	W	1–12	6–10	Eyes															x				x*	x
174	C	1–13	1–15									x							x				x*	
207	W	2–24	3–10																				x*	
209	W	2–24	3–14						x			x		x				x					x*	x
263	C	3–30	3–31				x		x		x	x		x										
264	C	3–30	4–21				x	x	x	x		x	x	x		x							x*	
267	C	3–31	3–31				x		x		x	x		x										
276	C	4–4	4–27						x	x		x	x	x				x						
287	W	4–12	6–6				x										x	x						
290	W	4–12	4–27						x	x	x	x		x		x		x	x		x	x		
296	C	4–13	5–11				x		x	x		x		x			x						x*	
298	C	4–13	6–8														x						x*	x
				13 Rabbits			38	8	54	31	23	69	15	54	8	15	23	31	23	0	8	8	62	23
369	C	5–15	6–25	1087	1087	100	x		x								x						x*	x
371	C	5–15	5–25	Eyes				x	x	x		x	x	x				x					x*	
374	C	5–15	7–6	Nervous system.																			x*	x
375	C	5–15	6–23	Mental Cloud																			x*	x
383	C	5–24	6–23																				x*	x
384	C	5–24	6–6																				x*	
402	C	6–7	7–6																				x*	x
403	C	6–7	7–6																				x*	x
410	C	6–14	7–13																				x*	x
418	C	6–14	7–1						x									x			x*		x*	x
419	C	6–15	7–13																				x*	x
				11 Rabbits			9	9	27	9	0	9	9	9	0	0	9	18	0	0	9	0	100	82
143	C	12-28	1–5	987	987	83	x	x	x														x*	
151	C	1–3	1–16	Eyes			x	x	x					x	x	x								
155	C	1–5	1–22	Rheumatism												x	x*		x*				x*	
156	C	1–5	1–5											x										
157	C	1–5	1–18				x							x					x*					x
158	C	1–4	6–20														x*							x
160	C	1–7	1–18				x		x						x				x*					x
161	C	1–7	2–19						x									x*	x*				x*	
162	C	1–7	2–26					x	x									x*	x*			x	x*	x
163	C	1–7	1–14				x																x*	x
164	C	1–7	1–20				x							x	x	x	x*	x*	x*				x*	x
165	C	1–7	1–19				x								x		x*						x*	x
				12 Rabbits			58	25	42	0	0	0	0	33	33	25	33	25	50	0	0	8	58	58
				Average		82	35	14	41	13	8	26	8	32	14	13	22	25	24	0	6	5	73	54

FIGURE 146.

Few tissues have shown a higher percentage of localization than eyes when the process is an acute one. It does seem necessary, however, that the culture be taken from the tooth at a time when the patient's eye is in a state of acute reaction. This is illustrated in Figure 146 on Eyes, which shows three cases.

In the first, thirteen rabbits were inoculated from the cultures of the teeth of a patient with two types of lesions of the eyes: exophthalmos and extreme pain from the rupturing of blood vessels. This case is shown in Chapters 65 and 66. Of the thirteen rabbits 62 per cent showed eye involvement, 69 per cent showed intestines and digestive tract involvement, and many other severe lesions are recorded.

In the next case, eleven rabbits were inoculated and all developed acute eye involvements. This patient was almost totally blind in one eye and about four-fifths blind in the remaining eye. Note: With the exception of one rabbit which developed multiple lesions, most of the other rabbits developed only eye involvement.

The next patient had acute recurring involvement of both eyes, as part of a blood stream infection complication. Of the twelve rabbits, seven or 58 per cent developed eye involvements; 50 per cent developed rheumatism, from which the patient was suffering severely, and 58 per cent developed heart involvement, from which the patient also suffered.

A group of inoculations selected from patients with acute digestive tract disturbance, shown in Figure 147, will illustrate the variableness of this quality of elective localization of the organism growing in the teeth, where the patient is suffering from acute or chronic processes.

In the first case four rabbits were inoculated and all four developed acute intestinal involvement. The patient suffered from a diarrhea with movements every fifteen minutes. All of these rabbits developed diarrhea with this strain. All four were acutely involved.

In the next case the patient suffered from acute digestive tract disturbance. Of the six rabbits, three developed involvement of the stomach and intestines, one also of the gall-bladder and liver.

An effort was made to extract a toxin by passing the cultures in their media through Berkefeld filters. The filtrate, bacteria-free, was inoculated, and produced in nine rabbits, or 44 per cent, in-

DIGESTIVE TRACT

R. No.	Material	Date Injected	Date of Death	Case No. and Lesions	Case No	Percentage with Major Lesions	Heart	Lungs	Liver	Gall-bladder	Stomach	Intestines	Appendix	Kidneys	Urinary Bladder	Generative Organs	Joints	Muscles	Rheumatism Clinical	Nerve Trunks	Spinal Cord	Brain	Eyes	Lassitude	Chloroform
187	A	2–15	2–21	1019 Diarrhea. Lameness in right shoulder and arm. Lassitude	1019	100			x			x*			x			x*	x*						x
189	C	2–16	3–28						x	x		x*							x*					x*	x
190	C	2–16	3–28									x*													x
200	A	2–22	2–28						x	x	x	x*							x*					x*	x
				4 Rabbits			0	0	75	50	25	100	0	0	25	0	0	25	75	0	0	0	0	75	100
319	A	4–28	6–9	1098 Digestive tract Lassitude	1098	83																			
341	A	5–9	5–26				x	x	x*	x*		x*												x*	x
347	A	5–9	6–9				x					x*		x				x						x*	
349	A	5–9	5–17				x	x	x*		x*			x				x						x*	x
362	C	5–11	6–9				x							x											
363	C	5–11	5–21															x						x*	x
				6 Rabbits			67	33	33	17	17	33	0	50	0	0	0	50	0	0	0	0	0	67	50
231		3–16	3–29	Stock Toxin					x			x													
232		3–16	3–17																						
233		3–16	3–22				x																		x
239		3–18	6–24															x						x	x
240		3–18	4–3				x		x			x		x					x					x	x
252		3–23	4–1						x	x				x					x					x	
253		3–23	5–8				x		x			x						x	x						
254		3–23	4–1						x	x				x										x	
255		3–23	4–4						x			x		x				x						x	
				9 Rabbits			33	0	67	22	0	44	0	44	0	0	0	33	33	0	0	0	0	56	33
229	W	3–15	5–13	1057 Digestive Tract Heart. Rheumatism Acidosis	1057	83																			
241	C	3–18	5–13																						x
243	C	3–18	5–13																x*					x	x
256	W	3–28	4–15				x*												x*					x	x
259	C	3–29	4–10				x*		x*	x*				x				x*							
261	C	3–29	4–6				x*		x*	x*														x	x
				6 Rabbits			50	0	33	33	0	0	0	17	0	0	0	17	33	0	0	0	0	50	67
				Average			38	8	52	31	11	44	0	28	6	0	0	31	37	0	0	0	0	62	63

C Culture
A Aspiration from cyst
W Washing

FIGURE 147.

testinal disturbances, liver involvements in 67 per cent, heart 33 per cent. This is discussed in detail in another chapter.

In the last case the patient suffered from a very acute digestive tract disturbance. Six rabbits were inoculated. None developed lesions in either stomach or intestines, three developed lesions in heart, two in liver, two in gall-bladder, three myositis and clinical rheumatism. This patient's primary condition was an acidosis, of which he died subsequently, but which had been apparently overlooked in his previous symptomatic treatment. His digestive disturbance, like several of his other expressions, was entirely secondary.

An analysis of one thousand rabbits, inoculated with cultures from dental sources of various patients, reveals some very interesting data in relation to the generative organs, as shown in Figure 148. It is very striking that in this large number of rabbits, approximately half of each sex, exceedingly few instances of involvement of the ovaries, tubes, and uterus of females, or testicles of males, have occurred where the patient from whom the culture was taken did not have acute infection or evidence of pathological condition of the generative organs.

