On Stomatitis Ulcerosa; Etiology and Therapeutics.

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PREFACE.

Before proceeding to give an account of my investigations many kind thoughts go to those who have assisted me in my work. My thanks are first and foremost due to Docent ERIK ADLERCREUTZ who has promoted my interest in this theme and who has assisted me in working out the plans for my work.

I am greatly indebted to Docent PAAVO VARA and Med. lic. ERKKI RISSANEN who, with never ceasing interest and without counting upon their valuable time, have helped me with advice in incorporating the various details into the programme of investigation and who have kindly arranged for places for my patients. I thank especially the latter who has assisted me in examining the technical details. I feel deeply grateful to all those who have sent me material for examination and who have given me valuable information.

Due to the prevailing circumstances I have not had access to any literature, which is very regrettable. English literature has not been available at all, and on this account the chapter on therapeutics with sulfonamides is very incomplete.

Helsingfors in November 1944.

The Author.

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Introduction and Questions.

A people, having to fight through difficult times such as at present predominant in many countries, is often subjected to diseases. External circumstances may be reflected in the frequency of the disease and, above all, certain pathological conditions may be noticed which, as might be said, follow in the wake of war. Accordingly, a plausible explanation may be given the wave of Stomatitis ulcerosa prevalent at the moment not only in the armies of the nations at war but also — although in a lesser degree — among civilians.

In this work I have aimed at finding an answer to the question: which is the agent or agents that have given rise to the present striking increase of Stom. ulc. especially among soldiers and persons on duty in the field. My investigations have been based partly upon thorough internal medical examinations, partly upon the frequency of the disease during different seasons. The material has been collected among persons in field-work and special attention has been given the statistics and a foundation as homogeneous as possible has been laid by the aid of probability-calculus.

Another task was to test a combined form of treatment in which the local therapeutics consist of a thorough care of the set of teeth and removal of decayed parts with a 10 per cent solution of chromium acid. The patients were treated internally with sulfonamide-preparations; a rest-cure and a more varied diet were arranged for. On account of these therapeutical measures the results obtained with local treatment were taken into consideration on the one hand; on the other hand they served the purpose of creating a hygienic state of the mouth cavity. But at the same time my aim was to support and accelarate the process of cure by a sulfonamide cure and to increase the power of resistance in the organism by temporarily improved conditions of life.

Survey of Historical Literature.

Before proceeding to the actual investigations I wish to give the reader an idea of our present knowledge of the appearance of ulcerous inflammation of the gums and the etiology and therapeutics thereof. SEIMBILLE gives us some valuable information on the prevalence of Stom. ulc. during various periods of times past. According to SEIMBILLE, BERGERON was able to demonstrate a malign inflammation of the gums among soldiers in the French army in Italy, in 1793, and it seems as if it should have spread further by the amassment of people. We must also take into consideration that deaths occurred almost entirely within the army and the civilians were spared. It seems as if the sailors in the French navy were more resistive, as, according to SEIMBILLE, they were immune to the contagion in question until 1872. In America again, according to ALBROY, PLANT and VINCENT, 1894 and 1896, give a detailed picture of the disease which resembles the present clinical conception of Stom. ulc. while other sources mention that the disease was not noticed in America until 1897. It is quite probable that it remained very rare in U.S.A. until 1914 when the frequency of the morbidity increased strikingly as soldiers returned from the great battle-fields in Europe. It is generally known that ulcerous inflammation of the gums was greatly spread among the military who had been posted in the trenches on the Western front for some time. Among the British the disease was referred to as "Trench mouth" and the Germans used the word "Schützengrabenmund". According to the unanimous opinions of a number of authors, NEWGARDEN, WITT and others, the disease is said to have become epidemic. After the war many experts in Germany took measures to prevent the disease and about 1920 they succeeded in obtaining an indisputable success by using a great number of therapeutical methods. On the other hand, ALBROY, BONNEY, MINER, WRIGHT and others confirm that "Trench mouth" — or "Vincent's infection", as it was generally called in the English speaking world — was extensively spread in England and America and that it now occurs both epidemically and endemically notwithstanding very particular care of the mouth cavity. After the war and post-war summit having been reached a period followed in which Stom. ulc. did not appear to any extent in Europe, whereas it was still quite common in the Union of Soviet in 1929 (WELIKANOWA).

HENTZE mentions a wave of Stom. ulc. in Haifa (Palestine) in 1932. Children between the age of 2 and 4 were greatly affected by the disease. The Italians in Abyssinia also suffered from Stom. ulc. (MATHIS). SHAW gives a rather surprising information of natives in Africa having been subjected to the disease. In the beginning of the World War in 1939 the frequency of the ulcerous stomatitis increased again. TRETTER says that the disease was largely prevalent among the German soldiers as early as in the campaign in Poland. UNGLEY and HORTON have given reports on the results of their recent investigations within the English navy. GROSS, STURM and VÖCKLINGHAUS, among others, supply information as to the spread of the disease on the Eastern front. An increase of the frequency of the morbidity was clearly stated among the Finnish troops and according to information received from Sweden they have to battle against the disease there too.

The descriptions above may perhaps impart the idea that men only are subjected to Stom. ulc. This is not the case, however, although certain authors assert that soldiers especially show a great susceptibility to the disease. MORELLI writes that ulcerous inflammation of the gums, according to statistics, occurs three times as often among men as among women, while KLEIN and DALEV do not share MORELLI's opinion and state that the disease occurs among both sexes.

It has been stated that certain ages are particularly susceptible. KLEIN says that children between the age of 2 and 10 and young men and women of 18—25 are specially disposed to the disease. DALEV, again, gives an age-limit of 20—50 years. Yet he considers that an increased predisposition may be stated in women of the age of 20—30 and men of 30—40. This opinion is partly shared by ALBROY although he gives a far greater age limit, i. e. 2—70 years. Even little children may be affected by Stom. ulc. — as gathered when the epidemy in Haifa was mentioned.

It is further evident that the number of cases fluctuate according to the seasons. DALEV, for instance, points out that he has been able to state an increase in the frequency during the first quarter of the year. MORELLI, too, states an increase in March and April and in the middle of summer. The author explains that the summit of the disease reached in spring is due to a reduced power of resistance owing to chills, while the high point in summer is caused by intestinal and stomachal disturbances. The greater frequency of the morbidity in men may be due to overstraining.

It cannot be stated, however, according to the author mentioned, that the increase in spring is due to lack of avitaminosis during the winter. NEHSE says, on the contrary, that he seems to have noticed an increased frequency during the transitionary seasons i. e. autumn to winter, winter to spring.

An interesting observation which should not be ignored in this connection and which may be of a certain etiological importance is the fact that Stom. ulc. has never been stated in a toothless mouth. ALBROY, v. MIKULICZ-KÜMMEL, KIVIMÄKI and others stress this fact.

The clinical picture is no doubt well-known to everyone but a repetition of the most important and remarkable symptoms may not, perhaps, be out of place. At the onset the patient notices a certain reddishness in the gingival which simultaneously feels taut. The papillaries swell and become bluish-red in colour while the other parts of the gums are very tender and are easily caused to bleed. At this stage lack of appetite, increased salivation and a temperature are noticeable in certain cases. The tenderness of the gingival causes a reluctance in the patient to clean the mouth whereby decaying particles of food and lack of mouth hygiene causes a strong Foetor. As the disease develops, pseudomembraneous formations on the papillaries arise which spread to the gingival — in far developed cases as far as to the mucous membrane of the cheek. The membranes are grey or greenish-grey in colour and when removed a sensitive, bleeding, and sharply demarcated surface remains which has the effect of being impressed (KLEIN). The tongue may swell and the regional lymphatic glands may become enlarged and tender. As the papillaries are converted into one necrotic mass the neck of the tooth is left bare which causes pain at the slightest touch — even such intense pain that the patient has a difficulty in taking food. In a highly developed stage a resorbance of the bone tissue is noticed with the natural consequence that the teeth lose their firmness and become loose.

A closer study of the various forms in which Stom. ulc. appears shows, in accordance with ZEMSKY and NEWGARDEN that they may be divided into the following groups: acute, subacute and chronic. STANLEY and MOORE are not of the same opinion and mention only an acute and a chronic state, while SEIMBILLE never considered himself able to diagnosticate a chronic stage.

LILLY agrees partly with ZEMSKY's grouping — he describes a gingival, a peridental, and a periostal form, while BONNET-ROY speaks of acute, febrile, torpid, and subacute forms. The opinions of all these authors show a certain connection, while for instance ROSENTHAL's classification is somewhat diverging in that he divides the forms of appearance into a primary "Vincent's infection" with symptoms in the surface tissues of the mouth or throat; a secondary stage, when the disease has reached the deep tissues and finally a third phase in which the disease has invaded other regions of the body, such as for instance the lungs. Finally, WRIGHT points out that only "one positive and one active-positive form" may be stated.

Etiology.

