

TISSUE TOLERANCE TO ORTHODONTIC BANDING:
A STUDY OF MULTIVITAMIN SUPPLEMENTATION

by

SHERWOOD ALLEN DUSTERWINKLE
//

A THESIS

Submitted in partial fulfillment of the requirements
for the degree of Master of Science in Dentistry in
the Graduate School of the University of Alabama

UNIVERSITY, ALABAMA

1965

ACKNOWLEDGEMENTS

The author wishes to express his gratitude to Dr. J. F. Volker, and the University of Alabama, for the opportunity to do graduate work at this institution.

For his guidance during the preparation of this thesis, the author is greatly indebted to his advisor, Dr. E. Cheraskin. Much appreciation is given to Dr. H. P. Hitchcock, and Dr. W. Marshall Ringsdorf, Jr. for their assistance with this research.

An expression of thanks is extended to Mrs. Alda McDowell and Miss Carolyn Bryant for their excellent secretarial assistance, and to Mrs. Dorothy Ginn for her laboratory cooperation.

Sincere appreciation is extended to the thesis committee, Dr. E. Cheraskin, Dr. E. C. Sensenig, Dr. H. P. Hitchcock, Dr. R. S. McMillan, and Dr. C. A. Schneyer, who gave of their time and effort in constructively criticizing this thesis.

The author also wishes to take this opportunity to thank the Eli Lilly Company for supplying the placebo and multivitamin preparations, and to the Southern Society of Orthodontists, whose financial support helped make this study possible.

T378
D949t
1965

TABLE OF CONTENTS

	Page
CHAPTER I. INTRODUCTION	1
CHAPTER II. REVIEW OF THE LITERATURE	3
Periodontal Sequelae in Orthodontics	3
Tissue Tolerance	4
Vitamins-Minerals and Cellular Metabolism	4
Cellular Metabolism and Tissue Tolerance	7
Scope of Vitamin and Mineral Deficiency States	8
Nature of Vitamin and Mineral Deficiency States	10
CHAPTER III. METHOD OF INVESTIGATION	14
General Information	14
Measurement of Clinical Findings	14
Gingival State	14
Clinical Tooth Mobility	17
Labial Debris	20
Therapeutic Design	20
Clinical Examination	22
Gingival State	22
Clinical Tooth Mobility	22
Labial Debris Score	23
Banding Procedures	24
Therapy Supplementation	24
Supplement Consumption	24
Experimental Period	27
CHAPTER IV. RESULTS	31
Gingival State	31
Clinical Tooth Mobility	33
Labial Debris	33
Total Clinical Picture	35
CHAPTER V. DISCUSSION	40
Multicausal Origin of Health and Disease	40
Arithmetic Formula of Health and Disease	40
Perfect Health	40
Perfect Resistance	40
No Resistance	40

Table of Contents Continued

	Page
No Local Trauma	40
Great Local Trauma	40
The Normal Child	45
Group 1b	45
Group 1a	48
Group 11b	50
Group 11a	50
The Ecology of Health and Disease	53
CHAPTER VI. LIMITATIONS	56
Limitations in Experimental Design	56
Sample Size	56
Age	57
Sex	57
Time and Dosage Factors	58
Examiner Problems	58
Patient Problems	59
CHAPTER VII. SUMMARY AND CONCLUSIONS	60
REFERENCES	61
APPENDIX	67

LIST OF TABLES

	Page
1. AGE AND SEX DISTRIBUTION-PLACEBO VERSUS MULTIVITAMIN GROUP	15
2. GINGIVAL SCORING SYSTEM	16
3. FREQUENCY DISTRIBUTION-CLINICAL SCORES	18
4. METHOD OF RECORDING CLINICAL TOOTH MOBILITY	19
5. METHOD OF RECORDING LABIAL DEBRIS SCORE	21
6. MULTIVITAMIN INGREDIENTS	25
7. PLACEBO INGREDIENTS	26
8. DISTRIBUTION OF PATIENTS BY NUMBER OF PILLS CONSUMED	28
9. EXPERIMENTAL PERIOD	29
10. EFFECT OF ORTHODONTIC BANDING VERSUS NONBANDING OF THE MANDIBULAR LATERAL INCISOR TEETH WITH AND WITHOUT MULTIVITAMIN SUPPLEMENTATION UPON GINGIVAL STATE	32
11. EFFECT OF ORTHODONTIC BANDING VERSUS NONBANDING OF THE MANDIBULAR LATERAL INCISOR TEETH WITH AND WITHOUT MULTIVITAMIN SUPPLEMENTATION UPON CLINICAL TOOTH MOBILITY	34
12. EFFECT OF ORTHODONTIC BANDING VERSUS NONBANDING OF THE MANDIBULAR LATERAL INCISOR TEETH WITH AND WITHOUT MULTIVITAMIN SUPPLEMENTATION UPON LABIAL DEBRIS	36
13. EFFECT OF ORTHODONTIC BANDING VERSUS NONBANDING OF THE MANDIBULAR LATERAL INCISOR TEETH WITH AND WITHOUT MULTIVITAMIN SUPPLEMENTATION UPON CLINICAL STATE	38

LIST OF FIGURES

	Page
1. ARITHMETIC FORMULA	41
2. ARITHMETIC FORMULA AND HEALTH AND DISEASE	42
3. ENVIRONMENTAL INFLUENCE ON ARITHMETIC FORMULA	43
4. PERFECT HEALTH AND DEATH SHOWN BY ARITHMETIC FORMULA	44
5. MODERATE DISEASE IN ARITHMETIC FORMULA	46
6. UNALTERED ORAL TISSUES IN ARITHMETIC FORMULA	47
7. INCREASE IN ORAL TRAUMA IN ARITHMETIC FORMULA	49
8. DECREASE IN HOST SUSCEPTIBILITY IN ARITHMETIC FORMULA	51
9. DECREASE IN HOST SUSCEPTIBILITY AND INCREASE IN ORAL TRAUMA SHOWN IN ARITHMETIC FORMULA	52
10. PICTORIAL REPRESENTATION OF THE AVERAGE PERCENTAGE CHANGE OF THE THREE PARAMETERS INDEPENDENTLY AND COLLECTIVELY FOR THE FOUR GROUPS.	54

CHAPTER I

INTRODUCTION

(The) characteristic of living tissue to function under handicap within limited conditions may be termed tissue tolerance. Without tissue tolerance our orthodontic practice would not be possible. Of course, this attribute is a variant and differs with every individual. It is this variation of tissue tolerance that renders some of the hazards of orthodontic practice unpredictable. Thus, the incidence of root resorption, periodontal disturbance, and disturbances of the pulps of the teeth during treatment depends a great deal upon tissue tolerance. The reason for this difference in tissue tolerance cannot be explained. We simply do not know. (*italics added*)

This quotation from one of the standard orthodontic texts (Fischer, 1957a) succinctly captures current thinking in this discipline and, in so doing, serves as an excellent prelude and justification for this study.

Firstly, it is generally agreed that justification for orthodontic banding is based on the proposition that the end-result (physiologic occlusion) justifies these necessary but obviously traumatic techniques. Secondly, it is recognized that the response to orthodontic therapy is quite variable. In other words, the tissues (the periodontal structures for purposes of this study) react differently in different patients. Finally, it is universally admitted that the

mechanisms in tissue tolerance are presently unknown.

The purpose of this study is to attempt to investigate some of the possible factors which contribute to tissue tolerance through an analysis of orthodontic banding versus nonbanding with and without multivitamin supplementation in a group of presumably healthy children seeking orthodontic therapy.

CHAPTER II

REVIEW OF THE LITERATURE

To set the proper stage for this study, it is appropriate to review published material regarding: (1) local orthodontic factors in the genesis of periodontal pathosis, and (2) known host factors in tissue tolerance.

Periodontal Sequelae in Orthodontics

Almost without exception, orthodontic (McCoy and Shepard, 1956; Fischer, 1957b; Gresham, 1957; Moyers, 1958a; Strang, 1958a; Anderson, 1960; Graber, 1961a; Tarpley, 1961; Thurow, 1962; Jarabak, 1963) and periodontic (Miller, 1950; Beube, 1953; Goldman and Cohen, 1957; Sorrin, 1960; Wade, 1960; Blake and Trott, 1962; Grant, Stern, and Everett, 1963; Glickman, 1964a; Goldman, Schluger, Fox, and Cohen, 1964) textbooks concur that banding of the teeth frequently produces undesirable changes in the periodontium. As representative of the statements which have appeared in the literature is the following (Glickman, 1964b):

Orthodontic appliances tend to retain irritating food debris, which causes gingivitis. Patients should be taught proper toothbrushing when appliances are inserted, and its importance should be stressed. The condition of the periodontium should be checked regularly during orthodontic treatment and periodontal care instituted at the earliest sign of gingival disease.

This quote is relevant for two reasons. Firstly, it underlines the possible deleterious effect of orthodontic banding. Secondly, it implies that the most important cause for debris and gingivitis, and the most effective solution, is improved oral hygiene.

Tissue Tolerance

Tens of books, scores of monographs and hundreds of published articles are available on the subject of tissue tolerance. For purposes of this study, consideration will be given to four subjects: (1) the importance of vitamins and minerals in cellular metabolism, (2) cellular metabolism and tissue tolerance, (3) scope of vitamin and mineral deficiency states, and (4) nature of vitamin and mineral deficiency syndromes.

Vitamins-Minerals and Cellular Metabolism: In the final analysis, the life or death of the total organism is a function of the sum total of health or sickness of the single cells. In turn, the status of the single cells is directly related to the efficacy of their metabolism. Finally, cellular metabolism is, in part, dependent upon the presence and activity of a critical constellation of vitamin fractions and their contributions to the cellular enzyme systems. This is well-shown in the following quotation (Brin, 1964):

Early studies showed that thiamine is an essential cofactor for the oxidation of alphaketo acids such as pyruvic and alphaketoglutaric acids. The oxidation of pyruvic acid is essential for the metabolism of carbohydrates, and the oxidation of alphaketoglutaric acid is a step in the Krebs' Cycle- the final common oxidation pathway for carbohydrate, protein, and fat. More recently it was shown that thiamine

was associated with the enzyme transketolase, which functions in the utilization of pentoses, or 5-carbon sugars. Moreover, the erythrocyte is rich in the transketolase enzyme. The involvement of 5-carbon sugars in ribonucleic acids (RNA) and the function of RNA in protein synthesis is well recognized. The importance of having sufficient thiamine for normal cellular biochemical function is therefore obvious. (italics added)

In this same report (Brin, 1964), the author studied a group of eight student volunteers fed varying amounts of thiamine. While the details of his investigation are not pertinent to this study, one of his concluding remarks is relevant:

The demonstration of a specific biochemical defect in a vitamin deficiency before the onset of clinical disease confirms the essential function of the vitamin in the maintenance of normal cellular metabolism. (italics added)

In another report (Brin, Schwartzberg, and Arthur-Davies, 1964), the following significant statement is made:

It is only in acute vitamin deficiency that we observe specific clinical signs of disease. In marginal deficiency (in which a biochemical defect may already be present), such clinical signs as appetite loss, irritability and insomnia, are definitely nonspecific either for vitamin deficiency or for other disorders.

The illustrations cited here deal only with thiamine. However, there are similar studies which could be quoted indicating that other vitamin fractions also play a significant role in cellular metabolism. The important point to be underscored is that many of the known vitamins exert an indispensable effect in cellular metabolism.