The first and third cases in this chart are the same individual whose case is described in Chapter 62. In the first group of six rabbits there were four females and two males. This patient was suffering from ovarian disturbance so acute that she was kept in bed nearly a week at the time of her periods and suffered exceedingly. She had been struck over her ovary in playing golf some years previously, since which time, she had always had severe distress at the time of periods. The four female rabbits all developed acute infection of the ovaries and tubes, as illustrated in the chapter referred to above. The two males showed no involvement of the generative organs. At the time of her first involvement, some seriously involved teeth were extracted, and a root resection was made of a central incisor, with the hope that it might be saved or made safe until the end of her college year. Her trouble, which had been relieved by the removal of the dental infections on the first occasion, recurred. This questionable tooth which had been resected was removed; the culture inoculated into three female rabbits, two of which developed acute infection of the ovaries and tubes. Incidentally, she has not had a moment's return of her trouble since the removal of the last of these infected teeth.

GENERATIVE ORGANS

R. No.	Material	Date Injected	Date of Death	Case No. and Lesions	Case No.	Percentage with Major Lesions	Heart	Lungs	Liver	Gall-bladder	Stomach	Intestines	Appendix	Kidneys	Urinary bladder	Generative organs	Joints	Muscles	Clinical Rheumatism	Nerve trunks	Spinal cord	Brain	Eyes	Lassitude	Chloroform
277	C	4–6	4–28	1085	1085	100F 67T																			
280	W	4–5	5–4	Ovarian Pain					x				x	x	x	x*									x
282	C	4–6	4–26	Headache			x		x			x		x	x	x*									x
283	C	4–6	4–19	Backache					x			x				x*									x
335	C	5–5	5–16	Lassitude			x		x			x				x*									
339	C	5–6	5–9						x	x		x		x										x*	
				6 Rabbits			33	0	83	17	0	67	17	50	33	67	0	0	0	0	0	0	0	17	50
337	C	5–6	6–14	1124	1124	100										x*									x
409	C	6–22	6–24	Pelvic inflamma-															x*						x
432	W	6–21	7–14	tion and dis-																				x*	x
433	W	6–21	7–4	charge. Neuritis			x	x				x		x			x*								
440	C	6–22	6–25	Rheumatism			x									x*	x*								x
441	C	6–22	7–14	Lassitude					x								x*	x*							x
442	C	6–22	7–14	Headache			x										x*							x*	x
				7 Rabbits			43	14	14	0	0	14	0	14	0	28	57	14	14	0	0	0	0	28	86
506	C	7–29	8–2	1085	1085	67			x																
507	C	7–29	8–2	Ovarian pain												x*								x*	x
508	C	7–29	8–2	Lassitude												x*								x*	x
				3 Rabbits			0	0	33	0	0	0	0	0	0	67	0	0	0	0	0	0	0	67	67
				Average		89	25	5	43	6	0	27	6	21	11	54	19	5	5	0	0	0	0	37	68

x—Rabbit
*—Patient

FIGURE 148.

When patients are suffering from endocarditis in its acute form, cultures from the infected teeth tend to reproduce heart involvements in animals in a very high percentage. This is illustrated in Figure 149 on hearts. In the first patient shown, five rabbits were inoculated, all developing acute heart involvement. In the second case, that of a child bedridden with acute endocarditis, three rabbits were inoculated with culture from a deciduous tooth. All three developed acute heart involvements. In the third case the patient was a woman between thirty and forty years of age with acute heart involvement and dyspnea. Six rabbits were inoculated. All developed acute heart involvement. In the fourth and last case, the patient had been bedridden for many months from acute heart involvement. Two rabbits were inoculated. Both developed acute heart involvement. These sixteen rabbits inoculated with cultures from four patients with heart involvement all developed acute heart involvement.

Other instances which illustrate the nature of the quality that organisms possess, of electing the same tissues that are involved in the patient, are shown in practically all the chapters from 59 to 69 in which are discussed the systemic expressions of dental infections, and in many of which, illustrations are produced of involvements in the animals.

We have in the preceding paragraphs of this chapter discussed the quality of the organism to elect a certain type of tissue. We wish now to discuss the quality of defense and susceptibility of the various organs and tissues of the body in relation to invading organisms. Few of our problems have been so difficult of approach as the study of the nature of these forces which decide what tissues will be selected, and why, in cases of dental and other focal infections. It is only recently recognized, that focal infections may express themselves in other and various tissues of the body. We will approach this subject first by a clinical analysis of the various cases presenting, to determine, if possible, whether there is any inherent force in the individual, or whether it is in the bacterium. Problems 2 and 4, discussed in Chapters 2 and 4, include a study of the nature of invading organisms and susceptibility. In these we found that an analysis of many hundreds of people suggests a classification into three fundamental groups: Those with absent susceptibility, those with acquired susceptibility, and those with an inherited susceptibility, with some tendency to combinations of these types. These also showed that in

HEARTS

R. No.	Material	Date Injected	Date of Death	Case No. and Lesions	Case No.	Percentage with Major Lesions	Heart	Lungs	Liver	Gall Bladder	Stomach	Intestines	Appendix	Kidneys	Urinary Bladder	Generative Organs	Joints	Muscles	Rheumatism Clinical	Nerve Trunks	Spinal Cord	Brain	Eyes	Chloroform
205	C	2-24	6-1	1009	1009	100	x*		x					x					x*					x
220	C	3-8	3-20	Heart			x*											x*	x*				?	
230	W	3-15	3-29	Arthritis			x*																	x
236	C	3-18	4-22				x*		x					x										
237	C	3-18	4-19				x*		x			x		x					x*					
				5 Rabbits			100	0	60	0	0	20	0	60	0	0	0	20	60	0	0	0	20	40
211	C	3-4	3-24	1058	1058	100	x*		x					x			x		x					x
212	C	3-4	3-17	Heart			x*	?	x		x			x			x		x					
238	C	3-18	5-3				x*		x			x		x			x	x						
				3 Rabbits			100	33	100	0	33	33	0	100	0	0	100	33	67	0	0	0	0	33
242	W	3-18	5-24	581	581	100	x*																	x
244	C	3-19	3-29	Heart			x*		x	x		x		x			x*	x*	x*					
251	W	3-23	5-24	Rheumatism			x*											x*	x*					x
269	W	3-31	5-24				x*		x										x*					x
270	C	4-2	4-25				x*	x	x		x	x	x	x				x*	x*					
271	C	4-2	4-7				x*		x	x				x			x*		x*					x
				6 Rabbits			100	17	67	33	17	33	17	50	0	0	33	50	83	0	0	0	0	67
206	C	3-4	6-3	311	311	100	x*												x*					x
223	C	3-9	5-21	Heart			x*	x						x			x*	x*						
				Rheumatism																				
2 Rabbits				Neuritis			100	50	0	0	0	0	0	50	0	0	50	50	50	0	0	0	0	50
Average						100	100	25	57	8	13	22	4	65	0	0	46	39	65	50	0	0	5	48

FIGURE 149.

cases of acquired susceptibility, a large percentage of the lesions were in the nervous system; and in inherited susceptibility, there were both the presence of a lowered defense to streptococcal infection, and a distinct tendency to similar localization in different members of the same family to similar tissue and organ localization.

It is a matter of very great importance whether defense is a purely general quality in which the tissue selected in elective localization is purely accidental selection, or whether that tissue which breaks in a given individual has a predisposition, rather than a predilection on the part of the organism, for that tissue. To determine this, we have analyzed a large number of susceptibility records and case and family records. One of these studies is shown in Figure 150 under the title of "Organ Susceptibility" in which ten patients and their families are used in each group. We have taken ten typical patients each with rheumatism, heart involvements, nerve tissue involvements, and internal organ involvements. In the ten families in which the patients were suffering from rheumatism, we find that the total number of cases of rheumatism in all the ten families is 59. This includes the ten patients, their parents, the brothers and sisters of their parents, the patients' brothers and sisters, and the patients' grandparents, but not the patients' children, since the other parent would furnish half of the influence. The number of cases of heart involvement in all the members of these ten families combined is 7, nerve involvements 19, internal organ involvements 19.