When proceeding to make clear the various causes of Stom. ulc. LILLY's fundamental plan will be followed in principle. According to LILLY the etiological factors may be divided into the following two principal groups:

1) predisposed and
2) existing causes.

The former group may again be divided into two subgroups: a) local, and b) general causes.

The local factors may be of different kinds. Some authors, v. MIKULICZ-KÜMMEL, NEWGARDEN, HIRSCHFELD, FABER, LILLY, KÖHLER and KIVIMÄKI, consider that mouths in which there are remains of roots, carious teeth, defective crowns, bridges and stoppings and badly fitting artificial teeth represent the primary causes of the suffering. Physiological irritations of chronic nature, such as tartar (SEGELCKE, v. MIKULICZ-KÜMMEL, LILLY), and pockets in the gums must be taken into consideration. The pockets seem, according to a number of experts, to play a dominating rôle, probably even a decisive rôle in the origin of the disease (BONNET-ROY, ALBROY, NEWGARDEN, SEIMBILLE, KIVIMÄKI, and others). For instance, CATELAN (quot. SEIMBILLE) points out, as early as in 1877, that Stom. ulc. may be a local infection of nervous origin caused by the penetration of the wisdom tooth. Similar observations were made by HEYDENREICH (quot. SEIMBILLE) one year later. CORIOT treats this etiological factor more thoroughly and endeavours to explain the mechanism thereof as follows: the cutting of the wisdom tooth causes a

"Quetschung" of the mucous membrane followed by an infection spreading especially along the lymphal ways. His opinion is founded upon statistics from a hospital with on an average 50 cases a year. 94—96 per cent of the illnesses were of "dental origin". A further proof of the accuracy of this theory has been given by LEBEDINSKY (quot. CARIOT); in bilateral processes he has been able to cure the one side by extracting the wisdom tooth on this side while the infection continued on the side which was not operated. A complete cure was not obtained until the remaining wisdom tooth had been extracted; relapses did not occur. However important this cause may be we must not allow it to be decisive — there may be other agents of the disease. Local causes may probably comprise unclean tooth-brushes, dishes, towels, pipes, pencils, and kissing (LILLY and others). HIRSCHFELD speaks of inferior cigarettes being of great importance in the causal connection. KÖHLER and FABER are decidedly opposed to the above mentioned authors — they consider that no independent, so called primary stomatitis can be stated. If the disease has arisen from local irritations it is called Stomakace and, according to these authors, there is no connection between it and a real stomatitis, the origin of which must lie much deeper.

KÖHLER's and FABER's opinions take us over to the second group of predisposed causes, i. e. those of a general nature. LUFKIN and DISRAELI point out in their work that the question must so far be left unanswered whether the disease should be considered to be of primary or secondary infection originating in organs which have lowered the power of resistance of the cellular tissue. TRETTER, MORELLI, MATHIS, FABER, and others, again point out that the etiological form of the question comprises a collaboration of several factors such as a bacteriological flora and a disposition due to various causes. In other words, the equilibrium between the flora of the mouth and the mucous membrane of the mouth has been disturbed. The reduced power of resistance may either be local or general — the former should be considered the important one. It would not, however, be in accordance with the importance of the question, to ignore the disposing factors of general nature even if their pathogenetic importance represents varied values. As such are quoted primarily: severe general illness, disturbance of the liver (VERONESE), lues (LILLY), malaria (WELIKANOWA), affection of the blood-forming vessels (KÖHLER, HENTZE), diabetes (BONNET-ROY, v. MIKULICZ-KÜMMEL, HENTZE,

BONNEY, KIVIMÄKI, VÖCKLINGHAUS, WEINBERGER), mercurial or uraemic intoxication (BONNET-ROY, LILLY, KLEIN), rhinitis, affection of the subsidiary cavities (WEINBERGER) and pregnancy (MATHIS, STIEBERT-SCHNEIDER). WEINBERGER gives a most interesting interpretation of the mechanism of the origin of the disease: "Bei den Formen der Stom. ulc. die mit gastro-intestinalen Störungen einhergehen, haben wir den Eindruck, dass es sich hierbei vielfach um Eliminationsstomatiten handelt. Es käme hier nach dieser Auffassung zu einer Ausscheidung krankhafter Stoffwechselprodukte durch die Mundschleimhaut, zu einer entzündlichen Alteration derselben mit konsekutiver Keimbesiedelung führend. In manchen Fällen, nämlich bei sensibilisierten Individuen, kann sowohl die Ausscheidung biogener Amine aus den Gingivalkapillaren (SCHWARTZ) zu allergischen Manifestationen im Bereiche der Mundschleimhaut führen."

Other factors of general nature which predispose the patient to the disease are lack of general hygiene (VÖCKLINGHAUS, v. MIKULICZ-KÜMMEL and LILLY), fatiguing marches, living in crowded, dark quarters, and radical changes in the mode of living (KÖHLER and MATHIS). Finally, Stom. ulc. may in certain cases be considered a hyperergic inflammation (in which the allergies are uncooked milk and milk products) which is emphatically stressed by STURM, inter alia.

An etiological factor of ulcerated inflammation of the gums often mentioned in the literature is the lack of certain vitamines. TRETTER considers that a certain want of the active vitamine of the mesenchyme and lack of C-vitamines are probable causes. STURM, STANLEY, PRINZ, KRASNOGORSKI, JELINEK and HENTZE share his conception while DANZIGER and FABER consider a lack of A and B vitamines to be the cause of the disease. Results obtained by investigations in latter years have awaked a doubt as to this theory which is proved by investigations carried out by GROSS, UNGLEYS and HORTONS and MATHIS.

As the predisposing factors of ulcerous stomatitis have now been examined the "existing" causes will be closer looked into in the following. We have, in the first place, bacteriological agents, and in these a great number of authors see the primary factor, the origin of the evil. We find — after the discovery of Spirocheta Vincenti by VINCENT and after he has proved that this disease, in connection with Bacillus fusiformis, is always present in Stom. ulc. — the whole of the American and English group of authors

accept and defend the statement mentioned. BARRET, COBE-MARSHALL-GRACE, HIRSCHFELD, KATZ-CHAPTER, LUFKIN-DISRAELI, MINER, NEWGARDEN, PRINZ, REICHMANN, STANLEY, WRIGHT, ZEMSKY and others belong to this group. Authorities on the Continent, too, i. e. BONNET-ROY, DIMAS-ARUTI, FABER, JELINEK, LILLY, SEIMBILLE, and ROCHETTE are quite of the same opinion.

In this connection I wish to say a few words on the actual bacteriological questions. After VINCENT having laid out his theories on the symbiosis between Spirocheta Vincenti and Bacillus fusiformis as the agent of Stom. ulc. some authors have tried to find an answer to the question why the mucous membrane in certain stages reacts to these constant quests of the cavity of the mouth. The reduced power of resistance of the tissues should, in this case, play a decisive rôle and not an increased active pathogenity in the bacteria (MORELLI, CORIOT, GRYTHE, TRETTER, and others), but the mechanism itself has not been solved hereby. CARPENTER (quot. GRYTHE) stated in 1:27 a certain pathogenity in a mixture of spirochetes and streptococci which, with an addition of fusiformed staves show pathogenity. GINS (quot. GRYTHE) stresses that spirochetes and fusiforms become pathogenetic anaerobinosis. ZINZERLING, too (quot. TRETTER) states that pure cultivated spirochetes are not pathogenetic; they may give rise to diseases only in symbiosis with bacteria which possess a protheolytic capacity (Fusiforms). This is quite in accordance with VINCENT's and REICHE's opinions that the spirilles increase the virulence only, but not in accordance with GREENBERG who sees the truly dangerous microbes in the spirilles. ALBROY stated, in 1929, that the fuso-spirillary symbiosis becomes pathogenetic in connection with various cocci, the latter playing the agressive part, i. e. they penetrate the tissues, while the other two micro-organisms produce gangrene, a characteristic of the disease. MOORE, LEHMANN, WITT, GRYTHE, MATHIS and IMMING are principally of the same opinion. IMMING even goes so far as to say that the primary cause of the disease is found in "inspecific" cocci and states that the symbiosis spirilles-fusiforms representing the agents of Stom. ulc. has not yet been fully proved — anyhow not as regards the chronic stage.

This question has now been made clear thanks to KLEIN'S excellent investigations in 1943. The author presents his results in regard to the importance of combinations of bacteria in the

following words: "Bacteriologically the fusiform bacilli and Trepanoma Vincenti (earlier Spirocheta Vincenti) are preponderant. Trepanoma micro-organisms are present, too, but the picture of the fusiform bacilli and Trep. Vincenti is so typical that the name of the disease has been derived therefrom.