The critical position of minerals in cellular metabolism has

also been investigated and summarized in biochemistry (White, Handler, Smith, and Stetten, 1959a; Kleiner and Orten, 1962a) and nutritional (Follis, 1958a; Nizel, 1960; Wohl and Goodhart, 1960a) textbooks. While most investigators agree that many elements are essential to normal metabolism, there is widespread controversy as to the specific requirements of the individual nutrients. In one standard textbook (White, Handler, Smith, and Stetten, 1959b), the authors list sodium, chlorine, potassium, calcium, phosphorus, iodine, magnesium, manganese, and copper, as known trace elements required by man. A similar report (Moore, 1960a) on human mineral requirements includes the following eight elements as being essential at present: cobalt, copper, fluorine, iodine, manganese, molybdenum, selenium, and zinc. As is true of vitamins, some of these elements play an indispensable role in enzymatic systems (Vallee, 1955). For example, magnesium is important in many of the enzymic reactions of carbohydrate metabolism. In addition, manganese, copper, zinc, molybdenum, iron and cobalt are constituents of a number of specific enzymes.

The following quotation serves as an excellent summary of the functions of the essential elements (Follis, 1958b):

Certain elements are of great importance in enzyme-catalyzed reactions. Here they may activate the enzyme system and thus become fully as important a cofactor as are certain vitamins. Of the inorganic ions related to the various steps of carbohydrate metabolism, magnesium is found to be of pre-eminent importance. Manganese enjoys an important role in the reactions which make up the citric acid cycle. In both the glycolytic and Krebs cycles other elements such as potassium,

calcium, cobalt and zinc may play important roles.

While some of the intricate metabolic dependencies upon minerals have been established, many associations have yet to be determined. It is noteworthy, however, that minerals, like vitamins, play a vital part in cellular metabolism.

Cellular Metabolism and Tissue Tolerance: It is now generally recognized that disease is a function of the interplay of one or more environmental challenges and host resistance or susceptibility (tissue tolerance). The importance of this ecologic formula for disease has been abundantly reviewed with regard to nutrition in infectious diseases. The following statement (Scrimshaw, 1962) is representative of current thinking:

There is much evidence indicating that many different types of moderate to severe nutritional deficiencies lower resistance to many infections. There is a strong tendency to assume that the principal mechanism of resistance to infection is the anamnestic one; that is, the capacity of the body to form antibodies against specific organisms or their toxins. No doubt this is very important component of resistance and both the primary and the secondary antibody response to an antigen may be impaired by severe nutrient deficiencies. This has been demonstrated for deficiencies of tryptophan, protein, vitamins A and D, ascorbic acid, thiamine, riboflavin, niacin, pyridoxine, pantothenic acid, folic acid and vitamin B₁₂.
(italics added)

The evidence for the role of nutrition in host resistance to infectious disorders is more plentiful than is the proof for nutrition and host factors to nonmicrobial challenges. Nonetheless, there is at

least presumptive evidence that nutrition may play a role in body defense to physical and chemical environmental traumata.

El-Ashiry, Ringsdorf, and Cheraskin (1964a; 1964b; 1964c) published three reports dealing with the changes in gingival state, sulcus depth, and clinical tooth mobility following prophylaxis with and without synthetic vitamin C, synthetic vitamin C plus water-soluble bioflavonoids, and natural vitamin C with bioflavonoids. This series of studies is particularly pertinent to this investigation because it demonstrates the effect of altering the host to a nonmicrobial insult (prophylaxis). These studies are also relevant because they are concerned with stomatologic problems similar to those investigated in this paper (gingival state and clinical tooth mobility). The conclusions drawn are that the effectiveness of prophylaxis is enhanced by the addition of vitamin C and further improved when the water-soluble bioflavonoids are added. Included here is part of the summary of one of the three papers:

Re-examination three weeks later (following prophylaxis with and without vitamins) showed a significant reduction in gingivitis in those groups given local (prophylaxis) and/or systemic (vitamin) therapy. It appears, within the limits of this study, that the combined treatments yielded the best results. (*italics added*)

It, therefore, appears, from these and many other published reports that it is possible to improve tissue tolerance to many and diverse environmental traumata by nutritional means.

Scope of Vitamin and Mineral Deficiency States

Evidence has been offered that nutrition and, more specifically

vitamins play a role in host resistance and susceptibility. The question now arises as to how often this problem of vitamin deficiency may occur in clinical practice. In other words, it would be desirable to learn the status of vitamin intake in presumably healthy people.

While there seems little doubt that the American public is quantitatively well-fed, there is considerable question as to whether the quality of the diet is adequate. Among the many authorities who have expressed themselves in this regard is the following (Goodhart, 1964b):

The papers summarized in this report indicate that variable but substantial proportions of subgroups of our population consume diets which fall short of the Recommended Dietary Allowances for vitamin A, thiamine, riboflavin and ascorbic acid, the vitamins routinely studied in nutrition survey work. Wherever body fluid levels were determined, like proportions of the population were found to have levels of these vitamins below accepted normal values.

For different groups of persons and for the different vitamins, the proportions consuming less than two-thirds of the Recommended Dietary Allowances ranges from less than five to more than 70 per cent.

While the studies discussed here do not represent a valid cross section of the population of the United States, we believe that they, in conjunction with those in our first report on "How Well Nourished are Americans?" (National Vitamin Foundation, 1960 Annual Report) provide incontrovertible proof that a great many Americans of all ages and socio-economic brackets are subsisting on diets which fall substantially short of the Recommended Dietary Allowance in respect to one or

more essential nutrients. (italics added)

It would appear, from such published reports, that a significant segment of the American public is receiving suboptimal amounts of many nutrients and some fall into the category of the vitamins. This point bears on the present study which concerns itself with presumably healthy children.

Although the preponderance of evidence seems to incriminate vitamins as the etiologic factors in nutritional deficiency states, there are also various reports implicating the minerals (Kato, 1937; Baxter, 1953; Richert and Westerfeld, 1953; Darby, 1956; Fitzgerald, 1956). In addition, the standard nutritional textbooks are well documented with clinical syndromes precipitated by deficiencies of specific biochemical elements. An excellent example is reported by Follis (1958c). The author implies that mineral deficiencies may actually be more prevalent than reported. However, due to inadequate diagnostic procedures and the difficulty in establishing the optimum requirements, detection has been inadequate. It is equally possible, therefore, that suboptimal amounts of trace minerals are being consumed by the American public.

Nature of Vitamin and Mineral Deficiency Syndromes

The standard textbooks (Goldman, 1953; Bernier, 1955; Orban and Wentz, 1955; Cheraskin and Langley, 1956; Moyers, 1958; Strang, 1958b; Kerr, Ash, and Millard, 1959; Tieke, Stuteville, and Calandra, 1959; Thoma and Goldman, 1960; Burket, 1961; Graber, 1961b; McCarthy and Shklar, 1964) are replete with illustrations and accounts of

various clinical states associated with specific vitamin deficiencies. Thus, there are to be found classical descriptions of scurvy, beri-beri, pellagra, ariboflavinosis. While it is generally agreed that these classical forms of disease are exciting and dramatic, it is also admitted that they are indeed rare.

The evidence which is accumulating suggests that the clinical problems are usually of multiple rather than single origin. This is supported by two different sets of observations. Firstly, it is clear from clinical experience as well as controlled studies that, in the natural state, it is rare to find a single vitamin deficiency syndrome. Many investigators have made this point, and the following is an eloquent representation (Follis, 1958d):

Perhaps the most important point to realize in a consideration of the naturally occurring deficiency disease in man is that "these diseases result from a lack of multiple nutrients rather than the deficit of a single essential. More and more attention is being given to the multiple nature concept of most deficiency disease syndromes; this should help to clarify our understanding of them and allow us to investigate them more intelligently.

The other point which should be underlined is that almost every nutrient which has been investigated can and does influence one or more other nutrients. In other words, even in controlled studies employing a single vitamin deficit, there is evidence that the end results may be due to disturbances associated with other than the deprived vitamin. This is clearly summarized in the following statement (Harte, Chow, and Sure, 1960a):

In the last issue of this book a comprehensive review of relevant literature to 1953 included well over 200 references on the various facets of the problem of dietary interrelationships. Perhaps the most striking impression received from evaluation of that literature is that hardly any study undertaken with any pair of nutrients has failed to show a significant interaction in terms of some nutritional or biochemical criterion. This is not surprising though, since each step of the chain of reactions through which a nutrient goes as it follows an appropriate metabolic pathway is mediated by at least one enzyme system, and the function of every enzyme system calls for the combined action of an apoenzyme (made up for the most part of amino acids) and a coenzyme (which usually includes a vitamin and/or a mineral element). (italics added)

Since many of the laboratory tests for mineral deficiencies are in their infancy, diagnostic procedures are severely limited. It has been shown that specific deficiencies do cause clinical signs and symptoms of disease. As is true for vitamins, it would seem logical that these disease entities might be of multiple mineral origin. The same authors (Harte, Chow, and Sure, 1960b) have discussed the interrelationships between minerals. For example, copper has been repeatedly shown to influence the incorporation of iron into hemoglobin. Also, a close relationship is reported to exist between calcium and phosphorus, as well as between sodium and potassium.

Perhaps more important than mineral interrelationships is the interdependence of vitamins and minerals. The following statement by Harte, Chow, and Sure (1960c) suggests this possibility:

Since one of the major roles in many of the vitamins is to function as coenzymes in the

host of intracellular enzyme systems responsible for overall metabolism, and since, in so many instances too, the trace minerals play critical roles in enzyme systems, it follows that there must be literally hosts of interactions between these two classes of nutrients.

In summary, therefore, it is well to indicate that single deficiency states in both vitamins and minerals appear to exist, but their occurrence is rare. Far more common, however, are multiple deficiency syndromes involving these two groups of nutrients.

CHAPTER III

METHOD OF INVESTIGATION

The design of this project is such that the method of investigation is best reviewed in three categories: (1) general information, (2) measurement of clinical findings, and (3) therapeutic design.

General Information

The patients selected for this program presented a variety of types of malocclusion. All participants were selected from a waiting list on current file in the Department of Orthodontics at the University of Alabama School of Dentistry. With one exception, all children were Caucasian. Table 1 outlines the frequency distribution in terms of the supplement employed, the age, and the sex. The youngest patient was nine; the oldest 18 years. Table 1 indicated that the majority (35.7 per cent) were in the 13-14 year age group. The number of patients are equally divided according to the type of supplement. The male-female ratio is approximately equal.

Measurement of Clinical Findings

Three clinical parameters were studied including gingival state, clinical tooth mobility, and labial debris.

Gingival State: The gingivae of the mandibular right and left lateral incisor teeth were graded on a four-point scale (Table 2). This scoring system is one of a number of available techniques, all of which possess some advantages and disadvantages. This particular

TABLE 1
 AGE AND SEX DISTRIBUTION
 (PLACEBO VERSUS MULTIVITAMIN GROUP)

age groups	Group I		total	Group II		total	total group
	placebo male	group female		multivitamin male	group female		
9-10	4 (5.7%)	4 (5.7%)	8 (11.4%)	4 (5.7%)	7 (10.0%)	11 (15.7%)	19 (27.1%)
11-12	6 (8.5%)	7 (10.0%)	13 (18.6%)	6 (8.5%)	2 (2.9%)	8 (11.5%)	21 (30.1%)
13-14	8 (11.4%)	5 (7.2%)	13 (18.6%)	8 (11.4%)	4 (5.7%)	12 (17.1%)	25 (35.7%)
15-16	0 (0.0%)	1 (1.4%)	1 (1.4%)	2 (2.9%)	1 (1.4%)	3 (4.3%)	4 (5.7%)
17-18	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (1.4%)	0 (0.0%)	1 (1.4%)	1 (1.4%)
total	18 (25.7%)	17 (24.3%)	35 (50.0%)	21 (30.0%)	14 (20.0%)	35 (50.0%)	70 (100.0%)

TABLE 2
GINGIVAL SCORING SYSTEM

score
0 = no gingivitis present
1 = slight hyperemia, swelling and loss of stippling; patient unaware of the condition
2 = moderate hyperemia, swelling and loss of stippling; tendency to bleed upon pressure and may be tender or painful
3 = marked hyperemia, swelling and loss of tissue tone; "so-called" spontaneous bleeding; may be ulcerated and is tender

method was chosen because (1) it is simple, (2) lends itself to application by the private practitioner, and (3) has been employed by others in similar studies with reasonable success (Keller, Ringsdorf, and Cheraskin, 1963; Cheraskin and Ringsdorf, 1964a; El-Ashiry, Ringsdorf, and Cheraskin, 1964a). One hundred and forty measurements were made at the initial visit (70 children times two teeth per subject) as indicated in Table 3. The greatest number of scores (77.1 per cent) is one indicating slight gingivitis. Approximately one out of every five scores (21.4 per cent) suggests moderate gingival pathoses. Only two of the 140 measurements can be considered to be ideally physiologic. Finally, Table 3 shows that the mean and standard deviation is 1.20 ± 0.43 which indicates that 68 per cent of the cases ranged from 0.77 to 1.63.