ORGAN SUSCEPTIBILITY

Ten Patients and Families in each Group

	Total No. of These Lesions in Family.				No. of Patients Affected with Lesions			
	Rheumatism	Heart	Nerves	Internal Organs	Rheumatism	Heart	Nerves	Internal Organs
Rheumatism	**59**	7	19	19	**10**	2	4	4
Heart	24	**57**	25	13	6	**10**	7	3
Nerves	15	9	**142**	28	6	2	**10**	7
Internal Organs	13	9	30	**90**	4	0	9	**10**

FIGURE 150.

In the next group, ten patients with heart involvement, the total number of cases of heart involvement in the ten families was 57, of rheumatism 24, nerve tissues 25, internal organs 13. In the ten patients with involvements of the nervous system, the total number of nerve lesions in the ten families was 142, rheumatism 15, heart 9, internal organs 28. In the ten patients with affections of the internal organs, the number of cases of serious involvement of internal organs in all the members of the ten families was 90, rheumatism 13, heart 9, nerves 30.

The second part of this chart shows a similar study limited to the ten patients, not including the various members of their families. In the group of ten with rheumatism there were two cases of break in the heart, four of the nervous system, and four of internal organs. In the group of ten patients with heart involvement six had rheumatism, seven nerve involvements, and three internal organ involvements. In the ten patients with nerve involvements, six had rheumatism, two heart involvements, seven internal organ involvements. In the ten patients with internal organ involvements, four had rheumatism, none had heart involvements, and nine had nerve tissue involvements. It should be noted in passing (and I have discussed it in other chapters), the important relationship between involvements of the nervous system and internal organs.

In Chapter 2, I have discussed the behavior of organisms and have found that they tend very strongly to acquire qualities in accordance with the culture medium; that they do not have physical expression or elective localization in accordance with their biologic classification as members of the streptococcus group. An analysis of the data in Figure 41 of Chapter 4 and also in Figures 42, 43, and 44 of the same chapter, reveals that with increased susceptibility, there is a marked increase in the number of breaks of the rheumatic group, but that these tend to appear limited to certain tissues in any given individual, but that these tissues differ widely in different susceptible individuals, but in accordance with a common tendency within that family. The constantly heard reference to certain rheumatic lesions, such as that rheumatism and heart involvement prevail in certain families, is borne out in every phase of our study of the more exact relationships in this matter of susceptibility.

In Chapter 4, we recorded that in 681 individuals (being the cases selected from 1400 cases) studied with their families, those cases whose records were considered sufficiently complete to justify comparisons and deductions, all others being rejected, there were 940 cases of heart; and that over half of these appeared in

PROGRESSIVE TISSUE AND OR

Susceptibility Group	Average Age	No. of Males	No. of Females	No. of Patients Having Lesions In: Tonsils	Rheumatism	Heart	Neck	Nerves	Internal Organs	Special Tissues	No. of Patients Having Relatives Affected: Brothers	Sisters	Father	Father's Relatives	Mother	Mother's Relatives
Absent	40.7	8	7	4	2		2	2	1	1			4	1	5	3
Acquired	47.9	6	9	6	7	3	8	13	8	8	4	1	2		2	
Inherited 1 side mild	43.4	5	10	8	14	2	9	12	7	7	5	7	9	6	10	9
Inherited 2 sides mild	40.9	3	12	9	13	6	11	12	10	11	9	11	12	7	14	11
Inherited 1 side strong	39.4	4	11	7	14	4	12	15	10	13	5	6	10	6	12	10
Inherited 2 sides strong	33.9	1	14	12	11	12	14	15	14	13	12	11	15	15	15	14

FIGURE 151.

100 families, while over half of the families or groups contained no heart involvement. Figure 151 shows the progressive nature of this quality with the factor of intensity of heredity in a series of groups of individuals beginning with those without susceptibility or those with absent susceptibility, going through acquired susceptibility, to those with strongly inherited susceptibility. From these data, we find strong evidence that the quality of susceptibility, when it obtains as a part of inheritance, tends strongly to be a factor of the organ rather than of the whole system, in which latter case, granting an inherited susceptibility to the rheumatic group lesions, it might be expected to attack organs and tissues entirely at random, in which case the percentage of incidence for given tissues and organs, while they might vary through a wide range in various families, would tend to average similarly in different groups.

In Chapter 21, Influences which Modify Defensive Factors, we found that many forms of overload contributed to causing a break in a particular tissue. We can, accordingly, understand how a nervous system that is overloaded might tend to break; similarly, a heart that is exhausted. It is difficult on such a basis to account for the frequent occurrence of localization in a tissue of the animal corresponding with the tissue that was removed from the patient, but removed because of infection and disease. To illustrate:

A case of an unmarried woman about forty-six with marked

GAN INVOLVEMENT OF GROUPS

Total No of Lesions per Group in:							Total No. of Lesions in Group		Average No. of Lesions per Family		No. of Patients Having:					
Tonsils	Rheumatism	Heart	Neck	Nerves	Internal organs	Special Tissues	Severe	Severe & Mild	Severe	Severe & Mild	Caries	Periodontoclasia	Open	Locked	Rarefying	Condensing
4	7	2	2	7	7	2	16	31	1.07	2.07	6	6	6	9	10	0
6	11	4	9	37	14	15	63	96	4.2	6.4	12	5	5	13	5	3
12	45	13	18	66	28	19	144	201	9.6	13.4	10	5	6	13	5	3
21	61	29	19	92	56	30	227	308	15.3	20.53	14	3	4	12	6	5
16	51	30	27	105	78	31	258	338	17.2	22.53	12	3	3	12	4	5
47	106	77	50	254	106	114	483	754	32.2	50.27	14			13	1	10

FIGURE 151—CONTINUED.

involvement of the cervical and dorsal plexuses (involvement of the neck and upper part of spine) was under study. It was decided that the teeth were involved. They were removed and cultured, and animals were inoculated, with the result that several female rabbits developed acute infective processes of the ovaries, tubes, and uterus. This patient had had first one ovary and tube removed, and later the other ovary, tube, and uterus removed because of acute involvement.

In the chapter on Primary and Secondary Sex Organs, we discuss a number of cases where the tissue affinity involves these organs, and make comparisons with the percentage of incidence of affection of these tissues where the patient was not affected, which latter is almost zero.

This raises the important question as to whether that quality or property of the bacterium, which determines that it shall localize in some particular part of the animal into which it is injected (and which quality has in Chapter 2 been shown to be very transitory and easily lost) is derived from one of the three following factors: First, a condition obtaining in the focus, the tooth in this instance, tending to develop in the organism a quality for selecting this type of tissue; second, the fact that the organ is diseased furnishes to the organisms growing in the focus an appetite or affinity for that diseased tissue, through some substance passing to it through the culture medium, derived from the circu-