"In histo-pathological cuts from the typical ulcerating, progressive form, three sharply demarcated zones may be observed: the extreme, necrotic zone, consisting of a dead tissue with masses of all kinds of bacteria; the middle zone, that is but partly dead, with fusiform bacteria, in preponderance which often occur in palisade-formations and in enormous quantities; in the third zone, which is situated close to the healthy tissue, the spirochetes are almost in sole control but besides them there is nothing diseased in this zone. The fact that the spirochetes conduct the infection is considered by ZINZERLING a satisfactory proof of the spirochetes being the most active factor in the anaerobic symbiosis between fusiform bacilli and spirochetes." He summarizes his final statement as follows: "It is temptingly close at hand to suppose that the spirochetes possess a certain causal importance in this connection". This opinion conforms in many instances to the conception prevalent previously and which we have already shared. The results of these investigations are a solution to the bacteriological side of the great etiological complex of questions which gives the micro-organisms a prominent place in the causal connection.

In this connection I also wish to draw attention to an observation by some authors regarding Stom. ulc. SEIMBILLE informs us that DUBOIS, in 1921, found the stomatitis among the soldiers of the French army in Italy in 1793, described by BERGERON, to be identical with Angina Plaut-Vincenti. After him, authorities such as WEINBERGER, MORELLI, GRYTHE, TRETTER, FABER and KLEIN have become supporters of the conception of the identity of the disease in question. CORIOT goes closer into the matter and believes, on account of similar clinical, pathologic-anatomical and bacteriological symptoms, in a similarity between Stom. ulc. and the mentioned angina which he declares, on the grounds below, to be the same affection with a different modality:

1) Persons of the age of 18—30 years, living in poor conditions and circumstances, sicken very frequently.

2) Both affections start "insidiously" with a feeling of unpleasantness when chewing and swallowing.

3) Simular symptoms. Slight excitement in the general condition, lesions often rather one-sided.

4) Benignancy a rule; relapses frequent.

5) From pathologic-anatomical point of view two stages may be distinguished: the characteristic pseudo-membraneous introduction which is followed by the ulcerous period.

6) Bacteriologically the fuso-spirillar symbiosis is characteristic.

Therapeutics.

If one wishes to discover some connected principles in the chaos of therapeutics found in the literature difficulties will arise from the very beginning. No principal line for the treatment of Stom. ulc. can be found as is evident from the fact that about 50 different treatments (FLIEGE and HEUSER) were suggested between 1927 and 1937. The fact remains that nobody has been able to discover a specific therapeutic up to the present. In many cases it seems as if the treatment had not been sufficiently causal. The clinical picture has been allowed to play too predominant a rôle and results have not been satisfactory, whereas the results might have been more successful if greater importance had been attached to the causative agent of the disease. The impression of groping about and old dogmas being clung to cannot be avoided; in a few cases they have been useful. For this reason many modern authors stress the importance of the therapeutics having to subordinate the etiology in principle and yet be dependent thereof.

The therapeutic methods may be divided into three main groups, i. e. 1) local treatment only, 2) internal treatment, and 3) combined local and general treatment. If we study the local treatments more closely we find among them a few traits in common. The most natural treatment or the one that we first think of is rinsing the mouth cavity which is advized by a number of authors among which may be mentioned STANLEY, WITT, WEISSFLOG, WASSMUND (quot. IMMING), GOLOB (quot. HENTZE), ZEMSKY and PINDBORG. The following therapeutics have been used: boracic acid, various tinctures, hydrogen peroxide, potassium chloride, potassium permanganate, borate of sodium, (favoured by American authors), rivanol, chloramine etc. Closely connected with these treatments we may consider painting with salicylic acid, iodine, trypaflavin, tannin, pyocyanas (extract of

bacteria of Bac. pyocyaneus) (SCHWANK) etc. Authorities, such as FLIEGE, HEUSER and v. RECKOW suggest that the surface of the sore should be sprinkled with an antiseptic powder, for instance yatren, iodoform, salvarsan or urinosal. Salvarsan has been much used for the treatment of ulcerous inflammation of the gums, in preference in connection with gingival packings, i. e. the intradental spaces. This is made undoubtedly clear from statements by many investigators (RYGGE, BONNEY, ROSENTHAL, LUFKIN-DISRAELI, KATZ-CHAPTER, WITT). These investigators apply a salvarsan-glycerine paste to the gingival. The physic mentioned is not the only one used as packings. We find the following mentioned in the literature: borate of sodium (TAVLIN, ROSENTHAL), boracic acid (GOTTLIEB), camillosept (SEGELCKE) camillosan cream and iodoform powder (WITT), Lacalut (an astringent mouth powder of neutral chemical reaction) (SUGAR), trypaflavin and rivanol paste (WEISSFLOG), and sulphate of zinc (KIVIMÄKI). A general treatment is corrosion of the necrotic parts which may be carried out with various chemicals such as $ZnCl_2$ (BARRET) Argent. nitric., Acid trichloracetic., Kal. bichromic., Argyrol (NEWGARDEN) or Acid. chromic. (WRIGHT, LUFKIN-DISRAELI, ZEMSKY). Notwithstanding good results having been obtained by these methods some warning voices have been raised. ALBROY, FABER, and KIVIMÄKI consider that corroding remedies should not be used in acute stages, but possibly while the process is abating. A few examples of exceptional methods given in the literature may be mentioned here: cauterization (FLIEGE—v. RECHOW) and freezing of the decayed parts with ethyl of chloride (TARSOVATZ).

Of extreme importance in Stom. ulc. — the therapeutics may be local, internal or combined internal-local — is of course the hygiene of the mouth cavity. Yet it must be remembered, which is also emphatically stressed by many authorities (RYGGE, SEGELCKE, FLIEGE-HEUSER, WITT, WASSMUND, ROY (quot. IMMING), FABER, KIVIMÄKI and BORGMANN), that extractions and thorough removal of tartar and other necessary care must be left until the acute stage has been overcome, after which a thorough cleansing is essential.

In exclusively internal treatment primary importance was attached to introducing antiseptics into the organisms which is evident from articles written by NEWGARDEN, GINESTEDT and CASTERMAN and DELPIANO (quot. HENTZE), REICHMANN and

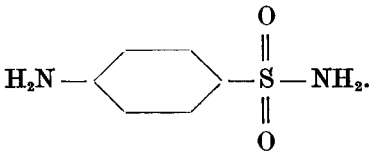
TIETZE, and others. These authors suggest As, Hg or Bi injections. FLIEGE and v. RECKOW recommend KCl, KJ or Urotropin per os. ROCHETTE maintains that neosalvarsan injections are ineffective as a rule, an opinion that has been supported by a few authors; among them is KIVIMÄKI. At present the principal interest lies in a strengthening, and in some cases in an anti-scorbutic therapeutic. This is evident from the works of SHAW, KRASNOGORSKI, FEILS and RÖSSING, among others. The last mentioned author has been able to show good healing with amide of nicotine acid (B). WILHELMI and BLOESSER (quot. IMMING) find a positive solution to the therapeutical problem in an alkalization of the organism while RYGGE, STANLEY, GRYTHE and INGENDAAY attach importance to the hereditary resistance which may be made absolute by vaccination with therapeutical or prophylactic vaccine. In 1927 LUKOMSKI published a treatment diverging greatly from that of other authors. By using Pilocarpin he says that he was able to bring on a strong flow of alkalified saliva which should have an antiseptic effect. WEBER and HERMANSDÖRFER (quot. HINTZE) contradict this statement. They maintain "dass die Pufferung des Speichels eine Vernichtung der pathogenen Mikroben durch Verschiebung des Aciditätsgrades ihres Nährbodens nicht erlaubt", and furthermore consider that an acid reaction is physiological in inflamed areas. According to PATTISON (quot. HINTZE) the calcium content in the saliva affects the power of resistance of the mucous membrane of the mouth and on that account he suggests a diet rich in vitamines soluble in fat.

Of the three different kinds of treatment there still remains the combined treatment which has gained many supporters in present times. It is very difficult to distinguish between any special lines of treatment in this group excepting, perhaps, the American school of salvarsan packings (perhaps some corroding) combined with injections of the same As-compound (ALBROY, LILLY, KLEIN, BONNET-ROY, and others), and often in connection with measures for improving the power of resistance (THOMPSON and HIRSCHFELD). Many authorities, especially those who attach importance to vitamine treatment, consider a general strengthening of the power of resistance to be the most important therapeutical factor. Certain authors point out the necessity of informing patients of the beneficial influence of rest and hygiene upon the course of the disease, SEIMBILLE, HENTZE, MORELLI, KÖHLER, MATHIS, TRETTÉR, v. POLANYI, FABER and TAKAHASI

(quot. IMMING), of course in connection with a very particular local treatment. WEINBERGER even says that he should like to introduce a complete "cleansing" of the digestive organs after a local treatment of painting with 2 per cent Pyoktanin. STIEBERT and SCHNEIDER, finally, give a therapeutic for combating Stom. ulc. originating from pregnancy or hormone disturbances. They used a preparation of follicle hormones (Progynon) but did not obtain results that were as good as such obtained with other therapeutics.

Before proceeding to the most up to date forms of treatment of Stom. ulc. in which some sulfonamide preparations are used, their chemical composition and mechanism of effect will be explained in a few words.