Clinical Tooth Mobility: The mobility of the mandibular left and right lateral incisor teeth were graded on a four-point scale (Table 4). This is one of several available techniques. This particular method was chosen because of its simplicity, application in clinical practice, and success by other investigators (Cheraskin and Ringsdorf, 1963a; Cheraskin and Ringsdorf, 1964b). An examination of Table 3 shows that the majority of the teeth demonstrated very slight looseness (80.7 per cent). Only one in eight had absolutely no mobility. In 7.2 per cent of the teeth there was mobility of approximately one millimeter. No teeth showed mobility greater than one millimeter. The mean and standard deviation for the entire sample proved to be 0.95 ± 0.44 which can be interpreted to mean that two-thirds of the sample (one standard

TABLE 3
 FREQUENCY DISTRIBUTION
 CLINICAL SCORES
 (INITIAL VISIT)

clinical scores	gingival state	clinical tooth mobility	labial debris	total
0	2 (1.5%)	17 (12.1%)	16 (11.4%)	35 (8.3%)
1	108 (77.1%)	113 (80.7%)	96 (68.6%)	317 (75.5%)
2	30 (21.4%)	10 (7.2%)	28 (20.0%)	68 (16.2%)
3	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
total	140 (100.0%)	140 (100.0%)	140 (100.0%)	420 (100.0%)
mean	1.20	0.95	1.09	1.08
S.D.	0.43	0.44	0.56	0.49

TABLE 4
METHOD OF RECORDING
CLINICAL TOOTH MOBILITY

score
0 = none
1 = slight
2 = approximately one millimeter
3 = more than one millimeter

deviation) ranged from 0.51 to 1.39.

Labial Debris: The labial debris deposits of the left and right mandibular lateral incisor teeth were graded on a four-point scale (Table 5). The method selected is that of a modification of the Greene-Vermillion index. It was chosen because it is simple and rapid, adaptable to the clinical practice, and has been employed with reasonable success by others (Greene and Vermillion, 1960). Table 3 shows that the majority of the areas (68.6 per cent) had soft debris covering not more than one-third of the tooth surface or the presence of extrinsic stains without other debris regardless of the surface area covered. In 20.0 per cent of the examined areas there was soft debris covering more than one-third, but not more than two-thirds, of the exposed tooth surface. In 11.4 per cent of the cases there was no debris and no stain present (zero scores). No teeth disclosed more than two-thirds of the exposed surfaces covered by debris.

Therapeutic Design

It should be recalled (see GENERAL INFORMATION) that all of the participants in this study were obtained from a waiting list on current file in the Department of Orthodontics at the University of Alabama School of Dentistry. The original sample consisted of 90 subjects. However, due to missing lateral incisors, the central incisors were banded in four of the subjects. Furthermore, 16 patients were taking a multivitamin preparation at the time of the initial visit. Since this study involved the lateral incisor teeth and the vitamin supplement, it was thought advisable to delete these 20 children from

TABLE 5
METHOD OF RECORDING
LABIAL DEBRIS SCORE

score

0 = no debris or stain present

1 = soft debris covering not more than one-third of the tooth surface, or the presence of extrinsic stains without other debris regardless of surface area covered

2 = soft debris covering more than one-third, but not more than two-thirds, of the exposed tooth surface

3 = soft debris covering more than two-thirds of the exposed tooth surface

the study. The sample size was, therefore, reduced to 70 subjects, equally divided into a placebo group (Group I) and a multivitamin group (Group II). Each patient was examined on two separate occasions.

Clinical Examination

At the initial visit, gingival state, clinical tooth mobility, and labial debris were noted as described for the mandibular right and left lateral incisor teeth. Hence, a total of 420 measurements were recorded at the initial session (Table 3).

Gingival State: The gingivae encircling a tooth was evaluated on a four-point scale (Table 2). If there was no evidence of gingivitis present, a score of zero (0) was given. A rating of one (1) was recorded if there was slight hyperemia, swelling and loss of stippling, and if the patient was unaware of the condition. The presence of moderate hyperemia, swelling and loss of stippling, a tendency to bleed upon pressure and the condition of being tender or painful, qualified a patient for a score of two (2). If there was marked hyperemia, swelling and loss of tissue tone, plus "so-called" spontaneous bleeding which may or may not have been associated with tenderness and ulceration, the patient was scored a three (3). Table 3 indicates that there were no three scorings.

Clinical Tooth Mobility: The clinical mobility was established digitally on a four-point scale. The blunt ends of two explorers were used for this purpose. The absence of any mobility was recorded as a zero (0). The presence of slight mobility was given the score of one (1). If approximately one millimeter of clinical tooth mobility was observed, a grade of two (2) was recorded. More than one millimeter

of mobility was scored as three (3). Table 3 shows that there were no three readings.

Labial Debris Score: By clinical examination with mouth mirror and explorer, the soft debris found on the labial surface of the mandibular right and left lateral incisors was recorded on a four-point scale. The scoring was patterned after a system perfected by Greene and Vermillion (1960).

Firstly, a score of zero (0) was awarded when no debris or stain was present. Secondly, the presence of soft debris covering not more than one-third of the tooth surface, or the presence of extrinsic stains without other debris regardless of surface area covered, was scored as one (1). Thirdly, soft debris enveloping more than one-third but not more than two-thirds of the exposed tooth surface was scored as two (2). Finally, soft debris covering more than two-thirds of the exposed tooth surfaces was recorded as three (3). Table 3 points up the absence of three (3) ratings.

In view of the fact that the central incisors represent adjacent teeth, it was thought advisable to band the lateral incisors. Accordingly, on a random basis, either the mandibular right or left lateral incisor was banded. This would serve two purposes: (1) the contra-lateral incisor could be used as a control within each patient, and (2) there would be no danger of superimposing the effects of banding upon the adjacent tooth.

Banding Procedures

Using Unitek perma-chrome strips (.003 x .125 inches), bands were constructed for either the left or right mandibular lateral

incisor. In addition, a twin-tie twin-wire bracket was welded to the labial surface of the band. Each band was cemented to the tooth with the cervical edge of the material located approximately one millimeter subgingivally on the mesial and distal sides. All excess cement was then carefully removed.

Therapy Supplementation

At the conclusion of the initial visit, each patient was given either a multivitamin or a placebo capsule indistinguishable in size, shape, and color. Both capsules were prepared and donated by the Eli Lilly Company of Indianapolis, Indiana. The ingredients of each capsule are listed in Tables 6 and 7.

The supplement distribution was made on a random selection basis. The examiner was unaware of the dispensing of the supplements. Each participant was instructed to take daily one capsule during breakfast and one capsule during the evening meal. The next appointment was scheduled approximately thirty days later. A total of 65 capsules was placed in a cardboard pill-box and given to the patient with a request that the pill-box be returned at the next visit.

Supplement Consumption: At the second visit, the pill-box was returned and the number of remaining capsules counted. Table A (see APPENDIX) shows the number of capsules consumed by each patient. It is of interest to note that only three cases (1744, 1757, and 1764) of the total 70 subjects failed to return the pill-box. Further, it can be seen from Table A (see APPENDIX) that the average number of capsules consumed was approximately equal in both the placebo group (51.8)

TABLE 6
MULTIVITAMIN INGREDIENTS

Each multivitamin-trace mineral capsule (Mi-Cebrin T) contained the following ingredients:

thiamin mononitrate (vitamin B ₁)	15.00 mg.
riboflavin (vitamin B ₂)	10.00 mg.
pyridoxine hydrochloride (vitamin B ₆)	2.00 mg.
pantothenic acid (as calcium pantothenate, racemic)	10.00 mg.
nicotinamide	100.00 mg.
vitamin B ₁₂ (activity equivalent)*	7.50 mg.
ascorbic acid (as sodium ascorbate) (vitamin C)	150.00 mg.
alphatocopherol (as alphotocopheryl succinate) (vitamin E)	5.00 mg.
vitamin A synthetic (25,000 units)	7.50 mg.
vitamin D synthetic (1,000 units)	25.00 mcg.

contains also:

iron (as ferrous sulfate)	15.00 mg.
copper (as the sulfate)	1.00 mg.
iodine (as potassium iodide)	0.15 mg.
cobalt (as the sulfate)	0.10 mg.
boron (as boric acid)	0.10 mg.
manganese (as the glycerophosphate)	1.00 mg.
molybdenum (as ammonium molybdate)	0.20 mg.
zinc (as the chloride)	1.50 mg.
magnesium (as the oxide)	5.00 mg.

*obtained from extractives of suitable microbial organisms and determined microbiologically against vitamin B₁₂ standard

TABLE 7
PLACEBO INGREDIENTS

Each placebo contained the following ingredients:

milk sugar	333.330 mg.
starch powder	141.330 mg.
sorghum starch	26.600 mg.
magnesium stearate	10.200 mg.
acacia	15.000 mg.
talc	40.000 mg.
cellulose acetate phthalate	10.000 mg.
diethyl phthalate	2.000 mg.
shellac	1.000 mg.
calcium carbonate ppt.	100.000 mg.
calcium sulfate	200.000 mg.
sugar, cane	400.000 mg.
F D and C yellow no. 5	0.065 mg.
F D and C red no. 3	0.009 mg.
polysorbate 80	0.007 mg.
trace quantities of coating material	
titanium dioxide	
glycerin	
gelatin	
propyl parahydroxybenzoate	
beeswax, white	
methyl parahydroxybenzoate	

and the multivitamin group (52.0). Table 8 demonstrates the distribution of patients by number of pills consumed. By consolidation, five distinct classes are listed. The mode in both groups falls within the range 50-59 total capsules consumed. In each group this represents 22.4 per cent of the sample size. From Table A (see APPENDIX), the least number of capsules consumed is shown to be 25, and the greatest quantity to be 65 (all the capsules within the pill-box).

Experimental Period: At both the initial and final visits the date was recorded and the number of days between visits established. Table A (see APPENDIX) shows the number of days between visits for each patient. It will be observed that the shortest experimental period is 15 days in one patient in the placebo group (case #1800). The longest experimental period is 35 days and occurs in two subjects (cases #1739 and #1740) in the placebo group and in one individual (case #1790) in the multivitamin group. It is also clear from Table A (see APPENDIX) that the average number of days between visits is somewhat different in the two groups. The experimental period for the multivitamin group was approximately one day longer than the corresponding period for the placebo group. Table 9 shows the mode for both groups to be 29 days. In the placebo group this represents 17.1 per cent of the total sample. The mode for the multivitamin group includes 14.3 per cent of the 70 subjects. It is noteworthy that 5.7 per cent of the patients (all within the placebo group) have an experimental period less than 27 days.