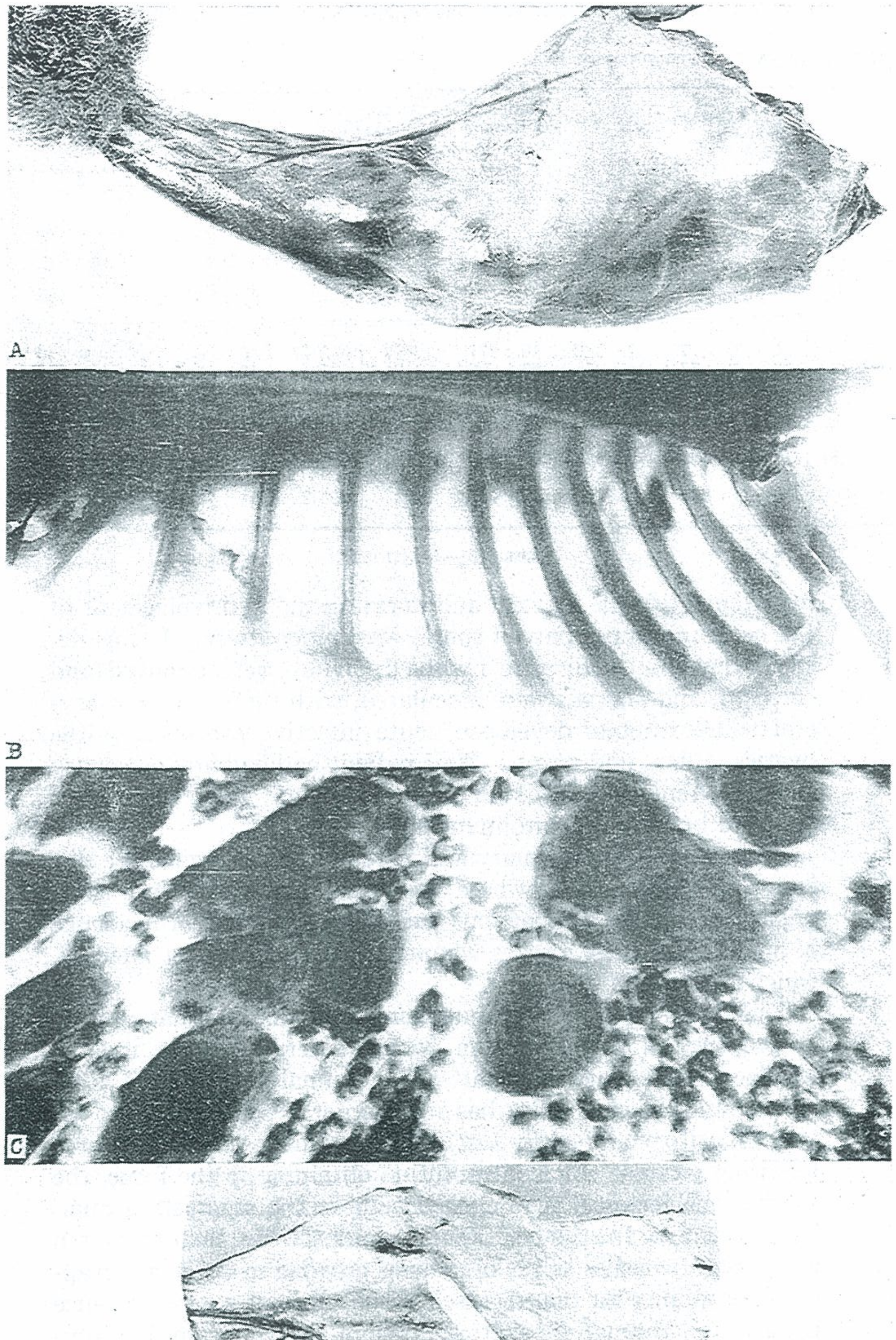

FIGURE 152. SPONTANEOUS HEMORRHAGES CAUSING DEATH IN TWELVE HOURS. A, THIGH; B, CHEST WALL; C, HEART MUSCLE; D, STOMACH LINING.

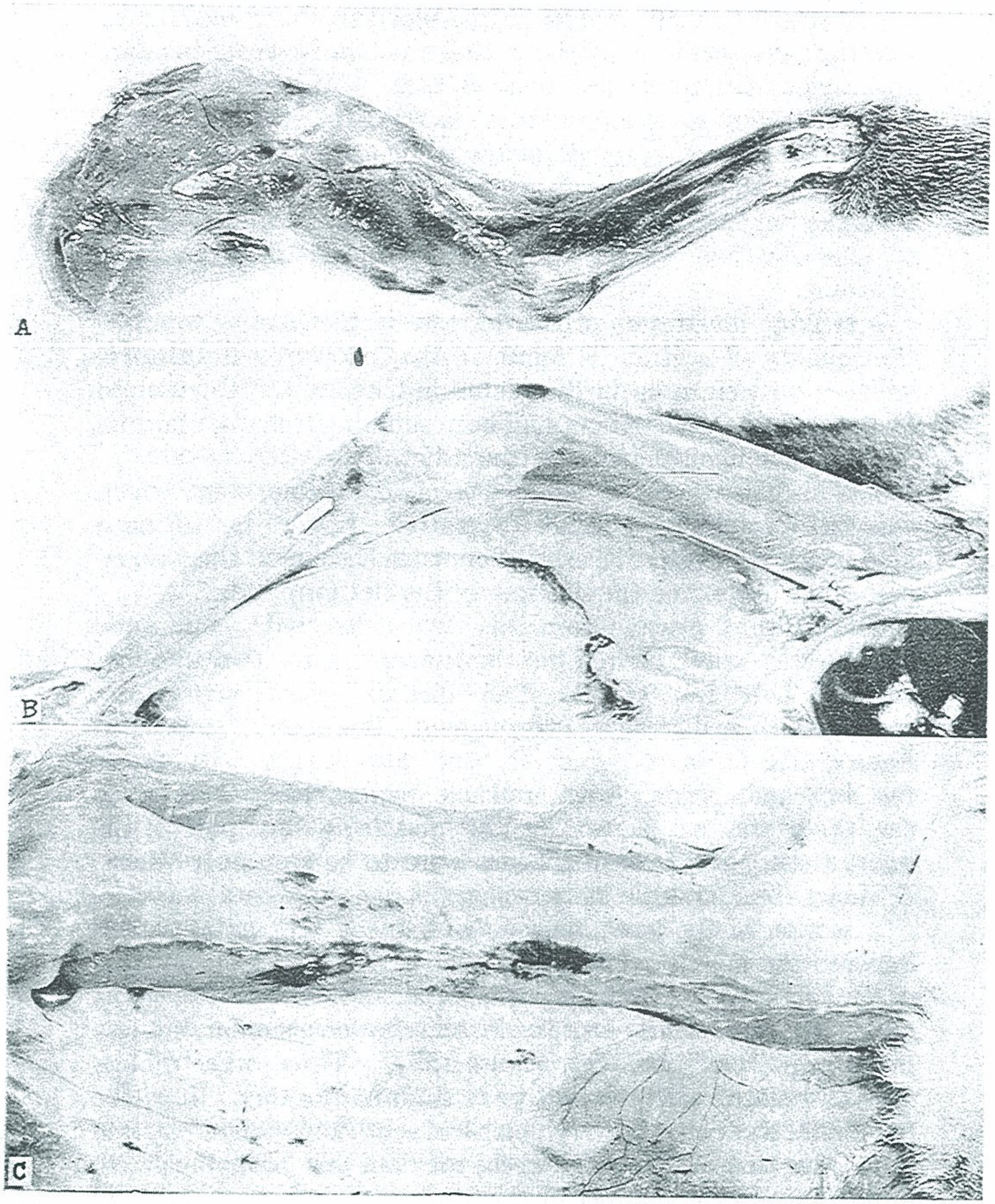

FIGURE 153. SECOND RABBIT WITH SAME CULTURE AS FIGURE 152. A, OTHER THIGH OF FIGURE 152; B AND C, ANOTHER RABBIT RECEIVING SAME CULTURE; B, HEMORRHAGE INTO KNEE; C, BACK MUSCLES.

lating blood and lymph; and third, whether normal defense provides in the blood stream a series of defensive factors suited to each and all of the various tissues of the body, the absence of any one of which tends to develop in the blood stream a medium so affecting the organism growing in the focus, that it tends to select that tissue when transferred to a new host. This has suggested a series of studies to determine these factors. In other chapters I discuss the general systemic factors which seem to relate quite similarly to all types of localization. These three problems are so fundamental and the researches that I have made upon them are so extensive that we will discuss them in the three chapters following.

A striking illustration of the decrease in the elective localization quality of a strain is found in the following case history, which is written up in further detail in Chapter 60 (Circulation Disturbances). The patient had been suffering from myocarditis and stomach involvement. Within a few hours after the extraction of his first two teeth, he had a secondary hemorrhage which was very profuse and difficult to control. In fact, he had been kept in the ward after the extraction, both because of the seriousness of his illness and the slowness of the clot formation.