After ROBERT KOCH (1881) having been unsuccessful in his trials to cure infectious diseases with "internal antiseptics", and BEHRING, 1890, having shown that such remedies were much more poisonous to the organism than to the bacteria, the idea of chemotherapeutics was placed in the background for some time to give precedence to serum therapeutics. In the beginning of the 20th century EHRLICH took up the idea again for retrial and by his discovery of salvarsan he laid the foundation to the chemotherapeutics of the future. Very small progress was made in this province right up to 1935. Then DOMAGH was able to cure streptococci infections by oral or parental addition of azo dyes (Prontosil). As the azo-dye-combinations, introduced by DOMAGH, proved to possess a reliable effect in vivo while they were completely inactive in tests in vitro, tests were ardently undertaken for the purpose of solving the problem of the effective principle. The French investigators TREFOUEL, NITTI and BOVET (quot. v. OSTEN) finally succeeded in determining that the chemotherapeutical effect of Prontosil is derived from p-aminobenzoesulonamid



The mechanism of effect of sulfonamides is now, based upon FILDES', JENSEN's and SCHMIDT's (quot. KEIDING), 1941, completed investigations, considered to act as follows: The process

of metabolism of bacteria cannot take place unless certain enzymes are present. Like all enzymes, they consist of proteinic (Apo-enzymes) and certain substances of other kinds (Co-enzymes). These groups, separately, have no enzymic effect but when conditions are optimal they unite into biologically effective symplexes (enzymes.) Such enzymes, necessary for the metabolism of bacteria, contain acid of p-aminobenzoe as Co-enzymes. The bacteria themselves contain proteinic matter which is an elemental body. The process is illustrated by the following scheme:

$$\underset{\text{(Apo-enzyme)}}{\text{Proteinic matter}} + \underset{\text{(Co-enzyme)}}{\text{acid of p-aminobenzoe}} \rightleftarrows \underset{\text{(Enzyme)}}{\text{Symplex}}$$

In this chemical process the sulfonamide (which, like the acid of p-aminobenzoe is a p-aminoderivate of benzol) may take the place of the acid of bezoe. If so happens, the enzymes that the bacteria necessarily require are not formed, but a compound of sulfonamide-proteinic substance which has no enzymic quality is formed, and simultaneously the chances have decreased for the development of the bacteria.

In this connection it should still be mentioned that it is not only acid of p-aminobenzoe that serves as a checking substance; other acids related to the acid mentioned have shown similar qualities, for instance novocaine (diethylaminoethylester of acid of aminobenzoe). In practice these may denote a certain sulfonamidic resistance in the bacteria in tissues infiltrated with novocaine (STAUB).

The above mentioned theory on the effect of mechanism tells us, too, that the therapeutical doses of a sulfonamide preparation must be large enough abortive (therapeutics) to be able to eliminate the effect of the acid of p-aminobenzoe otherwise the process, in accordance with the law of mass action, continues into forming enzymes.

With this background in mind the conception is understandable that in sulfonamide treatment the therapeutics may be started with the most effective preparations although the etiological bacterium has not been stated.

As soon as the superiority of sulfonamide therapeutics in infectious diseases have been accepted they will be brought into use in odontology for treatment of Stom. ulc. MORGEN informs us, as early as 1937, that he used Prontosil locally with success instead of neosalvarsan packings. TRETTER, OCHMANN, REY, ERICSON and

HOLST join the supporters of terminal sulfonamide therapeutics; the last mentioned author gives dentists a warning against the use of sulfonamide preparations orally unless supervised by a physician on account of its toxic secondary effect. BORGMANN, again, considers the effectiveness of sulfonamides probably overrated.

During latter years sulfonamide preparations are being used more and more both orally and parentally for the cure of ulcerous inflammation of the gums. SCHRANZ administered Prontosil intravenously as early as in 1937, but the method did not become popular until about 1940, and is often combined with local treatments with the same preparation (FERNEX, HESS). IMMING and v. OSTEN consider the local therapeutics unnecessary and concentrate entirely upon an internal treatment. IMMING says that Stom. ulc. must be considered a general infectious disease and on this account the treatment must be internal. He uses the typical abortive therapeutics with the derivate of pyrimidine, Pyrimal, while v. OSTEN, on the same grounds, carried out investigations with sulfethylthiodiazol (Globucide). Both these authors are astounded by the extraordinarily successful results. ADLERCREUTZ, in 1942, started treatments of Stom. ulc. with sulfatiazol internally and obtained excellent results. These treatments must be considered pioneer work in this sphere in Finland.

Sulfonamide preparations are, however, apt to produce complications, which should be noted. At the same time as the medicine destroys the cells of the bacteria it may be injurious to the cellular function of the host. The injury, however, proved to be quite insignificant and subclinical if the correct doses were administered (NISSEN). When clinical symptoms arise it must be taken into consideration that they may not be due entirely to the chemical preparation; an abnormal reaction in the organism may often play a dominating rôle in the case. Complications may arise both on account of lengthy and too large administrations, correct and recognized dosing, and with treatment with small doses.

NISSEN and KORHONEN give a clear picture of the most important complications in their paper: i. e. cyanosis, vomiting, nervous state and neuritis, exanthemes, rise of temperature, anemia, leucopeny and aggranulocytosis, haematuria, anuria and blood-urea. Further, acidosis, toxic hepatitis, porphyria and stomatitis. The last mentioned toxic secondary effect seems to be somewhat

paradoxal but may, however, produce a secondary inflammation of the gums, i. e. after an injury to the blood forming organs has been caused or uremic intoxication has arisen.

Although it does not lie within the frame of this work to go closer into the complications it should, however, be pointed out that although they are generally very slight and easily cured, some very malign cases have been observed, cases that have even led to death. It has been noticed that the various preparations possess different toxic qualities. This is a reminder to dentists never to prescribe a sulfonamide cure unless a physician is consulted and the patient is placed in his care, and to observe moderation when using these kinds of medicines (MERKELBACH).

Personal Investigations.

Frequency during Different Seasons.

The first question to be considered in my investigations was DALEY'S and MORELLI'S statements that Stom. ulc. shows a fluctuating frequency of the morbidity in different seasons. As these authors have given somewhat diverging information I wished to find out whether such a frequency could be observed in the material at my disposal and if the etiological question could be solved by such an investigation. On this account I collected monthly statements of Stom. ulc. cases from two detachments in the front lines, let us call them X and Y. The cases were diagnosed by physicians who kindly promised to assist me and to follow my instructions. Monthly reports of the total strength of the units were obtained. The investigations were carried out during one year. The results from both detachments are found in Table I (column 1 and 2); column 3 gives their total value.

The results were combined for the purpose of affording the total foundation a wider basis and at the same time a greater homogeneity. The uniformity is also stressed by the fact that the persons are very much of same age, all conscripts. It must be observed, however, that these cases should not be considered an *absolute* frequency of the morbidity though it may be presupposed with all probability that a rise in the frequency must be noticed also in an increase of the number of visits to the detachement physician.

Table I.

Month	x	Case	y	Case	x + y	Case	Per cent per month
VI	2790	6	2610	18	5400	24	0.44 ± 0.09 %
VII	2740	10	2647	16	5387	26	0.48 ± 0.10 %
VIII....	2938	10	2858	29	5796	39	0.67 ± 0.11 %
IX	2845	20	2899	31	5744	51	0.89 ± 0.12 %
X	3002	17	3103	20	6105	37	0.61 ± 0.10 %
XI	2993	26	3034	21	6027	47	0.78 ± 0.12 %
XII	2981	27	3113	13	6094	40	0.66 ± 0.11 %
I	2941	20	3090	4	6031	24	0.40 ± 0.08 %
II	2946	11	2976	4	5922	15	0.25 ± 0.07 %
III	3001	13	2938	1	5939	14	0.24 ± 0.06 %
IV	3413	14	3464	18	6877	32	0.47 ± 0.08 %
V	3419	19	3445	17	6864	36	0.52 ± 0.08 %

[1]) 0.89 ± 0.12 %
0.78 ± 0.12 %
Diff. 0.11 ± 0.14 %
(1 × 0.14 = 0.14)

[2]) 0.89 ± 0.12 %
0.67 ± 0.11 %
Diff. 0.22 ± 0.14 %
(2 × 0.14 = 0.28)

[3]) 0.89 ± 0.12 %
0.48 ± 1.10 %
Diff. 0.41 ± 0.14 %
(3 × 0.14 = 0.42)

If we work out the percentage of frequency during the different months, Table I, column 4, a single per centage figure will not suffice. It would not stand a grave statistical criticism as it should be remembered that the information comes from different people and a certain inaccuracy in the percentage is possible. Such premisses oblige us to increase the exactitude by the aid afforded by probability calculus. In latter years the importance of such a procedure has been more and more realized in quantitative investigations, and in medical literature we have numerous examples of how completely misleading just one average or one per centage value may be. To be able, on the whole, to judge a material statistically we must be acquainted with the nature, size, way of collecting, and especially of the homogeneousness thereof and then only, by the aid of some statistical method, we may draw our conclusions.