At the second visit, the clinical state (gingival score, clinical tooth mobility, and labial debris score) was again evaluated in a

TABLE 8
 DISTRIBUTION OF
 PATIENTS BY NUMBER
 OF PILLS CONSUMED

number of pills	placebo group	multivitamin group	total group
20-29	1 (1.5%)	0 (0.0%)	1 (1.5%)
30-39	4 (5.9%)	1 (1.5%)	5 (7.4%)
40-49	5 (7.5%)	11 (16.4%)	16 (23.9%)
50-59	15 (22.4%)	15 (22.4%)	30 (44.8%)
60-69	9 (13.4%)	6 (9.0%)	15 (22.4%)
total	34 (50.7%)	33 (49.3%)	67 (100.0%)

TABLE 9
 EXPERIMENTAL PERIOD
 (DAYS BETWEEN VISITS)

number of days	placebo group	multivitamin group	total group
<27	4 (5.7%)	0 (0.0%)	4 (5.7%)
27	0 (0.0%)	4 (5.7%)	4 (5.7%)
28	6 (8.6%)	5 (7.1%)	11 (15.7%)
29	12 (17.1%)	10 (14.3%)	22 (31.4%)
30	4 (5.7%)	6 (8.6%)	10 (14.3%)
31	2 (2.9%)	3 (4.3%)	5 (7.2%)
32	3 (4.3%)	2 (2.9%)	5 (7.2%)
33	1 (1.4%)	1 (1.4%)	2 (2.8%)
34	1 (1.4%)	3 (4.3%)	4 (5.7%)
35	2 (2.9%)	1 (1.4%)	3 (4.3%)
total	35 (50.0%)	35 (50.0%)	70 (100.0%)

manner similar to the initial visit. The band was then removed, the tooth cleaned, and the patient and parent instructed in proper oral hygiene technique.

CHAPTER IV

RESULTS

It should be recalled (see CHAPTER THREE: METHOD OF INVESTIGATION) that three different clinical parameters (gingival state, clinical tooth mobility, labial debris) of the periodontal state were studied before and after orthodontic banding versus nonbanding with placebo versus multivitamin supplementation. These three clinical measures will now be analyzed independently and collectively in the light of the changes in the local environment and host status.

Gingival State

Table B (see APPENDIX) lists the original data for the 70 participants. Included are the case numbers, the initial and final gingival scores for the placebo-banded (Group Ia) and nonbanded (Group Ib) lateral incisor teeth and for those with multivitamin supplementation with (Group IIa) and without (Group IIb) orthodontic bands. Table 10 is a summary of the raw data listed in Table B.

Attention is directed to the percentage variation in the four groups. The greatest increase is found in Group Ia. Specifically, it will be noted that the mean gingival score increased ten per cent in the children provided with the placebo supplement and in whom the mandibular lateral incisor teeth were banded. According to Table 10, next in order, the gingival state worsened nine per cent in Group IIa.

TABLE 10
 EFFECT OF ORTHODONTIC BANDING VERSUS
 NONBANDING OF THE MANDIBULAR LATERAL
 INCISOR TEETH WITH AND WITHOUT
 MULTIVITAMIN SUPPLEMENTATION
 UPON GINGIVAL STATE

gingi- val grades	Group I (placebo group)				Group II (multivitamin group)			
	Group Ia (banded)		Group Ib (nonbanded)		Group IIa (banded)		Group IIb (nonbanded)	
	(initial)	(final)	(initial)	(final)	(initial)	(final)	(initial)	(final)
0	2	0	0	2	0	0	0	4
1	28	28	29	27	24	20	27	25
2	5	7	6	6	11	15	8	6
3	0	0	0	0	0	0	0	0
total	35	35	35	35	35	35	35	35
mean	1.09	1.20	1.17	1.11	1.31	1.43	1.23	1.06
S.D.	0.45	0.42	0.38	0.48	0.48	0.51	0.42	0.54
P	>0.200		=0.500		>0.200		>0.100	
percentage change	+10%		-5%		+9%		-14%	

These teeth were also banded. However, the subjects received multi-vitamin supplementation. In those with no therapy, so to speak (Group Ib), there is a five per cent decrease in the mean scores. Finally, Group IIb demonstrates a decrease of 14 per cent. These are the children provided with the vitamin and in whom banding did not take place. However, it is also clear from student t-test (Snedecor, 1956) analyses (Table 10) that none of the groups is statistically significant at the five per cent confidence level.

Clinical Tooth Mobility

Table C (see APPENDIX) lists the original data for the 70 subjects. Included are the case numbers, the initial and final clinical tooth mobility scores for the placebo-banded (Group Ia) and non-banded (Group Ib) lateral incisor teeth and for those with multivitamin supplementation with (Group IIa) and without (Group IIb) banding. Table 11 is a summary of the information shown in Table C.

Table 11 shows that the mean clinical tooth mobility score increased ten per cent in the participants provided with the placebo supplement and in whom the mandibular lateral incisor teeth were banded (Group Ia). According to Table 11, the mean percentage change (-9 per cent) is the same for Groups Ib (nonbanded-placebo) and IIa (banded-multivitamin). In effect this suggests that those children receiving orthodontic banding and multivitamin-mineral supplementation, fared no worse nor better than those who received no band and a placebo capsule. Finally, Group IIb indicates a decrease of 29 per cent in clinical tooth mobility scores. It is noteworthy that it is only in this group

TABLE 11
 EFFECT OF ORTHODONTIC BANDING VERSUS
 NONBANDING OF THE MANDIBULAR LATERAL
 INCISOR TEETH WITH AND WITHOUT
 MULTIVITAMIN SUPPLEMENTATION
 UPON CLINICAL TOOTH MOBILITY

clinical tooth mobility grades	<u>Group I</u> (placebo group)				<u>Group II</u> (multivitamin group)			
	Group Ia (banded)		Group Ib (nonbanded)		Group IIa (banded)		Group IIb (nonbanded)	
	(initial)	(final)	(initial)	(final)	(initial)	(final)	(initial)	(final)
0	5	2	5	4	4	7	3	11
1	28	31	25	30	30	27	30	24
2	2	2	5	1	1	1	2	0
3	0	0	0	0	0	0	0	0
total	35	35	35	35	35	35	35	35
mean	0.91	1.00	1.00	0.91	0.91	0.83	0.97	0.69
S.D.	0.45	0.34	0.54	0.37	0.37	0.45	0.37	0.48
P	>0.300		>0.400		>0.400		<0.010*	
percentage change	+10%		-9%		-9%		-29%	

* statistically significant

that there is a statistically significant change ($P < 0.010$).

Labial Debris

Table D (see APPENDIX) lists the original information for the 70 children. Shown are the case numbers, the initial and final labial debris scores for the placebo-banded (Group Ia) and nonbanded (Group Ib) lateral incisor teeth and for those with multivitamin supplementation with (Group IIa) and without (Group IIb) banding. Table 12 summarizes the data listed in Table D.

It is clear from Table 12 that the greatest change, an increase of 39 per cent in mean labial debris score occurred in Group Ia. Thus, the subjects receiving no vitamin with banding showed the greatest clinical alteration. Secondly, Table 12 indicates an 18 per cent rise in debris with banding and multivitamin therapy. There is an increase of ten per cent in Group Ib (the group with no local or systemic manipulation). Finally, the only group demonstrating a reduction in labial debris is Group IIb (those not banded but given the multivitamin).

It is of particular interest that only two of the groups indicate statistically significant changes during the experimental period. Specifically, there is a statistically significant ($P < 0.005$) rise in debris with banding and placebo therapy (Group Ia); there is a statistically significant decrease in labial debris with multivitamins and nonbanding ($P < 0.050$) as shown in Group IIb.

Total Clinical Picture

Tables 10-12 indicate, on a mean percentage basis, one striking common denominator. In all instances, Group Ia proved to have the

TABLE 12
 EFFECT OF ORTHODONTIC BANDING VERSUS
 NONBANDING OF THE MANDIBULAR LATERAL
 INCISOR TEETH WITH AND WITHOUT
 MULTIVITAMIN SUPPLEMENTATION
 UPON LABIAL DEBRIS

debris scores	Group I (placebo group)				Group II (multivitamin group)			
	Group Ia (banded)		Group Ib (nonbanded)		Group IIa (banded)		Group IIb (nonbanded)	
	(initial)	(final)	(initial)	(final)	(initial)	(final)	(initial)	(final)
0	4	1	4	6	3	3	5	12
1	24	15	24	16	24	18	24	20
2	7	18	7	13	8	13	6	3
3	0	1	0	0	0	1	0	0
total	35	35	35	35	35	35	35	35
mean	1.09	1.54	1.09	1.20	1.14	1.34	1.03	0.74
S.D.	0.57	0.62	0.57	0.73	0.54	0.69	0.52	0.62
P	<0.005*		>0.100		>0.100		<0.050*	
percentage change	+39%		+10%		+18%		-28%	

* statistically significant

greatest rise in the three clinical parameters investigated. Group IIb showed the greatest reduction in all three instances. It was thought advisable to restudy the data in composite form.

Table 13 indicates that the greatest increase in clinical findings (the constellation of gingival state, clinical tooth mobility and labial debris) was found in Group Ia. In other words, the children banded and given the placebo regime fared worst (21 per cent) in overall clinical state. The other group of children (Group IIa) also banded but with multivitamin supplements, showed a seven per cent increase in total clinical state score. Thirdly, the group (Group Ib) which in effect received nothing (no banding and placebo) shows a reduction of one per cent. Finally, on a mean percentage basis, the only group which significantly improved (Group IIb) was characterized by no banding and multivitamin supplementation (-23 per cent).

Two points deserve special note. Firstly, the mean percentage changes for the total clinical state follow the earlier described mean percentage changes for the individual clinical parameters (Tables 10-12). It should be recalled, that by statistical analyses, gingival state was not significantly altered in any of the four groups (Table 10). In contrast, Table 11 (clinical tooth mobility) showed a statistically significant reduction of 29 per cent in Group IIb (nonbanded-multivitamin). Finally, Table 12 suggests that two of the groups demonstrated statistically significant variations (Groups Ia and IIb). Now it is clear from Table 13 that there is a statistically significant overall increase in clinical state ($P < 0.005$) with banding and

TABLE 13
 EFFECT OF ORTHODONTIC BANDING VERSUS
 NONBANDING OF THE MANDIBULAR LATERAL
 INCISOR TEETH WITH AND WITHOUT
 MULTIVITAMIN SUPPLEMENTATION
 UPON CLINICAL STATE

clinical grades	<u>Group I</u> (placebo group)				<u>Group II</u> (multivitamin group)			
	Group Ia (banded)		Group Ib (nonbanded)		Group IIa (banded)		Group IIb (nonbanded)	
	(initial)	(final)	(initial)	(final)	(initial)	(final)	(initial)	(final)
0	11	3	9	12	7	10	8	27
1	80	74	78	73	78	65	81	69
2	14	27	18	20	20	29	16	9
3	0	1	0	0	0	1	0	0
total	105	105	105	105	105	105	105	105
mean	1.03	1.25	1.09	1.08	1.12	1.20	1.08	0.83
S.D.	0.49	0.52	0.50	0.55	0.49	0.61	0.47	0.69
P	<0.005*		=0.500		>0.300		<0.005*	
percentage change	+21%		-1%		+7%		-23%	

* statistically significant

placebo therapy. On the other hand, those in Group IIb (nonbanded-multivitamin) demonstrated a 23 per cent decrease or improvement in the overall clinical picture. It is also noteworthy that the remaining two groups were not statistically significant.