The culture grown from his two extracted teeth, one an infected vital pulp, was inoculated into two rabbits. One of these rabbits (R. 1065) died in twelve hours from spontaneous hemorrhages throughout the body, shown in Figure 152-A, B, C, and D, and also 153-A. A shows the hip and thigh, with multiple hemorrhages; and when the thigh was cut across, or the muscle in any part of the body, these multiple hemorrhages were to be seen everywhere. B shows these multiple hemorrhages in the intercostal muscles; C a section of the heart muscle, with blood cells extravasated between the muscle cells; and D, a hemorrhage into the mucosa of the lining of the stomach. The other rabbit (No. 1064) was chloroformed in twenty-four hours, and showed a similar, but less pronounced, condition. See Figure 153-C. Three other rabbits were inoculated with this culture on the following day. It will be noted that these showed very much less acute and violent reaction than those that were inoculated the previous day, probably due to a difference in the number of generations of organisms that had developed in the new environment, the first generation of organisms always being the most virulent and specific. One of these (R. 1069) was chloroformed in four days and showed subcutaneous hemorrhages in the lumbar region and over the tibiae. This is shown in Figure 154, B and C. Another (R. 1068) showed

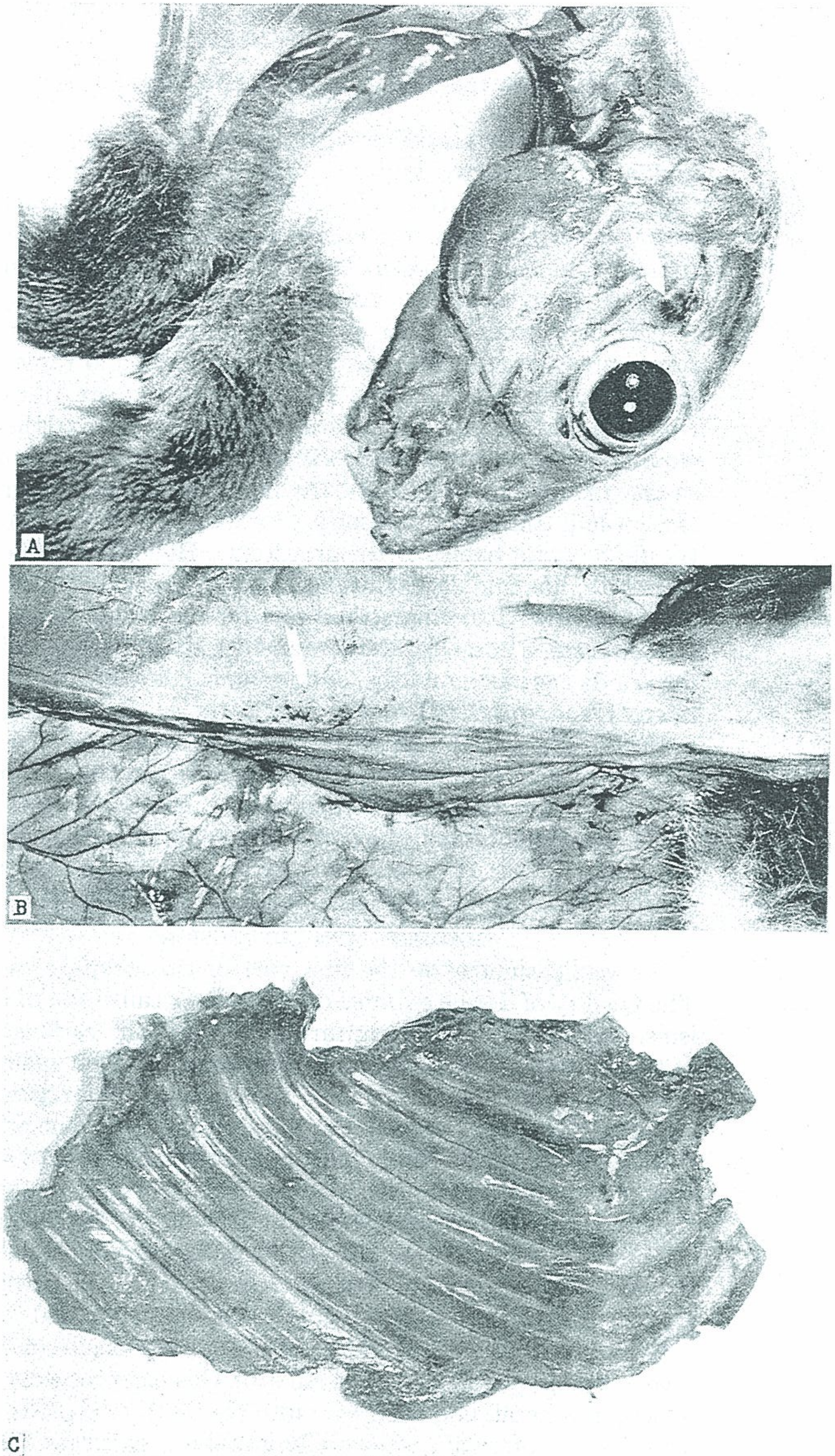

FIGURE 154. LESS SEVERE LESIONS PRODUCED BY LATER GENERATIONS OF SAME CULTURE. A, HEMORRHAGE FROM EYE; B, BACK MUSCLES; C, CHEST WALL.

hemorrhages in the muscle tissue of the lumbar region in the back and also the ventral region in the psoas muscle, shown in Figure 153-B. The third (R. 1067) showed a periosteal hemorrhage over the right eye, seen in Figure 154-A. Note that the patient's ailments had been diagnosed as involvements, primarily, of the heart and stomach with expressions in the nervous system.

It is very probable that every culture medium influences to some extent, and in most cases very markedly and rapidly, the elective localization qualities of organisms that are transferred from the host to such artificial media and there multiplied. We would not expect a child, that was transferred from the camp of a savage man-eating tribe to the environment of a modern civilization, would, through its posterity for many generations, exhibit the flesh eating habits of the ancestors. Biologically, the cycle of growth, life, and degeneration, is very comparable in all forms of life, whether it be fourscore years for the man, twenty years for the horse, a dozen for the cow and dog, half that many for the fowl, a day for many moths, and perhaps hours for many of the microörganic forms. However, just as the egg is a resting stage in the life of the fowl, just so the bacterium may take on a condition of suspended animation, or resting stage, in which it may or may not retain its aggressive qualities. In this substance with which we are concerned, the organisms apparently through their wonderful power of adaptation, quite rapidly change in the quality of elective localization and tissue affinity.

SUMMARY AND CONCLUSIONS.

We would summarize the first part of this chapter—namely, The Quality of Tissue Affinity or Elective Localization of Organisms—as follows: The organisms constituting the various strains of the streptococcus group are very different in their main characteristics from microörganisms that produce contagious and infectious diseases. These latter have, in the main, one principal type of expression. The organisms producing parotitis or mumps may also involve some other tissue as the testes, but in the main they tend to select parotid glands; and, similarly, Bacillus typhosus produces typhoid fever with its characteristic involvements of Peyer's patches, enlargement of the spleen and mesenteric glands, and catarrhal inflammation of the intestinal mucous membrane; and, similarly, diphtheria, measles, small-pox, scarlet fever. Unlike these the organisms of this streptococcus group which, in general, find their way into the teeth to establish there a habitat, are often, if not generally, relatively harmless, non-virulent strains. There is probably no one quality which makes this streptococcus so unique and characteristic, as its exceedingly great quality of adaptability to its environment. In Chapter 2 we have discussed this quality of adaptability from another phase and have found that, regardless of the biologic classification, the organism tends to pick out certain tissues of the body in accordance with forces, other than those which establish the biologic differentiations. We also found that they could grow and learn to thrive in the presence of poisons, which originally were so toxic to them as completely to inhibit their growth in one-tenth of the concentration on which they ultimately flourish. Our results of inoculating these strains which have grown for periods of time in infected non-vital parts of the teeth, and sometimes in the vital structures, show these organisms to have taken on qualities of invasiveness which the free strains of the mouth do not obtain and which, doubtless, this organism did not have when it resided in the mouth. We have also found, as shown in Figures 143, 144, etc., that these organisms come to take on a quality, which is, incidentally, very transient and easily lost, of selecting not only a special tissue, when inoculated into rabbits, but very often the same tissue as that from which the patient was experiencing a severe lesion, as heart or kidney, eyes, etc. We also found that this quality of elective localization seems directly related to an acute process; that patients, for example, with acute heart involvements, acute rheumatism, iritis, or retinitis, are more likely to have the organism taken from dental infections, exhibit elective localization qualities, than if the organism is taken from the teeth at a time when they do not have these acute processes.