In the investigations at hand I have, to begin with, worked out the percentage of the frequency per month, based upon diagnosed cases in the actual strength of the unity. (Table I, column 4.) The mean errors of the percentage, according to RITALA'S formula

$$\varepsilon(p) = \pm \frac{\sqrt{pq}}{n}$$

have been taken into consideration in which ε is the notation generally used in mathematical statistics for mean errors. ε (p)

denotes the mean error of p, which is the statistical percentage. In the formula q is = 100—p, and n finally denotes the total of the cases. The calculation is quickly worked out with the use of RITALA's tables and is quite reliable.

If a statistically accurate idea of the mutual relationship of the values is desired the difference between the values must be worked out and also the difference of the mean errors according to the formula

$$\varepsilon (p_1 \pm p_2) = \pm \sqrt{\varepsilon^2 (p_1) + \varepsilon^2 (p_2)}$$

in which ε, as known, denotes the mean error, p_1 and p_2 are expressions of two, independent temporary variables (e. g. statistical per centages or arithmetic averages); $\pm$ between p_1 and p_2 denotes that it is indifferent whether you take the mean error of p_1 and the total of p_2 or the difference between them; it is the same in both cases. The work is carried out in this case too with the aid of RITALA's tables and the results are given in the three columns in Table I.

A detail of principle should be mentioned in this connection. If the difference of the mean errors are multipliable by three and the result does not exceed the differences of the per centages the final result is considered *exact*. If it is multipliable by two only the result is *probable* while in the case that it is multipliable by one only the result is considered *possible*. On the basis hereof we learn that in the statistical material comprised in this investigation, in round figures 6,000 persons per month, the greatest frequency of the morbidity (statistically exact) may be observed at the end of the year (August to December). The summit probably being reached in September. Based upon these results there is a certain inclination to consider the prevalence of Stom. ulc. at its height during the climatologically unfavourable months of the year.

The statistical results are represented graphically in Fig. 1, in which the frequency of the disease in the investigated material is evident.

Vertically: percentage of frequency.

Horizontally: the months.

The results of the frequency which have been obtained give us reason to believe that climatological conditions may affect the morbidity. Certainly PELKONEN has, in his very valuable work, pointed out that no direct *parallellism* between the frequency

curve of certain infectious diseases, which VILLEBRAND, however, has been able to find in the case of Scarlatina, and such factors as air pressure, temperature, direction and speed of winds, cloudiness and the relative and absolute moisture of the air. And yet we know by experience (as P. points out) that there is a certain *reciprocity* in the appearance of some diseases and the change of weather. For this reason, I have, in the present investigation, in conformity with PELKONEN's suggestions and reasoning, taken the

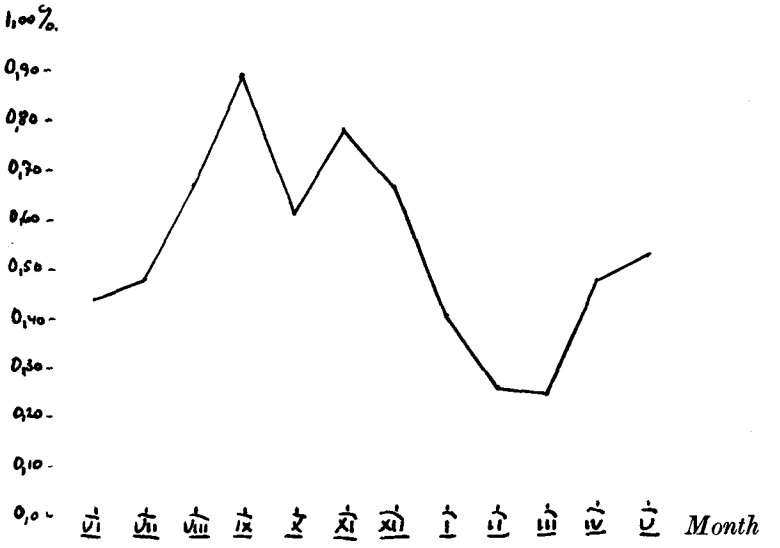


Fig. 1.

daily temperature, as being the most useful for the purpose of comparison. Fig. 2 represents a connected curve of the daily changes of temperature (minimum temperature) for the year during which statements of the morbidity have been collected.

If this curve is compared with the diagram of the appearance of the disease (Fig. 1) we find that the summit of the curve of the frequency appears after a period of the highest daily temperatures, i. e. after the summer months.

On account of these results it seemed to me that a connection might perhaps be found between gastro-intestinal disturbances and Stom. ulc. For the purpose of ascertaining the correctness of this assumption, similarly as for the Stom. ulc. cases, information of all diagnosed stomach and intestinal disturbances, was

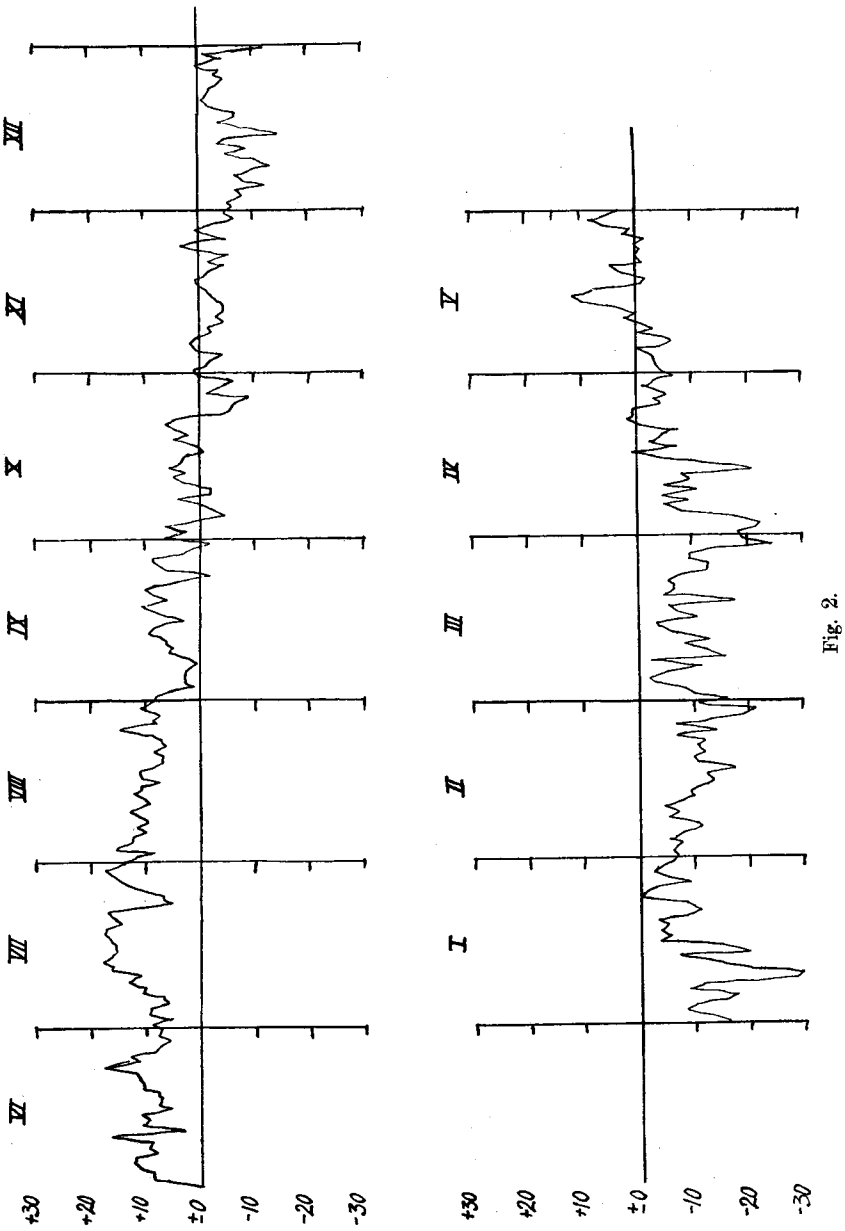


Fig. 2.

Table II.