CHAPTER V

DISCUSSION

It is generally recognized that health and disease is of multicausal origin (Kruse, 1953; Dubos, 1959; Rogers, 1960; May, 1961; Selye, 1961). Whether one remains well or becomes ill is the end-result of the interplay of one or more diverse environmental threats, and the capacity of the organism to cope with the environment successfully (via host resistance) or unsuccessfully (through host susceptibility). This ecologic concept may be expressed simply in algebraic form (Figure 1).

For illustrative purposes only, it is fair to assume that zero on the right side of the equation signifies no disease or, percentagewise, zero per cent disease (perfect health); one hundred represents total disease or death (Figure 2). To pursue this hypothesis, it is reasonable to assign a ten-point scale to the environment. Thus zero would represent no environmental threat, and ten a maximal challenge (Figure 3). Likewise, host state can be quantitated on a ten-point scale. The complete absence of host susceptibility (perfect resistance) indicated by zero and maximal susceptibility (no host resistance) by ten (Figure 4).

It is now clear that perfect health, presumably a hypothetical situation, is represented by no host susceptibility (zero) times no environmental bombardment (zero), which equals no disease (Figure 4).

Figure 1

$$\begin{array}{r} \text{host} \\ \text{resistance} \\ \text{or} \\ \text{susceptibility} \end{array} \times \begin{array}{r} \text{environmental} \\ \text{challenge} \end{array} = \begin{array}{r} \text{health} \\ \text{or} \\ \text{disease} \end{array}$$

Figure 2

$$\begin{array}{r} \text{host} \\ \text{resistance} \\ \text{or} \\ \text{susceptibility} \end{array} \times \begin{array}{r} \text{environmental} \\ \text{challenge} \end{array} = \begin{array}{r} \text{health (0)} \\ \text{death (100)} \end{array}$$

Figure 3

$$\begin{array}{rcccl} & & \text{no} & & \\ & & \text{environmental} & & \\ & & \text{challenge} & & \\ & & (0) & & \\ \text{host} & & & & \text{health (0)} \\ \text{resistance} & \times & & = & \\ \text{or} & & \text{severe} & & \\ \text{susceptibility} & & \text{environmental} & & \text{death (100)} \\ & & \text{challenge} & & \\ & & (10) & & \end{array}$$

Figure 4

no host susceptibility (0)	x	no environmental challenge (0)	=	perfect health (0)
high host susceptibility (10)	x	severe environmental challenge (10)	=	death (100)

At the other pole, marked susceptibility (graded ten) times great trauma (assigned ten) yields one hundred per cent disease or death (Figure 4).

These are the limits of health and disease- the white and black of the spectrum. While these poles are dramatic, the common clinical experience is represented by the infinite number of shades of gray between zero and one hundred per cent on the right side of the equation. Thus, the usual clinical problem may be algebraically expressed (Figure 5) and characterizes the routine patient with slight susceptibility to disease living in a moderately noxious environment.

The Normal Child

The children in this study were not grossly ill. On the other hand, it must be granted that they were not in perfect health. Hence, for illustrative purposes only, the value of two is assigned to host susceptibility and a like score of two for the minimal oral trauma which prevails in the usual clinical case. Therefore, at the start of this experiment, the average health status of the children could be regarded as closer to perfect health than total disease (Figure 5). An attempt will now be made to relate the observed results in this study to the ecologic concept of health and disease as pictured in the arithmetic formula.

Group Ib

In Group Ib host state was not altered as evidenced by the placebo therapy. Hence, host susceptibility remains unchanged (two). Likewise, in Group Ib, the lateral incisor tooth was not banded. Thus, no additional oral trauma has been introduced. Accordingly, the oral

Figure 5

$$\begin{array}{ccccc} \text{host} & & \text{oral} & & \text{moderate} \\ \text{susceptibility} & \times & \text{trauma} & = & \text{disease} \\ (2) & & (2) & & (4) \end{array}$$

Figure 6

no change in host susceptibility (placebo) (2)	x	no change in oral trauma (no band) (2)	=	no change in oral tissues (4)	gingival state (-5%)
					clinical tooth mobility(-9%)
					labial debris score (+10%)
					total clinical state (-1%)

environment remains two (Figure 6). Thus, the end result in terms of the oral parameters studied, should be unchanged. This is supported by the observations described earlier (see CHAPTER FOUR: RESULTS). It will be recalled that, in Group Ib, gingival state improved five per cent (Table 10), clinical tooth mobility decreased nine per cent (Table 11), labial debris score rose ten per cent (Table 12), and the total clinical state changed but one per cent (Table 13). More importantly, none of the mean clinical alterations proved to be statistically significant (Tables 10-13). It is noteworthy that the clinical results fit the ecologic hypothesis of health and disease.

Group Ia

In Group Ia host state was not altered as evidenced by the placebo supplement. Hence, host susceptibility remains unchanged with a rating of two (Figure 7). However, in these children an orthodontic band was placed on one of the mandibular lateral incisor teeth. Hence, in this instance, a local challenge has been introduced. For purposes of discussion, the initial score of two (Figure 5) has been increased to three (Figure 7). According to the equation, one would expect a worsening of the clinical picture from four to six. This is borne out by the observed data (see CHAPTER FOUR: RESULTS). It will be recalled that in Group Ia gingival score and clinical tooth mobility increased ten per cent (Tables 10-11), labial debris score rose 39 per cent (Table 12), and the total clinical state increased 29 per cent (Table 13). More importantly, statistically significant changes in the form of worsening were noted in both labial debris score and total clinical

Figure 7

no					
change		change	increase	gingival state	(+10%)
in		in	in	clinical tooth mobility	(+10%)
host	x	oral	=	labial debris score	(+39%)*
susceptibility		trauma		total clinical state	(+29%)*
(placebo)		(band)			
(2)		(3)	(6)		

*statistically significant

state. Thus, the clinical results appear to be consistent with the hypothetical algebraic formula.

Group IIb

Group IIb received no orthodontic band. Hence, the local environment remained the same (Figure 8). This can be expressed by maintaining the original score (two). However, the host state may have been altered by virtue of multivitamin supplementation. On the assumption that this is true, the previous score of two is reduced to one. Referring to the algebraic equation (Figure 8), the product now equals two. Therefore, the hypothesis is that there should be improvement in these children. This is supported by the findings (see CHAPTER FOUR: RESULTS). More specifically, gingival state improved 14 per cent (Table 10), clinical tooth mobility decreased 29 per cent (Table 11), labial debris was reduced 28 per cent (Table 12), and the total clinical state improved 23 per cent (Table 13). More importantly, statistically significant differences were noted in clinical tooth mobility, labial debris score, and total clinical state. Hence, the observed findings appear to be consistent with the arithmetic predictions.

Group IIa

Finally, in Group IIa, the local trauma was increased by virtue of the orthodontic band, and host susceptibility reduced by multivitamin supplementation (Figure 9). Pursuing the algebraic explanation, the environmental score is now raised from two to three, while the host susceptibility number is decreased from two to one. One would expect, if this equation is valid, that the clinical state would worsen, but not as much as when banding and placebo therapy were

Figure 8

decrease in host susceptibility (multivitamin) (1)	x	no change in oral trauma (no band) (2)	=	decrease in oral pathosis (2)	gingival state clinical tooth mobility labial debris score total clinical state	(-14%) (-29%)* (-28%)* (-23%)*
---	---	--	---	---	--	---

*statistically significant

Figure 9

decrease		increase		decrease	gingival state	(+9%)
in		in		in	clinical tooth mobility	(-9%)
host	x	oral	=	oral	labial debris score	(+18%)
susceptibility		trauma		pathosis	total clinical state	(+7%)
(multivitamin)		(band)				
(1)		(3)		(3)		

combined (Group Ia). It will be noted that the product of this equation is three. This score is indeed less than the grade of six observed in Group Ia, but more than the score of two in Group IIb. This is in agreement with the observations reported elsewhere (see CHAPTER FOUR: RESULTS). It is clear that, in Group IIa, there was a nine per cent increase in the gingival scoring (Table 10), a nine per cent reduction in clinical tooth mobility (Table 11), an 18 per cent rise in labial debris (Table 12), and a seven per cent overall worsening in clinical state (Table 13). More importantly, none of these changes is statistically significant. Thus, the observed results appear to be consistent with the hypothetical arithmetic formula.

The Ecology of Health and Disease

Figure 10 is a pictorial representation of the average percentage change of the three clinical parameters independently and collectively for the four groups. It is of interest that in Group Ia all of the parameters have worsened. This is consistent with ecologic thinking and supported by clinical observation (Fischer, 1957b; Moyers, 1958a; Anderson, 1960; Tarpley, 1961; Jarabak, 1963). Figure 7 shows that by utilizing the arithmetic system, there is also a considerable increase in clinical pathosis (six).

In contrast, Group IIb (Figure 10) indicates that all of the parameters independently and collectively show improvement. This is in parallel with the arithmetic prediction (Figure 8) as shown by the score of two.

It will also be observed (Figure 10) that Group IIa and Group Ib occupy intermediate positions. This is underlined by the values of

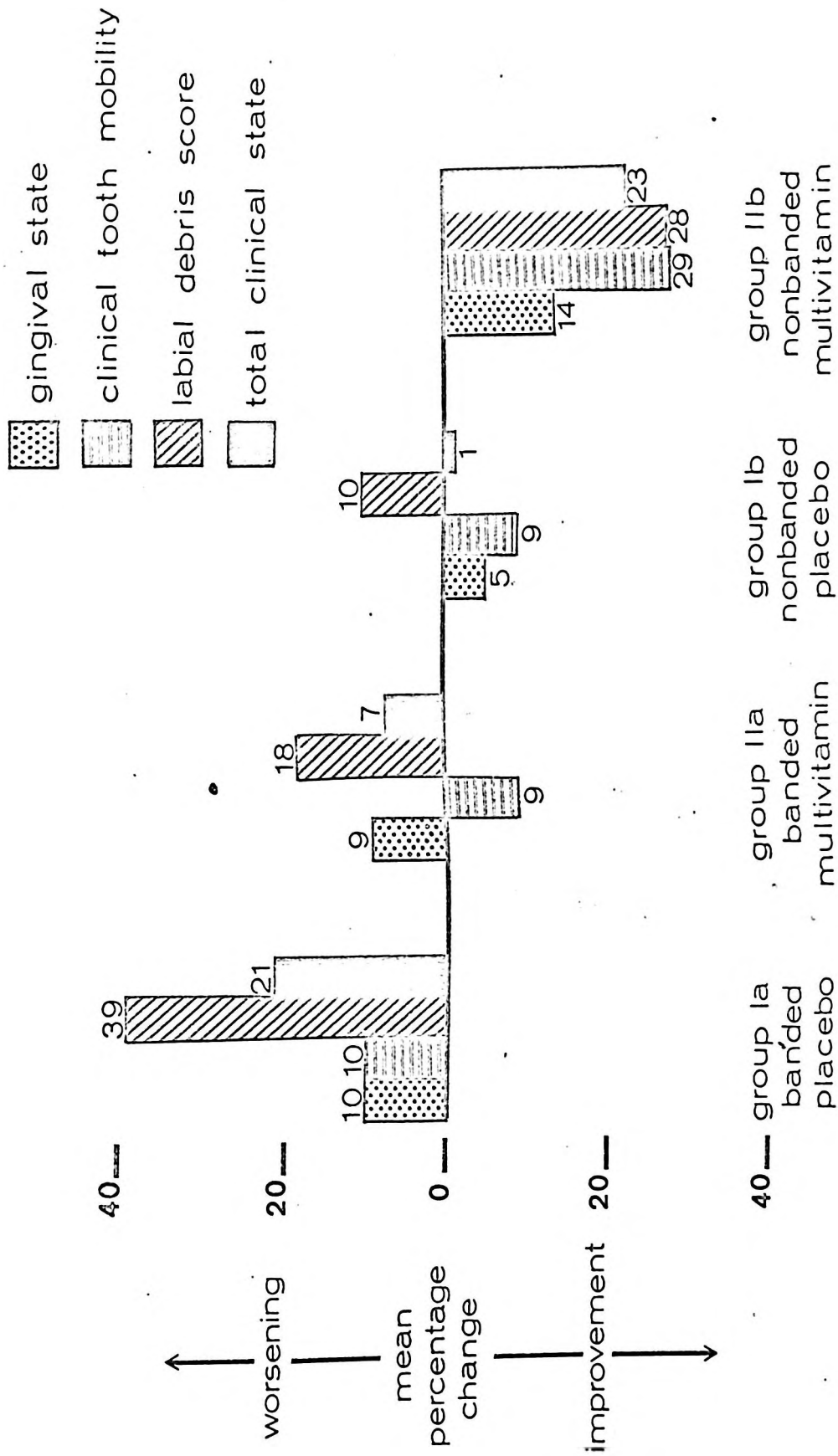


Figure 10

four and three (Figures 9 and 6) respectively in the algebraic formula.