With regard to the quality of varying defense of a given organ or tissue in different individuals, our studies have seemed to indicate that all organs and tissues tend to have, normally, a high defense for streptococcal involvements; that various types of overload make these special tissues especially susceptible; and also that there seems evidence of the presence or absence of a specific element of defense for individual tissues independent of other tissues of the body, and which defensive quality tends to be similar in different individuals of the same family. In other words, there is strong evidence that the quality of organ and tissue susceptibility follows the laws of mendelian factors. We would, therefore, briefly summarize results of this research as follows:

Dental infections may or may not contain organisms with a specific elective localization quality for certain tissues of the body. When they do so it is generally because the host is suffering, or has previously suffered, from an acute process in that tissue, which acute process frequently, entirely and permanently, disappears with the removal of the focus of infection. There is evidence to indicate that the complete removal of an organ so affected does not destroy that elective localization quality in the microörganism of the focus.

(Note: It should be noted that while I have discussed in the paragraphs of this chapter, the qualities of defense as expressed in organs and tissues, I do not imply, nor do I believe, that defense is limited to these structures. The researches on the nature and quality of the defensive forces in the circulating and other fluids of the body are discussed in other chapters).

CHAPTER XXIII.

THE ENVIRONMENT PROVIDED BY AN INFECTED PULPLESS TOOTH.

PROBLEM: What are the characteristics of the habitat and environment furnished for bacteria in an infected pulpless tooth?

EXPERIMENTAL AND DISCUSSION.

In studying this problem of the particular elements of environment which an infected pulpless tooth furnishes, we will review first some of the characteristics of the tissues involved. The dental pulp and tubuli are filled with cellular and protoplasmic structures such as are included in highly vascularized special tissues. These are contained within bony walls in the form of an almost infinite variety of labyrinths producing such a maze, that if all the tubuli of a single rooted tooth were connected end to end, the total length would constitute three miles of enclosed channels. When we realize the inaccessibility of a large part of this network of intercommunicating channels and canals, we understand why it is so difficult to sterilize a piece of infected dentin even when it is immersed in disinfectants. A streptococcus tends to select necrotic and degenerating tissue elements. Mechanically, a putrescent tooth furnishes a highly ideal hiding place for this germ. It can adapt itself to almost any shape of physical environment in that it may grow so small as to pass through a Berkefeld filter of such fine mesh, that all microscopically visible organisms will be taken out; and these minute organisms which have succeeded in passing through the meshes of this filter, may, on reaching other media, grow to a size that is relatively large for organisms of the cocci group.

Among Nature's most effective mechanisms of defense against this organism, are the phagocyting cells, chiefly the polymorphonuclear leucocytes. These cells have the wonderful property of being attracted to infective organisms, engulfing them, and neutralizing their toxic substance by the antitoxin within their own protoplasms. They may pass through cell walls into the interspaces by the wonderful mechanism of increased permeability of the capillaries in a state of capillary dilatation as a part of the inflammatory process, and after engulfing the toxic substance may retreat from the field of inflammation and pass again into the circulating blood, or be carried away through the lymphatics, chiefly the latter, or in case of an exit, such as an abscess, pass out through the discharge. They are attracted to pathogenic organisms by chemotropism, which is one of the marvels of the entire system of defense. They are completely baffled and helpless when it comes to the matter of entering an infected pulpless tooth to reach and destroy the organism producing the toxic substances, or completely to eradicate the nidus because of the physical environment.

It is difficult to conceive of a parallel where so complete a protection is furnished to an invading enemy parasite entirely within the group of unit cells which, otherwise, would maintain a defense against it; for while the organism is entirely protected from the defensive mechanisms of the body, only one of which are the leucocytes, there is no other source of exit for their poisons and fluids of the host, except as an exit may be possible into the oral cavity through the rcot of the tooth. In dental practice we undertake to change completely this environment by annihilating the inhabitants of this part of the non-vital tooth structure, assuming that the dentin is non-vital if the pulp is degenerated, and mechanically closing all entrance to this labyrinth and retaining it in a sweet and healthy condition as an inert, if not normally nourished, structure of the human body.

There are several phases of this problem involved in this study, one of which is the nature of the boundary surrounding the dentin of a tooth, particularly the dentino-cemental junction. There has been a great diversity of opinion as to whether or not there was direct communication between the dentin and cementum. (In another chapter I will discuss the matter of direct connection between the dentin and enamel). To determine this we have made a large number of histological sections, before speaking of which, however, we wish to refer to the excellent work of others, particularly that of Dr. Harold Box,[13] of Toronto. He seems to have demonstrated to his complete satisfaction the presence of communicating channels between the dentinal tubuli of the dentin and the canaliculi and the lacunae of the cementum. Marshall, on the other hand has furnished

13. See bibliography.

evidence which has seemed to demonstrate to him that the cementum is laid down upon the dentin as an entirely independent structure, completely separated from it. Mummery, Hopewell-Smith, Williams, Boedecker, and many others, have discussed the relationship of the dentin to the surrounding structures with especial consideration of the problem of nourishment of tooth structure. Our own studies indicate that, in general, the cemental border of the dentin and the dentinal border of the cementum constitute what is apparently the same tissue, though in fact like a common fire-wall built by two different contractors, with occasional openings for communicating channels extending from one side to the other, which openings, however, constitute a relatively small part of the total area in this common wall, and which openings seem generally, if not nearly universally, to be special channels extending from the pulpal tissue to the peridental membrane, and which may have little, if any, communication by anastomosis, with either the dentinal tubuli of the dentin or with the lacunae and canaliculi of the cementum. They are, in effect, either very minute or larger multiple foramina.

As one of several studies on this phase of the problem, I have placed metal tubes in the pulp chamber from the apex of the tooth, carefully cementing same, and have passed various fluids into the tooth under measured pressures. Several substances were tested, and it was found that both the sodium and the chlorine ions would pass from the pulp chamber of a freshly extracted tooth through both the dentin and cementum, and the dentin and enamel, in easily measurable quantities. Among the easily passed ions was that of the calcium.

When we study various tissues of the body, we find their function is determined by certain inherent qualities pertaining characteristically to their cells. For example, whereas the various soluble products that are found in urine are entirely inhibited from passing through the tissue constituting the bladder wall, that same fluid will pass with great freedom and rapidity through the wall of the small intestine; and similarly, the various glands and organs of the body have cells adapted to hold back certain substances and allow certain others to pass. In general, this problem is referred to as semipermeability, and it has to do with the function of every individual cell, as well as every type of cell, and is different in different tissues and cells. Electrolytes can pass through most membranes of the body; colloids through but few of them. Hence the colloids of the blood stream cannot pass through the blood vessel walls, not even through the capillary walls, though the electrolytes can. When the colloids, which are largely proteins, are split into the various amino acids, they may pass through tissue cells in accordance with the structure of that molecule and the function of the cell in question. Each cell, therefore, may be thought of as a lock which takes a certain type of key. The various molecules of various compounds can be thought of as the key, and only those fitting the particular combination in question, pass to that particular tissue. But electrolytes and certain of the amino acid groups and their derivatives, will pass through tissues which are impervious to colloids and to bacteria.

My studies of the dentino-cemental junction have led me to believe that certain substances can pass readily through this boundary, while the bacteria cannot. By placing organisms grown from infected teeth in sealed glass tubes, they very soon cease to multiply, and in a very short time nearly all are dead. Only a minute orifice is necessary to change quite completely the per cent of dead organisms. I have had sections cut from normal tooth structures, and have used these as semipermeable membranes to determine, if possible, whether or not food materials may pass through this structure while organisms may not, by connecting through glass tubes an infected and sterile culture medium, separated only by this partition of tooth structure, including the continuous wall of dentin and cementum cut from a suitable tooth. While these studies are not completed, the evidence available indicates that nutrient material can pass to the organisms through this structure, but that the organisms themselves cannot pass. If this be true, the tooth in all probability furnishes a protection for the organism, in that neither the leucocytes nor the defensive bactericidal elements of the blood can reach the organisms within the dentin. The organisms in that position may receive a continuous supply of nutriment through the semipermeable, but to them impenetrable wall, making the boundary to the dentin. They are here completely protected to produce a toxic substance which may pass to the host and continually irritate and injure the defensive forces of the host. If those defensive forces be sufficiently adequate to neutralize all toxic products immediately within the vicinity of the tooth, the warfare will be limited to that zone. This battle-ground and the battle are discussed in other chapters. If the products are not neutralized in the immediate vicinity of the tooth, because of the host's temporary or permanent inability to make that type of warfare, they may pass to the various organs and tissues of the body and irritate or sensitize them. The tooth, then, must be looked upon as an entirely different structure from flesh or living bone, since, if the pulp is dead, Nature has no mechanisms for disinfecting the dentin after it becomes infected, or of maintaining it if it is non-vital but sterile. She is compelled to make the warfare in the form of a quarantine about the tooth until such time as she can either absorb it or exfoliate it. A pulpless tooth is, therefore, a sequestrum; and if it becomes an infected sequestrum, must be dealt with as a foreign substance and not as a part of the host.