Month	x	Case	y	Case	x + y	Case	Per cent per month
VI	2790	19	2610	31	5400	50	0.92 ± 0.12 %
VII	2740	41	2647	49	5387	90	1.67 ± 0.17 %
VIII....	2938	48	2858	72	5796	120	2.07 ± 0.19 %
IX	2845	18	2899	32	5744	50	0.87 ± 0.12 %
X.....	3002	17	3103	30	6105	47	0.76 ± 0.12 %
XI.....	2993	27	3034	20	6027	47	0.77 ± 0.12 %
XII	2981	39	3113	20	6094	59	0.96 ± 0.13 %
I.....	2941	13	3090	28	6031	41	0.67 ± 0.11 %
II.....	2946	17	2976	39	5922	56	0.95 ± 0.13 %
III.....	3001	30	2938	54	5939	84	1.41 ± 0.15 %
IV	3413	25	3464	35	6877	60	0.87 ± 0.12 %
V	3419	18	3445	55	6864	73	1.06 ± 0.13 %

[1]) 2.07 ± 0.19 %
1.67 ± 0.17 %
Diff. 0.40 ± 0.21 %
(2 × 0.21 = 0.42)

[2]) 2.07 ± 0.19 %
1.41 ± 0.15 %
Diff. 0.66 ± 0.21 %
(3 × 0.21 = 0.63)

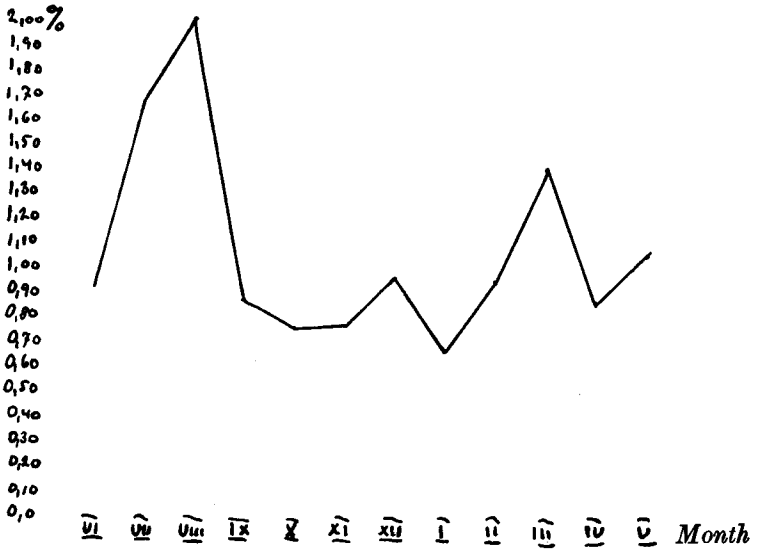


Fig. 3.

collected — from the same material of course as all the other statistical information. The results are given in Table II and Fig. 3, from which it appears clearly that among these 6,000 persons the statistically correct, highest frequency of gastro-intes-

tinal disturbances may be stated in summer and a summit reached in August in all probability.

If a conclusion of this phase of investigations is desired I feel very much inclined to say that Stom. ulc. appears with partiality during a period in which the stomach and intestines have been subjected to infection and there is furthermore a certain inclination to suppose that there may be a possibility of discovering the etiological connection in this particular gastro-intestinal area

Investigations Comprising the Etiology.

As the above mentioned investigations concerning the frequency have shown that Stom. ulc. may be considered a general infected state of the organism, I decided to include a thorough internal medical series of examinations in the etiological programme of investigations, and particularly so on account of some authors having stated certain general disturbances in the organisms. DEAN and DEAN, REICHMAN and WELIKANOWA have observed changes in the blood picture of leucocytes, while HENTZE has observed an inspecific anemia and an increase of the erythrocyte speed of decreasing. LILLY and ROCHETTE mention that the Wassermann-reaction may be of a transient positive nature.

When planning the internal medical series of investigations I have endeavoured to make them as complete as possible in the present circumstances. I hoped, hereby, to trace the general disturbances in the entire organism which may be supposed to represent an etiological connection with Stom. ulc. The investigations were, on this account, based upon reactions in the discharges of the body (urine and faeces) and upon the contents of the stomachal cavity.

In the haematological tests the blood picture, the sedimentation reaction (according to WESTERGREN) and the time of bleeding (according to DUKE) were examined. WASSERMANN and KAHN reactions for stating lues were included as well in this sector.

In the urine, besides the usual protein samples (HNO_2) and sugar (NYLANDER), ketonic substances according to LEGAL's and GERHARDT's reactions were determined. Further, test of the urine were made to find out the presence of bile pigment by the aid of the usual iodine tests, of urobilin (according to SCHLESINGER) and urobilinogen (according to EHRLICH). The reaction of the urine was observed as well.

In the excrements the presence of *worm-eggs* was of course tested and reactions for the purpose of stating a possible content of blood (WAGNER and WEBER).

The object of the last group of examinations was to determine the acidity of the gastric juice by the usual procedure — *Ewald's test meal.*

Besides the mentioned areas for tests the highest axillary temperature during the course of the treatment was noted.

The material was collected from among several different detachments. No importance was attached to the malignity of the disease — slight inflammations as well as severe cases are found among the patients treated. The principal importance was attached to the fact that the pathological state was sufficiently developed not to cause any discussion about the correctness of the diagnosis. I have endeavoured to obtain cases from detachments stationed further back as well and not only from those in the front lines. This was done for the purpose of making the material as multilateral as possible as it may be of etiological value.

The series of investigations comprise 150 cases in all, as seen from Table V. In detailed examinations of this collocation (in which there are, vertically, columns for all tests and, horizontally, for each patient) one is struck by the great number of reduced acidity values in the gastric juice; anacidity in 58 cases, or, 38.7 ± 3.97 per cent, and subacidity in 32 cases, or, 21.3 ± 3.34 per cent. It should be observed that the majority of the subacide finds show values bordering on achyli. In this connection it should further be noticed that the border between normal acidity and subacidity have been determined = 20 for free hydrochloric acid — on the basis of information in medical manuals.

The attention is drawn to another pathological state in the digestive channel, i. e. the presence of rather numerous worm-carriers (34, or, 22.7 ± 3.42 per cent). — Wagner and Weber reactions generally showed negative results. In 15 cases positive Wagner reactions were, however, stated but should not be considered a decisive fact as we have to remember that easily bleeding gums are a characteristic of the disease in question and the blood content in the excrements may be due to this fact. It should further be noted that none of the patients were subjected to a Weber-diet. And furthermore, Weber reactions in the whole series were negative.

Table

N:o	J. N:o	Blood									Urine						
		SR	Sahli	j	Er.	Le.	Duke	RR Hg	VaR	Kahn	Alb.	Nyl.	G.-t.	L.-l.	J.-pr.	E.-h.	S.-r.
1	837	6/2	83	0.77	5'17	8,100	1.15	110/70	—	—	—	—	—	—	—	—	—
2	858	16/7	81	0.74	5'66	6,950	1.30	110/85	—	—	—	—	—	—	—	(+)	+
3	891	6/5	38	0.76	5'44	6,200	1.30	130/75	—	—	—	—	—	—	—	—	—
4	896	2/3	90	0.70	5'17	5,100	1.45	135/85	—	—	—	—	—	—	—	—	—
5	916	6/11	83	0.77	5'37	11,700	1.20	145/100	—	—	—	—	—	—	—	—	—
6	929	5/4	85	0.76	5'56	9,650	1.15	110/70	—	—	—	—	—	—	—	—	—
7	947	18/19	81	0.70	5'78	5,600	1.15	100/60	—	—	—	—	—	—	—	—	—
8	968	18/5						110/70	—	—	—	—	—	—	—	—	—
9	975	5/4	87	0.88	4'93	4,800	1.30	125/90	—	—	—	—	—	—	—	—	—
10	1023	13/14	91	0.78	5'87	11,300	1.15	130/80	—	—	—	—	—	—	—	—	—
11	1029	2/3	89	0.76	5'86	8,100	1.00	120/80	—	—	—	—	—	—	—	—	—
12	1066	5/6	91	0.79	5'74	10,150	0.45	125/80	—	—	—	—	—	—	—	—	—
13	1067	5/7	78	0.81	4'81	10,000	1.00	140/100	—	—	—	—	—	—	—	—	—
14	1048	3/1	92	0.89	5'18	10,750	1.45	135/90	—	—	—	—	—	—	—	—	—
15	1073	13/8	88	0.88	5'00	9,400	1.00	120/75	—	—	—	—	—	—	—	—	+
16	1093	1/2	96	0.88	5'46	11,850	1.45	130/85	—	—	—	—	—	—	—	—	—
17	1094	5/5	92	0.88	5'22	7,450	1.15	120/80	—	—	—	—	—	—	—	—	—
18	1072	10/8	83	0.76	5'48	8,400	0.45	150/75	—	—	—	—	—	—	—	—	(+)
19	1114	17/10	85	0.89	4'75	9,900	0.45	135/90	—	—	—	—	—	—	—	—	—
20	1115	4/2	96	0.81	5'96	5,500	1.00	125/75	—	—	—	—	—	—	—	—	—
21	969	5/5	94	0.80	5'86	8,900	1.15	115/75	—	—	—	—	—	—	—	—	+
22	1116	14/5	78	0.89	4'54	6,950	0.45	113/70	—	—	—	—	—	—	—	—	(+)
23	1153	26/6	80	0.79	5'07	10,800	1.15	90/40	—	—	—	—	—	—	—	—	—
24	1047	30/5	82	0.77	5'33	4,000	2.00	135/80	—	—	—	—	—	—	—	—	+
25	1075	12/4	93	0.88	5'31	10,100	1.15	135/70	—	—	—	—	—	—	—	—	—
26	1107	40/10	73	0.81	4'50	7,000	2.15	145/100	—	—	—	—	—	—	—	—	—
27	1117	10/4	85	0.75	5'66	6,900	1.00	145/90	—	—	—	—	—	—	—	—	(+)
28	1160	13	86	0.76	5'66	6,100	1.15	125/85	+	+	—	—	—	—	—	—	+
29	1196	11/9	85	0.87	4'85	6,800	1.30	120/75	—	—	—	—	—	—	—	—	—
30	1213	7/5	90	0.79	5'68	8,300	1.00	115/70	—	—	—	—	—	—	—	—	(+)
31	1195	4/1	89	0.78	5'72	5,050	1.15	125/75	—	—	—	—	—	—	—	—	—
32	1241	5/7	88	0.88	5'00	5,300	1.15	113/80	—	—	—	—	—	—	—	—	—
33	1209	14/10	83	0.81	5'14	7,300	1.30	120/70	+	+	—	—	—	—	—	—	—
34	1244	8/8	83	0.76	5'49	8,450	1.00	125/85	—	—	—	—	—	—	—	—	—
35	1279	4/7	93	0.85	5'48	6,600	1.30	130/90	—	—	—	—	—	—	—	—	—
36	1286	2/8	87	0.82	5'34	7,500	1.00	110/65	—	—	—	—	—	—	—	—	—
37	878	2/7	96	0.83	5'77	5,350	1.45	125/90	—	—	—	—	—	—	—	—	—
38	1215	5/9	83	0.76	5'46	7,500	1.15	125/80	—	—	—	—	—	—	—	—	(+)
39	1234	6/7	80	0·76	5'26	7,500	1.00	135/85	—	—	—	—	—	—	—	—	(+)
40	1240	1/6	91	0·78	5'80	5,350	1.15	120/85	—	—	—	—	—	—	—	—	(+)
41	1243	3/1	91	0·78	5'80	5,350	1.15	105/65	—	—	—	—	—	—	—	—	—
42	1266	2/1	98	0·81	6'05	9,200	1.30	110/75	+	—	—	—	—	—	—	—	—
43	1276	8/7	93	0·83	5'60	12,400	1.00	120/98	—	—	—	—	—	—	—	—	—
44	1310	10/6	90	0·82	5'47	6,550	1.15	120/80	—	—	—	—	—	—	—	—	—
45	1318	10/5	88	0·86	5'11	6,700	1.00	105/70	—	—	—	—	—	—	—	—	—
46	1330	17/10	82	0·76	5'41	7,950	1.30	110/65	—	—	—	—	—	—	—	—	—
47	1331	15/10	83	0·70	5'94	7,350	1.00	90/65	—	—	—	—	—	—	—	—	—
48	1332	3/4	98	0·88	5'57	8,350	1.00	105/85	—	—	—	—	—	—	—	—	—
49	1333	13/6	96	0·78	6'20	8,800	1.15	115/70	—	—	—	—	—	—	—	—	—
50	1356	5/10	82	0·78	5'29	5,750	1.30	115/65	—	—	—	—	—	—	—	—	—