The evidence from Tables 10-13 and Figure 10 indicates that, orthodontic banding without systemic therapy produced a statistically significant worsening in clinical pathosis. The data from Tables 10-13 and Figure 10, when multivitamin therapy was supplied and oral trauma not introduced, show some betterment. This is consistent with various reports in the literature demonstrating that vitamin therapy does indeed yield beneficial periodontal effects (Boyle, 1941; Stuhl, 1943; Roth, 1945; Wellensiek, 1956; Roth, Greene, and Kirsch, 1957; Carvel and Halperin, 1961).

CHAPTER VI

LIMITATIONS

While this study demonstrates, in part, a practical solution to the question of tissue tolerance to orthodontic banding, it raises many other problems. Hence, it is appropriate to consider, in hindsight, some of the limitations of this investigation.

A number of items deserve elaboration and enlargement. In general, three areas merit special attention: (1) problems in experimental design, (2) examiner problems, and (3) patient problems.

Limitations in Experimental Design

Difficulties in experimental design have manifested themselves in various ways. Among the more obvious problems were those encountered in sample size, age, sex, and time and dosage factors.

Sample Size: It should be recalled (see CHAPTER THREE: METHOD OF INVESTIGATION) that the original experimental group consisted of 90 children. Because the mandibular lateral incisor was absent, requiring banding of the central incisor, or for other technical reasons, four of the 90 subjects were eliminated. Since this study was concerned with multivitamin supplementation, it was thought advisable to delete those subjects already taking vitamin preparations. Accordingly, the records of an additional 16 children were deleted. Hence, the entire project centered about the observations of 70 participants.

Obviously, this is a small sample particularly since it had to then be subdivided into Group I (placebo) and Group II (multivitamin). This point is worthy of note because, with such small samples, some of the statistical analyses bordered on being significant. It is likely that some would have been significant had the sample been larger. This is further underscored by the fact that, in Table 13 (see CHAPTER FOUR: RESULTS), there are significant differences in part because of the larger sample size. Therefore, it would be interesting to repeat this experiment utilizing a greater number of patients.

Age: Earlier mention was made (see Table 1, CHAPTER THREE: METHOD OF INVESTIGATION) that the experimental group consisted of young children ranging in age from nine to 18. It is obvious from the age range that some of the children displayed clinical dentitions in the transitional stage of development while others presented exclusively permanent dentitions. Because of the relatively small sample and consequent difficulties with further subdivision, age was not considered in this study. It would be desirable, however, to expand the sample size and investigate the influence of age upon the various clinical parameters employed in this project.

Sex: Although sex was recorded in the original data, it was not considered in this study. It was felt that sex analysis, like age, would yield groups of too small a size. Thus, it would be of interest to restudy a larger group of children with consideration given to possible sex difference.

Time and Dosage Factors: The initial therapeutic design called for an experimental period of thirty days. Obviously, in some

patients the experimental period was less than thirty days; in others, greater than thirty days (see Table A. APPENDIX). Likewise, the number of pills consumed varied greatly. This is to be expected, however, since capsule consumption is relatively proportional to the length of the experimental period. Since this study is concerned with the use of multivitamin supplementation, it is logical to assume that some of the children required this form of therapy; others did not. Further, of those receiving multivitamin supplements, there were undoubtedly some who would have fared better had they been given larger doses and for longer periods of time. Hence, it would be highly interesting to repeat this project varying the dosage and the experimental interval.

It was thought advisable, since this is the first study of its kind in orthodontics, to employ a multivitamin supplement. It would be very helpful if this program could be repeated utilizing independent vitamin fractions to determine whether they singly exert any effect and the relative merits of different vitamin fractions.

Examiner Problems

There is no completely satisfactory quantitative technique for the evaluation of gingival state, clinical tooth mobility, or labial debris. This was one of the complicating features of the study. Under the present circumstances, it would be helpful, if this study could be redone allowing several examiners to share the judgements.

Patient Problems

In any clinical investigation, there is the question as to whether the subjects have truly obeyed the instructions. As far as

can be determined, this group was most cooperative. This is attested to by the varying numbers of capsules remaining in the pill-box at the second visit.

It is further possible that environmental changes during the experimental period may have played a role. For example, there may have been an increase or decrease in dietary vitamin consumption, since the patients were at liberty to consume their ordinary diet. Therefore, a more complete study might be instituted if the children could be better supervised.

CHAPTER VII

SUMMARY AND CONCLUSIONS

An attempt was made in this investigation to study, under double blind conditions (multivitamin-mineral versus placebo supplementation), the effect of orthodontic banding upon clinical pathosis (gingival state, clinical tooth mobility, labial debris).

Within the limits of this experiment, it appears that there are substantial differences between the placebo and multivitamin groups. The evidence suggests that those children receiving an orthodontic band and multivitamin supplementation fared considerably better than those subjected to orthodontic banding and placebo supplement.

It is hoped that this investigation may catalyze further research into the etiology of the pathologic response to orthodontic banding.

REFERENCES

- Anderson, G. M. Practical orthodontics. Ninth edition. 1960. St. Louis, The C. V. Mosby Company. p. 458.
- Baxter, J. H. and Vanwyk, J. J. A bone disorder associated with copper deficiency. I. Gross morphological, roentgenological and chemical observations. Bull. Johns Hopkins Hosp. 93: #1, 1-13, July 1953.
- Bernier, J. L. The management of oral disease. 1955. St. Louis, The C. V. Mosby Company. pp. 358-367.
- Beube, F. E. Periodontology. 1953. New York, The MacMillan Company. pp. 278-279.
- Blake, G. C. and Trott, J. R. Periodontology. 1962. London, Butterworths. pp. 39-41.
- Boyle, P. E. Effects of various dietary deficiencies on the periodontal tissues of the guinea pig and of man. J. A. D. A. 28: 11, 1788-1793, November 1941.
- Brin, M. Erythrocyte as a biopsy tissue for functional evaluation of thiamine adequacy. J. A. M. A. 187: #10, 186-189, March 7, 1964.
- Brin, M., Schwartzberg, S. H. and Arthur-Davies, D. A vitamin evaluation program as applied to 10 elderly residents in a community home for the aged. J. Am. Geriat. Soc. 12: #5, 493-499, May 1964.
- Burket, L. W. Nutrition-vitamins-deficiency diseases. Burket, L. W. Oral Medicine. Fourth edition. 1961. Philadelphia, J. B. Lippincott Company. pp. 361-386.
- Carvel, I. and Halperin, V. Therapeutic effect of water soluble bioflavonoids in gingival inflammatory conditions. Oral Surg., Oral Med. and Oral Path. 14: 7, 847-855, July 1961.
- Cheraskin, E. Tests for detection of nutritional disturbances. Cheraskin, E. Diagnostic stomatology. 1961. New York, McGraw-Hill Book Company, Inc. pp. 208-242.
- Cheraskin, E. and Langley, L. L. Nutritional disturbances. Cheraskin, E. and Langley, L. L. Dynamics of oral diagnosis. 1956. Chicago, The Year Book Publishers, Inc. pp. 244-262.

- Cheraskin, E. and Ringsdorf, W. M., Jr. Periodontal pathosis in man: III. Effect of relatively high-protein low-refined-carbohydrate diet upon clinical tooth mobility. Ann. Dent. 22: #1, 13-18, March 1963.
- Cheraskin, E. and Ringsdorf, W. M., Jr. Diet and gingival state. New York State Dent. J. 30: #7, 275-278, August-September 1964a.
- Cheraskin, E. and Ringsdorf, W. M., Jr. Periodontal pathosis in man: VIII. Effect of protein versus placebo supplementation upon clinical tooth mobility. Periodontics. 2: #2, 69-70, March-April 1964b.
- Darby, W. J. The oral manifestations of iron deficiency. J. A. M. A. 130: #13, 830-835, March 30, 1946.
- Dubos, R. J. Mirage of health. 1959. New York, Harper and Brothers.
- El-Ashiry, G. M., Ringsdorf, W. M., Jr. and Cheraskin, E. Local and systemic influences in periodontal disease: II. Effect of prophylaxis and natural versus synthetic vitamin C upon gingivitis. J. Periodont. 35: #3, 58-67, May-June 1964a.
- El-Ashiry, G. M., Ringsdorf, W. M., Jr. and Cheraskin, E. Local and systemic influences in periodontal disease: IV. Effect of prophylaxis and natural versus synthetic vitamin C upon clinical tooth mobility. Internat. J. Vit. Res. 34: #2, 202-218, June 1964b.
- El-Ashiry, G. M., Ringsdorf, W. M., Jr. and Cheraskin, E. Local and systemic influences in periodontal disease: III. Effect of prophylaxis and natural versus synthetic vitamin C upon sulcus depth. New York J. Dent. 34: #7, 254-262, August-September 1964c.
- Fischer, B. Clinical orthodontics. 1957a. Philadelphia, W. B. Saunders Company. pp. 26-27.
- Fischer, B. Clinical orthodontics. 1957b. Philadelphia, W. B. Saunders Company. p. 157.
- Fitzgerald, M. G. and Fourman, P. An experimental study of magnesium deficiency in man. Clin. Sc. 15: #4, 635-647, November 1956.
- Follis, R. H. The inorganic elements. Follis, R. H. Deficiency diseases. 1958a. Springfield, Illinois, Charles C. Thomas, Publisher. pp. 19-82.
- Follis, R. H. Deficiency diseases. 1958b. Springfield, Illinois, Charles C. Thomas, Publisher. p. 19.
- Follis, R. H. Deficiency diseases. 1958c. Springfield, Illinois, Charles C. Thomas, Publisher. p. 20.