An important question has been the matter of transfer of fluid from the dentin to the cementum or the enamel, or through both of these from the pulp to the fluids surrounding the different structures of the tooth. Bunting and Rickert[14] have demonstrated the passage of fluids from the inside to the outside of a tooth.

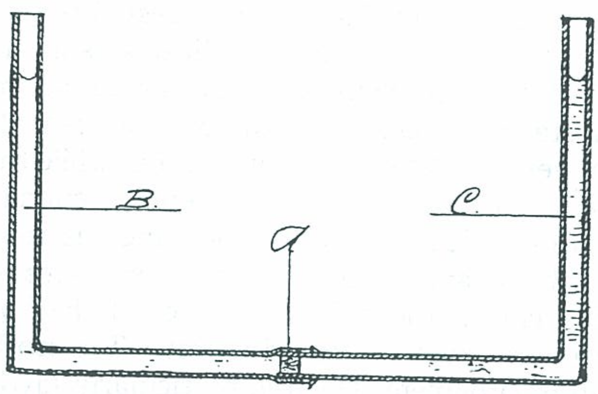

FIGURE 155. DIAGRAM OF THE APPARATUS WHICH USES A SECTION OF TOOTH AS A PERMEABLE MEMBRANE. A, TOOTH SECTION; B AND C, CONTAINERS FOR SOLUTIONS BEING TESTED.

Figure 155 shows the set-up of one of the experiments for determining the permeability of tooth substance for nutrient products in culture medium on one side of a sac of the tooth structure, and living organisms in a normal salt solution on the other. The organisms will not increase in number in normal salt solution to any very great extent. In the arm of the apparatus to the left, we have placed the sterile culture medium. If the organisms may pass through a sac of dentin and cementum, containing at least

[14] See bibliography.

one, and in some instances two, dentino-cemental borders, they can go from the salt solution to the culture medium. By culturing from time to time, as well as by its appearance, we can determine the development of organisms on the medium side. If there chances to be a multiple foramen, and some teeth have as high as seventy-five, they may pass through these and not have to pass the true dentino-cemental boundary. If the infected normal salt solution remains clear and if its total bacterial count per cubic millimeter does not increase, we can assume that organisms have not developed more rapidly than they have died off. If, on the other hand, the culture medium is able to pass to the salt solution, either in whole or only some of its contained products, the organisms may multiply. We have run several kinds of controls. All of these set-ups were autoclaved. Some have normal salt on both sides, one infected and the other not; some distilled water on one side and the other concentrated salt solution, to determine whether the tooth under these circumstances would act as a semipermeable membrane. Some contain electrolytes on one side and culture media on the other; others colloids.

SUMMARY AND CONCLUSIONS.

From the data here available it is suggested that the dentino-cemental border, under ordinary conditions, is an impenetrable barrier to bacteria; that while the organisms cannot pass through it, toxic substances produced by the bacteria can, the extent of which is not yet completely determined; that while the organisms cannot pass through this dentino-cemental boundary, nutrient substances from culture media surrounding the tooth can pass through this boundary. This boundary also forms a quite complete barrier against the defensive forces of the body, such as phagocyting leucocytes, bactericidins, etc. The tooth, therefore, furnishes an environment which is particularly favorable to the invading organism and unfavorable to the host, in that the former is protected from the aggressive defensive forces of the latter, while able, by the natural laws governing the behavior of liquids and gases, to secure a continuous supply of nutriment through the walls of the fort. The placing of an infected tooth beneath the skin of the rabbit is many times more dangerous to the rabbit than the introduction of that bulk of concentrated infected culture; and even though an infected tooth with apparently splendid root filling is thoroughly sterilized externally and placed beneath the skin of a rabbit, within a few days it shows evidence, by changes in the hematology and chemistry of the blood, of a progressive destructive change, which, in the majority of instances, terminates in death within two weeks. We would, therefore, change our conception of the significance of an infected tooth about as follows: from

> "(*Old interpretation*) *Since the presence or absence and the extent of danger of infection in a suspected tooth, are determined by the size of the pus sac at the end of the root, those teeth are dangerous which have this mass of infection external to the end of the root, and in proportion to its extent; and those are not dangerous which have little or no pus sac.*" to

> **Since an infected tooth is a fortress for bacteria within the tissues of the host, and since, in accordance with the laws governing the behavior of solvents and solutes, the dissolved substances within the tooth can pass to the outside of it, and, similarly, the dissolved substances outside the tooth can pass to the inside of it, together with the fact that the defensive mechanisms of the body are quite unable to enter and reach the bacteria within the tooth except in exceedingly small numbers through the natural openings of the root, which openings will, however, permit the organisms to pass at will from within the tooth to the outside, we must conclude that an infected tooth furnishes a condition and environment that is tremendously in favor of the invading organism inhabitating it, as compared with the host, since the latter may only rid itself of the menace by exfoliating it or absorbing it.**

CHAPTER XXIV.

ELECTIVE LOCALIZATION AND ORGAN DEFENSE. PART ONE: INFLUENCE OF DISEASED TISSUE ON ORGANISMS IN THE DISTANT FOCUS.

PROBLEM: Do diseased organs and tissues modify bacteria growing in the distant focus, or create in them a capacity for elective localization for those diseased tissues?

EXPERIMENTAL AND DISCUSSION.

This is an exceedingly difficult but very important problem, for if it is true it not only removes from the organisms some of the culpability but furnishes an explanation for some of the phenomena which develop by transferring the organisms from that focus to another host. An analysis of our clinical records and experimental data throws some direct light upon this problem. In the second chapter preceding I reviewed a case in which the bacteria from the dental infection of a patient suffering from acute involvements in the cervical and dorsal regions, when inoculated into experimental animals, developed acute involvements in the ovaries, tubes, and uterus, a lesion that is so rare, that not one per cent of experimental animals develops such a lesion from dental cultures of ordinary patients. Indeed, it had not been revealed in the taking of the physical history that this woman had had these operations, a secret which she carefully guarded. After the development of the lesions in rabbits, I asked her specifically regarding the history of disturbance in pelvic organs, when she gave me the history that one ovary and tube had been removed some years previously at one operation, and at a later time the other ovary, tube, and uterus. In this case, then, there were no such tissues to be involved.

In Chapter 62 on "Primary and Secondary Sex Organs" we report a case with a very similar history. The woman presented with a heart involvement and rheumatism quite severe. The inoculation of the rabbits developed acute involvements in the ovaries, tubes, and uterus, with an unusually severe involvement of the uterus with extensive suppuration. I sent for the patient and asked her if she had given me the history correctly. She said she had not and that she did not wish to think about it, let alone talk about it, since a purulent uterine discharge had been becoming more severe for six months and her physician had advised her that it was probably malignant and at her age an operation would not be justified. In this case, as shown in that chapter, this purulent discharge completely subsided with the removal of her infected teeth and has not recurred in two years except for a couple of days at the time that an infected sequestrum was giving trouble after one of the extractions. Had the infected uterus been the primary lesion we would not expect that the removal of the dental infection would have seriously modified the primary focus, which is very important.

In that same chapter we recite a case of a man from whose dental infection the cultures were taken and inoculated into rabbits, and which produced acute infections in the testes of each of three male rabbits, the culture being taken from three different teeth. On being questioned, his reply was "Can't a person have any secrets?" and confessed that he had had a recent severe involvement of the testes; that he had had gonorrhea twenty years previously which had been treated and supposedly cured.

In that chapter we refer to cases of ovarian cyst that have been operated and in which cases the cultures from dental infections developed ovarian cysts in the rabbits.