V.

Reaction	Ewald's test meal			Excr.			Temp. ad.	Remarks
	Wa.	We.	Mae.	Ko-o.	Hcl.	Ta.		
Acid	—	—	—	—	—	64	37.0°	Urticaria.
Str. alk.	—	—	—	+	7	46	37.0°	
Alk.	—	—	—	+	25	51	37.8°	
Acid	+	+	—	—	—	48	37.2°	
Acid	—	—	—	—	—	13	37.2°	
Str. acid	—	—	—	+	29	91	37.4°	
Acid	—	—	—	—	—	25	37.0°	
			—					Lues lat.
Str. acid	—	—	—	+	37	58	37.2°	
Acid	—	—	—	—	—	35	38.5°	
Acid	—	—	—	+	5	46	37.4°	
Acid	—	—	—	—	—	26	37.6°	
Str. acid	—	—	—	+	16	34	37.2°	
Acid	—	—	—	—	—	31	37.4°	
Acid	—	—	—	—	—	32	37.6°	
Acid	—	—	—	—	—	11	36.9°	
Acid	—	—	—	—	—	12	37.3°	
Acid	—	—	—	—	—	15	37.4°	
Alk.	—	—	—	+	9	28	37.0°	
Weak alk.	+	—	—	—	—	25	37.0°	
Acid	—	—	—	+	30	72	37.0°	
Alk.	—	—	+	+	12	30	37.0°	Helminth.
Acid	—	—	—	—	—	17	36.9°	
Acid	+	—	—	—	—	22	37.4°	
Weak alk.	—	—	+	+	49	78	37.5°	Scabies. Ot. med. supp. Helminth.
Alk.	+	—	—	(+)	5	48	37.7°	
Acid	—	—	+	—	—	27	37.6°	Helminth.
Alk.	+	—	—	+	17	33	37.3°	Lues serop.
Alk.	—	—	—	—	—	14	37.1°	
Acid	—	—	—	—	—	32	37.0°	
Acid	—	—	—	—	—	13	37.0°	
Weak acid	—	—	—	+	10	27	36.7°	
Str. alk.	—	—	+	—	—	36	37.0°	Lues serop. Ot. med. supp. Helminth.
Acid	—	—	+	+	22	52	37.0°	Helminth.
Acid	—	—	—	—	—	22	36.7°	
Weak acid	—	—	—	—	—	29	37.2°	
Acid	—	—	—	+	30	64	37.1°	Dyspepsia.
Acid	—	—	—	—	—	27	36.8°	
Weak alk.	—	—	—	—	—	31	36.8°	Paradentosis.
Weak alk.	+	—	+	(+)	5	28	37.6°	Helminth.
Weak alk.	—	—	+	+	38	57	37.4°	Helminth.
Str. acid	—	—	—	(+)	11	68	37.0°	
Acid	—	—	—	—	—	27	36.8°	
Acid	—	—	—	+	32	75	36.5°	
Acid	—	—	+	+	18	62	37.1°	Helminth.
Acid	—	—	—	+	36	81	37.0°	
Alk.	—	—	—	—	—	36	33.6°	
Str. acid	+	—	—	+	35	63	36.8°	Paradentosis.
Str. acid	—	—	—	—	—	45	36.9°	
Acid	+	—	—	(+)	7	62	37.3°	