- Follis, R. H. Deficiency diseases. 1958d. Springfield, Illinois, Charles C. Thomas, Publisher. p. 283.
- Glickman, I. Clinical periodontology. Third edition. 1964a. Philadelphia, W. B. Saunders Company. pp. 831-832.
- Glickman, I. Clinical periodontology. Third edition. 1964b. Philadelphia, W. B. Saunders Company. p. 831.
- Goldman, H. M., and Cohen, D. W. Periodontia. Fourth edition. 1957. St. Louis, The C. V. Mosby Company. p. 431.
- Goldman, H. M., Schluger, S., Fox, L. and Cohen, D. W. Periodontal therapy. Third edition. 1964. St. Louis, The C. V. Mosby Company. pp. 662-663.
- Goodhart, R. S. How well nourished are Americans? II. National Vitamin Foundation Report for 1961-1963. New York, New York. January 1964.
- Graber, T. M. Orthodontics: principles and practice. 1961a. Philadelphia, W. B. Saunders Company. pp. 427-428; 502-505.
- Graber, T. M. Orthodontics: principles and practice. 1961b. Philadelphia, W. B. Saunders Company. pp. 225-227.
- Grant, D., Stern, I. B. and Everett, F. G. Orban's periodontics. Second edition. 1963. St. Louis, The C. V. Mosby Company. p. 502.
- Greene, J. C. and Vermillion, J. R. Oral hygiene index: a method for classifying oral hygiene status. J. A. D. A. 61: 172-179. August 1960.
- Gresham, H. A manual of orthodontics. 1957. Christchurch, New Zealand, N. M. Peryer Limited. p. 95.
- Harte, R. A., Chow, B. and Sure, B. Dietary interrelationships. Wohl, M. G. and Goodhart, R. S. Modern nutrition in health and disease. Second edition. 1960a. Philadelphia, Lea and Febiger. p. 453.
- Harte, R. A., Chow, B. and Sure, B. Dietary interrelationships. Wohl, M. G. and Goodhart, R. S. Modern nutrition in health and disease. Second edition. 1960b. Philadelphia, Lea and Febiger. p. 460.
- Harte, R. A., Chow, B. and Sure, B. Dietary interrelationships. Wohl, M. G. and Goodhart, R. S. Modern nutrition in health and disease. Second edition. 1960c. Philadelphia, Lea and Febiger. p. 459.
- Jarabak, J. R. and Fizzell, J. A. Technique and treatment with the lightwire appliances. 1963. St. Louis, The C. V. Mosby Company. p. 298.

- Kato, K. Iron-cobalt treatment of physiologic and nutritional anemia in infants. Jour. Pediat. 11: 385-396, 1937.
- Keller, S. E., Ringsdorf, W. M., Jr. and Cheraskin, E. Interplay of local and systemic influences in the periodontal diseases. I. Effect of prophylaxis and multivitamin therapy on gingivitis score. J. Periodont. 34: #3, 259-280, May 1963.
- Kerr, D. A., Ash, M. M. and Millard, H. D. Oral diagnosis. 1959. St. Louis, The C. V. Mosby Company. pp. 180-181.
- Kleiner, I. and Orten, J. M. Inorganic metabolism and water balance. Kleiner, I. and Orten, J. M. Biochemistry. Sixth edition. 1962. St. Louis, The C. V. Mosby Company. pp. 586-624.
- Kruse, H. D. The interplay of noxious agents, stress and deprivation in the engenderment of disease. Milbank Mem. Fund. Quart. 31, 93, 1953.
- May, J. M. Studies in disease ecology. 1961. New York, Hafner Publishing Company.
- McCarthy, P. L. and Shklar, G. Nutritional deficiencies and lesions of the oral mucosa. McCarthy, P. L. and Shklar, G. Diseases of the oral mucosa. 1964. New York, McGraw-Hill Book Company. pp. 236-245.
- McCoy, J. D. and Shepard, E. E. Applied orthodontics. Seventh edition. 1956. Philadelphia, Lea and Febiger. p. 186.
- Miller, S. C. Textbook of periodontia. 1950. Third edition. Philadelphia, The Blakiston Company. p. 83.
- Moore, C. V. Iron and the essential trace elements. Wohl, M. G., and Goodhart, R. S. Modern nutrition in health and disease. Second edition. 1960. Philadelphia, Lea and Febiger. pp. 234-273.
- Moyers, R. E. Handbook of orthodontics. 1958a. Chicago, The Year Book Publishers, Inc. p. 361.
- Moyers, R. E. Handbook of orthodontics. 1958b. Chicago, The Year Book Publishers, Inc. p. 128.
- Nizel, A. E. Trace elements. Nizel, A. E. Nutrition in clinical dentistry. 1960. Philadelphia, W. B. Saunders Company. pp. 122-150.
- Orban, B. J. and Wentz, F. M. Nutritional and dietary disturbances. Orban, B. J. and Wentz, F. M. Atlas of clinical pathology of the oral mucous membrane. 1955. St. Louis, The C. V. Mosby Company. pp. 105-111.

Richert, D. A. and Westerfeld, W. W. Some interrelations of dietary protein, molybdenum, riboflavin and calories on liver and intestinal xanthine oxidase. Proc. Soc. Exp. Biol. and Med. 83: #4, 726-729, August-September 1953.

Rogers, E. S. Human ecology and health. 1960. New York, The Mac-Millan Company.

Roth, H. Vitamins as an adjunct in the treatment of periodontal disease. J. A. D. A. 32: #1, 60-66, January 1945.

Roth, H., Greene, H. and Kirsch, S. Preliminary progress report of a clinical study of the effect of C. V. P. on gingival hemorrhage. Oral Surg., Oral Med. and Oral Path. 10: #6, 590-600, June 1957.

Scrimshaw, N. S. Nutritional status and infectious disease. Illinois Med. Jour. 122: #5, 466-475, November 1962.

Selye, H. The pluricausal cardiopathies. 1961. Springfield, Illinois, Charles C. Thomas, Publisher.

Snedecor, G. W. Statistical methods. Fifth edition. 1956. Ames, Iowa, The Iowa State College Press. p. 46.

Sorrin, S. The practice of periodontia. 1960. New York, McGraw-Hill Book Company, Inc. p. 104.

Strang, R. and Thompson, W. M. Textbook of orthodontia. Fourth edition. 1958a. Philadelphia, Lea and Febiger, pp. 867-868.

Strang, R. and Thompson, W. M. Textbook of orthodontia. Fourth edition. 1958b. Philadelphia, Lea and Febiger. pp. 269-271.

Stuhl, F. Vitamin C subnutrition in gingivostomatitis. Lancet 1: #21, 640-642, May 22, 1943.

Tarpley, B. W. Technique and treatment with the labio-lingual appliance. 1961. St. Louis, The C. V. Mosby Company. p. 27.

Thoma, K. H. and Goldman, H. M. Oral pathology. Fifth edition. 1960. St. Louis, The C. V. Mosby Company. pp. 542-551.

Thurow, R. C. Technique and treatment with the edgewise appliance. 1962. St. Louis, The C. V. Mosby Company. p. 20.

Tiecke, R. W., Stuteville, O. H. and Calandra, J. C. Pathologic physiology of oral disease. 1959. St. Louis, The C. V. Mosby Company. pp. 84-93.

- Vallee, B. L. The metabolic role of zinc. J. A. M. A. 162: #1, 1053-1057, November 10, 1956.
- Wade, A. B. Basic periodontology. 1960. Bristol, John Wright and Sons Ltd. pp. 52-53.
- Wellensiek, E. K. Therapeutic evaluation of bioflavonoids in periodontal disease. Texas D. J. 74: #6, 289-292, June 1956.
- White, A., Handler, P., Smith, E. L., and Stetten, D. Chemical elements in nutrition. White, A., Handler, P., Smith, E. L., and Stetten, D. Principles of biochemistry. Second edition. 1959a. New York, McGraw-Hill Book Company, Inc. pp. 991-995.
- White, A., Handler, P., Smith, E. L., and Stetten, D. Chemical elements in nutrition. White, A., Handler, P., Smith, E. L., and Stetten, D. Principles of biochemistry. Second edition. 1959b. New York, McGraw-Hill Book Company, Inc. p. 991.
- Wohl, M. C. and Goodhart, R. S. Modern nutrition in health and disease. Second edition. 1960. Philadelphia, Lea and Febiger. pp. 234-273.

APPENDIX

TABLE A
 INDIVIDUAL PATIENT DISTRIBUTION
 IN TERMS OF THE NUMBER OF CAPSULES
 CONSUMED AND THE LENGTH OF THE
 EXPERIMENTAL PERIOD

case number	placebo group		case number	multivitamin group	
	capsules consumed	experimental period		capsules consumed	experimental period
1718	56	30	1717	63	31
1724	57	30	1719	58	30
1727	51	29	1720	58	30
1729	62	30	1721	46	27
1735	55	28	1722	47	27
1739	59	35	1728	58	29
1740	58	35	1730	55	30
1741	53	28	1731	52	28
1746	58	30	1733	48	29
1749	41	32	1734	35	29
1751	37	29	1736	40	30
1753	53	29	1737	40	29
1756	49	28	1738	43	27
1757	xx	28	1743	53	28
1758	50	29	1744	xx	28
1761	63	33	1745	63	32
1766	57	29	1747	40	30
1767	60	30	1750	54	28
1768	65	29	1754	48	29
1770	65	32	1755	54	29
1777	60	29	1759	58	29
1778	55	32	1760	50	27
1779	43	29	1763	60	32
1782	33	28	1764	xx	31
1784	65	31	1765	58	29
1785	63	31	1769	42	32
1788	61	34	1775	43	33
1791	38	29	1776	58	29
1792	46	29	1786	50	34
1795	51	28	1790	65	35
1799	50	24	1793	57	28
1800	51	29	1794	50	29
1802	40	24	1797	65	34

TABLE A
(CONTINUED)

INDIVIDUAL PATIENT DISTRIBUTION
IN TERMS OF THE NUMBER OF CAPSULES
CONSUMED AND THE LENGTH OF THE
EXPERIMENTAL PERIOD

case number	placebo group		case number	multivitamin group	
	capsules consumed	experimental period		capsules consumed	experimental period
1803	30	21	1798	61	34
1804	25	15	1801	44	31
mean	51.8	29.0		52.0	29.9

note: Case numbers 1744, 1757, and 1764 failed to return the pill-box. No actual consumption record has been recorded on these patients.

TABLE B

EFFECT OF ORTHODONTIC BANDING VERSUS
NONBANDING OF THE MANDIBULAR LATERAL
INCISOR TEETH WITH AND WITHOUT
MULTIVITAMIN SUPPLEMENTATION
UPON GINGIVAL STATE

case number	<u>Group I</u> (placebo group)				case number	<u>Group II</u> (multivitamin group)			
	<u>Group Ia</u> (banded)		<u>Group Ib</u> (nonbanded)			<u>Group IIa</u> (banded)		<u>Group IIb</u> (nonbanded)	
	initial	final	initial	final		initial	final	initial	final
1718	1	1	1	1	1717	1	1	1	1
1724	2	1	2	2	1719	1	1	1	0
1727	1	2	1	1	1720	1	2	1	1
1729	1	1	1	1	1721	1	2	1	1
1735	2	1	2	1	1722	2	2	2	2
1739	0	1	1	1	1728	1	1	1	1
1740	1	1	1	1	1730	1	1	1	1
1741	1	1	1	1	1731	1	2	1	1
1746	2	2	2	2	1733	2	2	2	2
1749	1	2	1	2	1734	2	2	2	2
1751	1	1	1	1	1736	1	1	1	1
1753	1	2	1	2	1737	2	2	2	2
1756	1	1	1	2	1738	1	2	1	1
1757	1	1	1	1	1743	1	1	1	1
1758	1	1	1	1	1744	2	1	2	1
1761	1	2	1	1	1745	2	1	1	1
1766	1	1	1	1	1747	1	1	1	1
1767	1	1	1	1	1750	2	2	2	1
1768	0	1	1	0	1754	1	2	1	2
1770	1	1	1	1	1755	1	1	1	0
1777	2	1	1	1	1759	1	1	1	1
1778	1	1	1	1	1760	1	1	1	1
1779	1	1	1	1	1763	1	1	1	1
1782	1	1	1	1	1764	1	1	1	1
1784	1	1	1	0	1765	1	1	1	0
1785	1	1	1	1	1769	1	1	1	1
1788	1	1	2	1	1775	1	2	1	1
1791	1	2	1	1	1776	1	2	2	1
1792	1	1	1	1	1786	2	2	1	1
1795	1	1	1	1	1790	2	1	1	1
1799	1	1	1	1	1793	1	1	1	1
1800	1	1	2	2	1794	1	1	1	1
1802	1	1	1	1	1797	1	1	1	0