It is not possible to state from these clinical cases to what extent either the dental infection was originally causative in the involvement of these special tissues or to what extent these special tissues influenced the organisms growing in the focus. In the first case mentioned, however, it was seven years since the last operation; in other words, this individual did not have in her body primary sex organs to be related directly to the dental infection. It is true, however, that the dental infections which were removed had been of probably twenty years standing and her first operation had occurred fifteen years previously.

These data suggested some special studies which were conducted as follows: Tissues were taken from several different organs of the rabbit's body, macerated and placed in culture media inoculated with the same organism, a passive strain which, when it grew out, was inoculated into a series of rabbits, and careful macroscopic and microscopic studies were made to ascertain if the placing of healthy tissue of a given organ would tend to create in the bacteria an appetite for that tissue. These results were all negative, which we interpreted as meaning that a normal healthy organ did not contribute elective localization qualities to organisms growing in a focus; and that if such condition does obtain—namely, that the infected organ develops the elective localization quality in a distant focus through the influence of that organ on the culture medium—light might be gotten on this phase of the subject by the placing in the culture medium of some of the diseased organ tissue from an animal developing a severe lesion, as a result of inoculation of the animal. Since the organism which produces such a lesion does in many instances do so in several animals, it is evident that the organism had to that extent the power of elective localization. When, then, a diseased organ tissue is used for modifying a culture medium to determine whether or not that tissue will influence the organisms growing in that medium, that diseased tissue carries with it some of the original organisms having that quality. It is, therefore, important to note that, when cultures are taken from diseased organs, which have become so because of elective localization qualities of bacteria, those cultures tend to produce lesions in that same tissue in the next animal passage, which quality we have assumed they were maintaining and not acquiring, having brought it from the original focus. We are, then, practically in the same position when we take a piece of diseased and infected organ to modify media, that we would be if we took the organisms without the tissue, in which we would expect elective localization qualities. This, therefore, makes it impossible to determine with this experiment whether an infected diseased organ, which disease was not produced by the dental focus, would so modify the organisms in a dental focus as to establish elective localization qualities.

We have made many inoculations with organisms grown from the lesions which have developed in rabbits from dental infections, and have seen many instances suggesting strongly that the organisms still had elective localization qualities. A first requisite for this experiment is to develop an acute or chronic lesion by any process in an internal organ; and, after it is well established, to establish a dental condition which would tend to become infected from the blood stream. To accomplish this I have undertaken the following experiment:

Since we frequently find teeth, as illustrated in Chapter 63 on "Kidneys and Related Excretory Organs" which, when removed from the mouth of a patient having acute nephritis and placed under the skins of rabbits, produce acute kidney involvement which can be definitely identified by the presence of albumin and casts, and later verified by macroscopic and microscopic study, as shown in that chapter, we must consider the matter an open question until further data are available, the experiments for which are in progress.

I wish to insert a warning at this point: Judging from the observation as to the conclusions that are liable to be made by persons who undertake to check an experiment of this kind, I shall anticipate that some persons will undertake to put teeth under the skins of rabbits to determine whether or not it is possible by this process to accomplish this result. They may have negative results which, I shall anticipate, they will conclude are a proof that there was an error in my experiments. My warning is twofold: Firstly, that just as all individuals that have infected teeth, do not develop kidney involvement, just so all rabbits that have inoculations made by any form do not always develop lesions; and, secondly, all cases of nephritis are not caused by dental infection, and the tooth may be selected either from such a patient, or it may be a tooth with an acute apical involvement, and the kidney lesion a chronic one, in which case it will usually be true that the dental infection will not show marked elective localization qualities.

There is another source of information, however, which is very important in answering this problem. The studies reported in the various preceding chapters, particularly Chapter 4, had to do with the particular tissue that tends to break in individuals with an inherited susceptibility, in which case they tend to break in the same tissues as did their ancestors and as do their brothers and sisters. It is abundantly demonstrated that this quality is transferred from generation to generation entirely regardless of its acute development in any particular link in the chain, in which case, if the individual was a carrier only of the quality without having had that lesion develop or a lesion in that organ develop, it could not be said that a diseased organ had had anything to do with the quality of elective localization, since that was neither evidence of organ involvement nor of focal infection. It becomes clear, then, that this quality is something inherent within the individual, which is transferred from generation to generation, which may skip, in accordance with the mendelian laws, individuals who themselves may be carriers of the quality.

PART II. NATURE OF THE DEFENSIVE MECHANISMS.

This brings us to the second phase of this problem: namely, whether normal defense provides in the blood stream a series of defensive factors suited to each and all of the various tissues of the body, the absence of any one of which tends to develop in the blood stream a medium so affecting the organism growing in the focus, that it tends to select that tissue when transferred to a new host.

This is a very difficult but very important problem, since a knowledge of these factors is essential for an explanation of the phenomena that develop in clinical practice in the study of the various types of reaction, and of complete absence to reaction from dental infections. In this statement of the problem we have a distinctly different situation from that in the preceding paragraphs of this chapter in which we discuss the ability of a diseased organ to produce in the bacterium an appetite and quality of localization for that diseased organ, when the organism is transferred to a healthy host. In this part of the chapter we are not dealing with the presence of a definite substance which is the product of a diseased organ, but with the absence of some substance which, if present, would constitute defense, but whose absence constitutes susceptibility.

A line of approach to this question is suggested by the experience of the eskimos in northern latitudes. It is said, for example, that in some of the North Sea Islands every eskimo, who became exposed to measles, died. We cannot say, however, that this was so because there was generated in his body some specific substance which made him susceptible, for history shows, that for centuries on these same islands, no eskimo died of measles because none were exposed to it. It is apparent that his absence of a defense, which the white races have in part built up through a process of infection in each generation and therefore of a transmitted immunity, is entirely due to a lack in his constitution of some qualities which, if present, would constitute defense, but which defense he did not require in the absence of being exposed to the contagion; in other words, the absence of a positive factor rather than the presence of a positive factor.

When we study, then, our carefully worked out clinical histories in their relation to presence and absence of defense, as we have done in Chapter 4, and their relation to the particular types of

CHARACTERISTICS OF ACTIVE AND DEFICIENT IMMUNITY

Susceptibility Group	*Characteristics of Dental Lesions*	*Susceptibility to Systemic Involvements from Dental Infections*	*Caries*	*Periodontoclasia*	*Blood Calcium*
Absent Susceptibility	Marked rarefying osteitis	None	Slight	Marked tendency to	Ionic calcium high
Acquired Susceptibility	Rarefying osteitis surrounded by condensing osteitis	Generally absent but recent acute attack	Recent acute	Previously a tendency; is latterly not extending	Generally high, recently lowered
Mildy Inherited Susceptibility	Mild or slight rarefying osteitis	Recurring attacks	Recurring through life	Very slight	Generally lowered
Strongly Inherited Susceptibility	Slight rarefying osteitis with marked tendency to condensing osteitis	Frequent and severe attacks	Chronically severe	Practically almost absent	Chronically low

FIGURE 156.

expression which the same infection tends to produce locally about the infected teeth in these different groups, and compare these with the blood chemical analyses, saliva analyses, etc., of the individuals of these groups, we find that we are dealing in the main with individuals having very definitely differentiating qualities, already reviewed in Chapter 4. We find evidence that this quality of complete defense, or, at least ample to defend the individual against systemic involvement for the major part of a lifetime from even several dental infections, has many identifying characteristics. If we group these various differentiating qualities for the different groups of individuals according to our classification, we find a condition as shown in Figure 156. There are many other important factors which are variable in these various groups, one of which, and perhaps the most important of all, variations in the defensive mechanisms of the blood in these various groups, is discussed in Chapter 41.

SUMMARY AND CONCLUSIONS.

We are led to conclude from the available data, that we do not as yet have sufficient information to draw a close distinction between the influences of the organisms on the affected organ, in contra-distinction to the influences of the diseased organ upon the organisms in the focus. The available data suggest strongly, if they do not definitely indicate, that both these conditions exist, in some instances, either one acting entirely alone, and in some others there are indications that both exist at the same time. These data have suggested very definite researches that are being organized to throw further light upon this subject.