TABLE B
(CONTINUED)

EFFECT OF ORTHODONTIC BANDING VERSUS
NONBANDING OF THE MANDIBULAR LATERAL
INCISOR TEETH WITH AND WITHOUT
MULTIVITAMIN SUPPLEMENTATION
UPON GINGIVAL STATE

case number	<u>Group I</u> (placebo group)				case number	<u>Group II</u> (multivitamin group)			
	<u>Group Ia</u> (banded)		<u>Group Ib</u> (nonbanded)			<u>Group IIa</u> (banded)		<u>Group IIb</u> (nonbanded)	
	initial	final	initial	final		initial	final	initial	final
1803	1	1	1	1	1798	2	2	2	2
1804	2	2	2	1	1801	2	2	1	1
mean	1.09	1.20	1.17	1.11		1.31	1.43	1.23	1.06
S.D.	0.45	0.42	0.38	0.48		0.48	0.51	0.42	0.54
P	>0.200		=0.500			>0.200		>0.100	
percentage change ·	+10%		-5%			+9%		-14%	

TABLE C

EFFECT OF ORTHODONTIC BANDING VERSUS
NONBANDING OF THE MANDIBULAR LATERAL
INCISOR TEETH WITH AND WITHOUT
MULTIVITAMIN SUPPLEMENTATION
UPON CLINICAL TOOTH MOBILITY

case number	<u>Group I</u> (placebo group)				case number	<u>Group II</u> (multivitamin group)			
	<u>Group Ia</u> (banded)		<u>Group Ib</u> (nonbanded)			<u>Group IIa</u> (banded)		<u>Group IIb</u> (nonbanded)	
	initial	final	initial	final		initial	final	initial	final
1718	1	1	1	1	1717	1	1	1	1
1724	1	1	1	1	1719	1	1	1	0
1727	2	1	2	1	1720	1	1	1	1
1729	1	1	1	1	1721	1	1	1	1
1735	1	1	0	1	1722	1	1	1	1
1739	1	1	1	1	1728	1	1	1	0
1740	1	1	2	1	1730	1	1	1	1
1741	1	2	1	1	1731	1	1	1	1
1746	1	1	1	1	1733	1	1	2	1
1749	1	1	1	1	1734	1	1	1	0
1751	1	1	1	1	1736	1	1	1	1
1753	0	1	1	1	1737	2	1	1	1
1756	1	1	1	1	1738	1	1	1	1
1757	1	1	2	1	1743	1	1	1	0
1758	0	1	1	1	1744	1	0	1	1
1761	1	1	1	1	1745	1	1	1	1
1766	1	1	1	1	1747	1	1	1	1
1767	1	0	1	1	1750	1	0	1	1
1768	1	1	1	1	1754	1	1	1	1
1770	2	2	2	2	1755	1	1	1	1
1777	1	1	1	1	1759	1	2	2	1
1778	0	0	0	0	1760	1	1	1	1
1779	1	1	1	1	1763	1	1	1	1
1782	1	1	0	0	1764	1	1	1	1
1784	1	1	1	1	1765	0	0	0	0
1785	1	1	1	1	1769	1	1	1	1
1788	0	1	0	0	1775	1	1	1	1
1791	1	1	1	1	1776	1	0	1	1
1792	1	1	1	1	1786	1	1	1	0
1795	1	1	1	1	1790	1	1	1	0
1797	1	1	2	1	1793	0	0	1	0
1800	0	1	1	1	1794	0	0	0	0
1802	1	1	0	1	1797	0	1	1	1

TABLE C
(CONTINUED)

EFFECT OF ORTHODONTIC BANDING VERSUS
NONBANDING OF THE MANDIBULAR LATERAL
INCISOR TEETH WITH AND WITHOUT
MULTIVITAMIN SUPPLEMENTATION
UPON CLINICAL TOOTH MOBILITY

case	<u>Group I</u> (placebo group)				case	<u>Group II</u> (multivitamin group)				
	<u>Group Ia</u> (banded)		<u>Group Ib</u> (nonbanded)			<u>Group IIa</u> (banded)		<u>Group IIb</u> (nonbanded)		
	number	initial	final	initial		final	number	initial	final	initial
1803	1	1	1	1	1798	1	0	0	0	0
1804	1	1	1	0	1801	1	1	1	1	0
mean	0.91	1.00	1.00	0.91		0.91	0.83	0.97	0.69	
S.D.	0.45	0.34	0.54	0.31		0.37	0.45	0.37	0.48	
P	>0.300		>0.400			>0.400		<0.010*		
percentage change	+10%		-9%			-9%		-29%		

* statistically significant

TABLE D

EFFECT OF ORTHODONTIC BANDING VERSUS
NONBANDING OF THE MANDIBULAR LATERAL
INCISOR TEETH WITH AND WITHOUT
MULTIVITAMIN SUPPLEMENTATION
UPON LABIAL DEBRIS SCORE

case number	<u>Group I</u> (placebo group)				case number	<u>Group II</u> (multivitamin group)			
	<u>Group Ia</u> (banded)		<u>Group Ib</u> (nonbanded)			<u>Group IIa</u> (banded)		<u>Group IIb</u> (nonbanded)	
	initial	final	initial	final		initial	final	initial	final
1718	0	1	0	1	1717	2	2	1	1
1724	0	3	1	2	1719	1	2	1	1
1727	1	1	0	1	1720	1	2	1	1
1729	0	2	0	1	1721	2	3	1	1
1735	2	2	1	2	1722	1	2	1	0
1739	1	1	1	1	1728	1	2	0	1
1740	2	2	2	2	1730	1	1	0	0
1741	1	2	1	0	1731	2	0	1	0
1746	1	2	1	1	1733	0	1	2	1
1749	2	2	2	2	1734	2	1	1	1
1751	1	2	1	2	1736	1	1	2	0
1753	1	2	1	2	1737	1	2	1	2
1756	1	1	1	2	1738	1	2	1	1
1757	1	2	2	2	1743	1	1	2	2
1758	2	1	1	1	1744	1	1	1	1
1761	1	1	1	1	1745	1	2	1	0
1766	1	1	1	0	1747	0	1	1	1
1767	1	1	1	1	1750	1	2	2	1
1768	1	1	1	1	1754	1	0	0	1
1770	1	2	1	2	1755	1	2	0	1
1777	2	1	2	1	1759	1	1	0	1
1778	1	1	2	2	1760	1	1	1	1
1779	1	2	1	2	1763	1	1	1	1
1782	1	1	1	1	1764	1	1	1	0
1784	1	0	1	0	1765	1	1	1	0
1785	0	2	0	0	1769	1	1	1	1
1788	1	1	1	0	1775	1	1	1	0
1791	2	2	1	1	1776	2	2	2	2
1792	1	1	1	1	1786	2	2	1	0
1795	2	2	2	1	1790	2	1	1	0
1799	1	2	1	1	1793	1	1	1	0
1800	1	2	1	2	1794	1	0	1	1
1802	1	1	1	0	1797	1	1	1	1

TABLE D
(CONTINUED)

EFFECT OF ORTHODONTIC BANDING VERSUS
NONBANDING OF THE MANDIBULAR LATERAL
INCISOR TEETH WITH AND WITHOUT
MULTIVITAMIN SUPPLEMENTATION
UPON LABIAL DEBRIS SCORE

case number	<u>Group I</u> (placebo group)				case number	<u>Group II</u> (multivitamin group)			
	<u>Group Ia</u> (banded)		<u>Group Ib</u> (nonbanded)			<u>Group IIa</u> (banded)		<u>Group IIb</u> (nonbanded)	
	initial	final	initial	final		initial	final	initial	final
1803	1	2	1	2	1798	2	1	2	1
1804	1	2	2	1	1801	0	2	1	0
mean	1.09	1.54	1.09	1.20		1.14	1.34	1.03	0.74
S.D.	0.57	0.62	0.57	0.73		0.54	0.69	0.57	0.62
P	<0.005*		>0.100			>0.100		<0.050*	
percentage change	+39%		+10%			+18%		-28%	

* statistically significant

TABLE E

EFFECT OF ORTHODONTIC BANDING VERSUS
NONBANDING OF THE MANDIBULAR LATERAL
INCISOR TEETH WITH AND WITHOUT
MULTIVITAMIN SUPPLEMENTATION
UPON CLINICAL STATE

case number	<u>Group I</u> (placebo group)				case number	<u>Group II</u> (multivitamin group)			
	<u>Group Ia</u> (banded)		<u>Group Ib</u> (nonbanded)			<u>Group IIa</u> (banded)		<u>Group IIb</u> (nonbanded)	
	initial	final	initial	final		initial	final	initial	final
1718	2	3	2	3	1717	4	4	3	3
1724	3	5	4	5	1719	3	4	3	1
1727	4	4	3	3	1720	3	5	3	3
1729	2	4	2	3	1721	4	6	3	3
1735	5	4	3	4	1722	4	5	4	3
1739	2	3	3	3	1728	3	4	2	2
1740	4	4	5	4	1730	3	3	2	2
1741	3	5	3	2	1731	4	3	3	2
1746	4	5	4	4	1733	3	4	6	4
1749	4	5	4	5	1734	5	4	4	3
1751	3	4	3	4	1736	3	3	4	2
1753	2	5	3	5	1737	5	5	4	5
1756	3	3	3	5	1738	3	5	3	3
1757	3	4	5	4	1743	3	3	4	3
1758	3	3	3	3	1744	4	2	4	3
1761	3	4	3	3	1745	4	4	3	2
1766	3	3	3	2	1747	2	3	3	3
1767	3	2	3	3	1750	4	4	5	3
1768	2	3	3	2	1754	3	3	2	4
1770	4	5	4	5	1755	3	4	2	2
1777	5	3	4	3	1759	3	4	3	3
1778	2	2	3	3	1760	3	3	3	3
1779	3	4	3	4	1763	3	3	3	3
1782	3	3	2	2	1764	3	3	3	2
1784	3	2	3	1	1765	2	2	2	0
1785	2	4	2	2	1769	3	3	3	3
1788	2	3	3	1	1775	3	4	3	2
1791	4	5	3	3	1776	4	4	5	4
1792	3	3	3	3	1786	5	5	3	1
1795	4	4	4	3	1790	5	3	3	1
1799	3	4	4	3	1793	2	2	3	1
1800	2	4	4	5	1794	2	1	2	2
1802	3	3	2	2	1797	2	3	3	2

TABLE E
(CONTINUED)

EFFECT OF ORTHODONTIC BANDING VERSUS
NONBANDING OF THE MANDIBULAR LATERAL
INCISOR TEETH WITH AND WITHOUT
MULTIVITAMIN SUPPLEMENTATION
UPON CLINICAL STATE

case number	<u>Group I</u> (placebo group)				case number	<u>Group II</u> (multivitamin group)			
	<u>Group Ia</u> (banded)		<u>Group Ib</u> (nonbanded)			<u>Group IIa</u> (banded)		<u>Group IIb</u> (nonbanded)	
	initial	final	initial	final		initial	final	initial	final
1803	4	5	5	2	1798	5	3	4	3
1804	3	4	3	4	1801	3	5	3	1
mean	1.03	1.25	1.09	1.08		1.12	1.20	1.08	0.83
S.D.	0.49	0.52	0.50	0.55		0.49	0.61	0.47	0.69
P	<0.005*		=0.500			>0.300		<0.005*	
percentage change	+21%		-1%			+7%		-23%	

* statistically significant