N:o	J. N:o	Blood													Kahn
		SR	Sahli	j	Er.	Le.	Ne.	Ly.	Mo.	Eo.	Ba.	Duke	RR Hg.	WaR.	
51	1334	14/10	77	0.73	5'28	8,600						1.15	110/70	—	—
52	1354	7/2	88	0.73	6'01	5,400						1.00	115/80	—	—
53	1355	5/19	86	0.76	5'65	6,500						1.45	110/75	—	—
54	1357	22/17	80	0.89	4'51	9,250						1.00	125/75	—	—
55	1371	4/7	80	0.80	5'02	4,900						1.15	105/60	—	—
56	1380	3/2	88	0.80	5'52	5,700						2.15	100/60	—	—
57	1385	2/1	83	0.75	5'57	8,400						1.30	115/65	—	—
58	1381	3/4	88	0.85	5'16	10,150						1.15	100/65	—	—
59	1399	13/7	80	0.74	5'40	9,400						1.30	115/75	—	—
60	1400	13/8	78	0.71	5'50	5,300						1.45	130/85	—	—
61	997	20/15	86	0.85	5'63	5,250						1.00	115/70	—	—
62	1411	5/5	90	0.79	5'66	5,800						1.45	105/70	—	—
63	1413	13/8	91	0.77	5'96	8,000						1.30	105/65	—	—
64	1414	5/3	81	0.81	4'99	7,750						1.00	115/75	—	—
65	1430	3/1	93	0.85	5'50	6,800						1.15	130/90	—	—
66	1401	6/7	85	0.76	5'61	7,300						2.30	120/80	—	—
67	1454	6/5	85	0.85	5'00	5,450						1.30	130/85	—	—
68	1460	2/2	96	0.87	5'51	8,900						2.00	125/90	—	—
69	1461	15/9	88	0.86	5'10	7,650						1.15	130/85	—	—
70	1475	8/10	72	0.85	4'25	5,700						1.15	140/100	—	—
71	1483	16/10	82	0.80	5'14	4,450						1.15	160/115	—	—
72	1194	24/8	56	1.10	2'60	5,950						1.30	105/65	—	—
73	1392	7/4	92	0.91	5'07	7,450						1.15	130/85	—	—
74	1525	3/2	89	0.88	5'03	11,500						1.30	125/85	—	—
75	1524	5/6	89	0.70	6'38	6,800						1.15	110/70	—	—
76	1462	42/4	95	0.85	5'58	11,150	%	%	%	%	%	1.15	115/75	—	—
77	1531	5/4	80	0.79	5'07	6,800	63	31	6	—	—	1.15	105/70	—	—
78	1532	18/12	92	0.92	4'99	5,700	71	23	5	1	—	1.15	135/90	—	—
79	1533	10/16	87	0.75	5'79	7,800	59	32	7	2	—	2.15	130/95	—	—
80	1547	3/3	83	0.72	5'77	8,600	69	23	5	3	—	1.45	135/55	—	—
81	1557	4/2	89	0.80	5'56	7,550	65	33	2	—	—	1.00	135/75	—	—
82	1561	6/5	78	0.80	4'87	7,400	60	32	7	1	—	1.45	125/85	—	—
83	3327/PKL	2/2	85	0.82	5'21	7,850	67	28	5	—	—	1.15	112/79	—	—
84	1578	20/16	83	0.84	4'93	9,800	75	21	4	—	—	1.30	115/80	—	—
85	1579	2/2	92	0.88	5'25	8,450	70	27	3	—	—	1.00	105/70	—	—
86	1597	11/11	74	0.81	4'58	5,250	65	26	8	0.5	0.5	1.30	110/65	—	+
87	1601	5/7	93	0.83	5'49	11,250	68	30	2	—	—	1.30	105/70	—	—
88	1602	11/9	82	0.80	5'13	7,850	73	19	7	1	—	1.15	110/80	—	—
89	1614	9/4	83	0.79	5'26	10,700	66	27	6	1	—	1.00	110/62	—	—
90	1609	2/2	89	0.78	5'72	12,950	74	17	9	—	—	1.00	145/95	—	—
91	1619	3/4	88	0.80	5'53	7,850	70	27	3	—	—	1.15	115/80	—	—
92	1637	10/8	90	0.83	5'39	7,000	76	19	5	—	—	1.45	110/70	—	—
93	1646	3/5	85	0.78	5'55	10,600	67	22	10	1	—	1.00	120/80	—	—
94	1677	2/3	88	0.82	5'38	8,700	71	19	8	2	—	1.30	120/80	—	—
95	1678	20/24	73	0.81	4'49	10,900	65	33	1	1	—	1.45	110/75	—	—
96	1662	5/5	82	0.77	5'32	7,500	74	21	5	—	—	1.50	140/100	—	—
97	1685	2/3	80	0.80	4'93	13,600	62	27	11	—	—	0.45	130/95	—	—
98	1707	11/13	85	0.78	5'48	8,350	63	31	5	1	—	1.45	125/90	—	—
99	1708	5/5	85	0.78	5'46	8,150	69	27	2	—	—	1.45	115/85	—	—
100	1140	12/8	84	0.77	5'46	12,250	61	33	6	—	—	0.45	115/83	—	—

Urine									Excr.			Ewald's test meal			Temp. ad.	Remarks
Alb.	Nyl.	G-t.	L-l.	J. pr.	E-h.	S-r.	Reaction	C-vit. mg %	Wa.	We.	mae	Ko-o.	Hcl.	Ta.		
—	—	—	—	—	—	—	Acid		—	—	+	—	—	22	37.8°	Helminth.
—	—	—	—	—	—	—	Alk.		—	—	—	—	—	50	37.3°	
—	—	—	—	—	—	—	Str. acid		—	—	—	+	33	79	37.0°	
—	—	—	—	—	—	—	Weak alk.		—	—	+	—	—	20	37.2°	Helminth.
—	—	—	—	—	—	(+)	Acid		—	—	—	+	32	80	36.5°	
—	—	—	—	—	—	—	Acid		—	—	—	+	24	51	36.3°	
—	—	—	—	—	—	—	Acid		—	—	—	—	—	21	36.8°	
—	—	—	—	—	—	—	Acid		—	—	—	+	10	38	36.4°	
—	—	—	—	—	—	—	Acid		—	—	—	—	—	32	36.5°	
—	—	—	—	—	—	—	Acid		—	—	—	+	47	95	36.9°	
—	—	—	—	—	—	—	Acid		—	—	—	+	19	58	39.0°	Inf. ac. Scabies
—	—	—	—	—	—	—	Str. acid		—	—	+	—	—	43	36.7°	Helminth.
—	—	—	—	—	—	—	Str. acid		—	—	+	—	—	35	36.6°	Helminth.
—	—	—	—	—	—	—	Acid		—	—	—	—	—	42	36.9°	
—	—	—	—	—	—	—	Weak acid		—	—	—	—	—	49	36.9°	
—	—	—	—	—	—	—	Acid		—	—	—	+	38	92	36.8°	
—	—	(+)	—	—	—	—	Acid		—	—	—	+	8	52	37.4°	V. inc. reg. gen. dx. infect.
—	—	—	—	—	—	—	Acid		—	—	—	+	23	73	36.9°	
—	—	—	—	—	—	—	Acid		—	—	—	+	48	105	37.0°	
—	—	—	—	—	—	—	Acid		—	—	—	+	31	72	36.8°	Lues lat.
—	—	—	—	—	—	—	Weak acid		+	—	—	—	—	55	36.9°	
—	—	—	—	—	—	—	Neutr.		—	—	+	(+)	4	35	37.2°	Anaemia hyper. chrom. botr.
—	—	—	—	—	—	—	Acid		—	—	—	—	—	15	37.2°	
—	—	—	—	—	—	—	Acid		—	—	—	+	25	50	37.2°	
—	—	—	—	—	—	—	Weak acid		—	—	—	(+)	8	60	36.8°	St. post pyelit. Furunculosis.
—	—	—	—	—	—	—	Acid		—	—	+	—	—	69	37.1°	Helminth.
—	—	—	—	—	—	—	Neutr.	0.15	—	—	+	+	10	23	37.0°	Helminth.
—	—	—	—	—	—	—	Acid	0.24	+	—	—	(+)	6	30	37.2°	
—	—	—	—	—	—	—	Acid	0.10	—	—	—	—	—	24	37.8°	
—	—	—	—	—	—	—	Acid	0.13	—	—	—	—	—	10	37.0°	
—	—	—	—	—	—	—	Alk.	0.06	—	—	+	+	10	37	36.8°	Helminth.
—	—	—	—	—	—	—	Acid	0.12	—	—	—	+	10	30	37.2°	Ecz. extr. inf. l. a.
—	—	—	—	—	—	(+)	Alk.	0.18	—	—	—	+	20	48	36.8°	
—	—	—	—	—	—	—	Acid	0.08	—	—	+	+	15	44	36.8°	Helminth.
—	—	—	—	—	—	—	Acid	0.04	—	—	—	—	—	13	36.8°	
—	—	—	—	—	—	—	Neutr.	0.13	—	—	—	+	9	34	36.8°	Lues serop.
—	—	—	—	—	—	—	Acid	0.07	—	—	+	+	45	69	36.8°	Helminth.
—	—	—	—	—	—	—	Acid	0.14	—	—	—	+	5	28	37.3°	
—	—	—	—	—	—	—	Neutr.	0.12	—	—	—	—	—	45	37.0°	
—	—	—	—	—	—	—	Acid	0.10	—	—	—	(+)	4	51	37.2°	
—	—	—	—	—	—	—	Neutr.	0.04	—	—	+	+	37	52	37.2°	Helminth.
—	—	—	—	—	—	—	Alk.	0.30	—	—	—	+	10	48	37.0°	
—	—	—	—	—	—	—	Weak acid	0.12	—	—	—	—	—	17	36.9°	
—	—	—	—	—	—	—	Alk.	0.37	—	—	—	+	10	47	37.8°	
—	—	—	—	—	—	—	Acid	0.32	—	—	—	+	20	78	37.1°	
—	—	—	—	—	—	—	Acid	0.13	—	—	—	—	—	15	37.0°	Paradentosis
—	—	—	—	—	—	—	Acid	0.17	—	—	—	—	—	27	36.7°	
—	—	—	—	—	—	—	Acid	0.06	—	—	—	+	29	50	37.0°	
—	—	—	—	—	—	—	Acid	0.04	—	—	—	(+)	4	58	36.8°	
—	—	—	—	—	—	—	Acid	0.08	—	—	—	+	25	80	37.0°	Lues lat.

N:o	J. N:o	Blood													
		SR	Sahli	j	Er.	Le.	Ne.	Ly.	Mo.	Eo.	Ba.	Duke	RR Hg.	WaR	Kahn
101	1738	24/9	80	0.83	4'83	5,850	66	22	9	3	—	1.00	135/95	—	—
102	1751	8/6	86	0.83	5'18	8,200	62	29	7	2	—	1.00	130/75	—	—
103	1765	10/16	86	0.83	5'23	5,800	63	26	9	2	—	1.15	125/85	—	—
104	1781	6/4	82	0.79	5'16	8,300	73	22	5	—	—	1.00	120/80	—	—
105	1740	2/2	82	0.75	5'50	6,100	72	16	12	—	—	0.45	130/83	—	—
106	1804	4/3	92	0.79	5'80	7,750	61	32	6	1	—	1.15	140/95	—	—
107	1844	6/1	95	0.80	5'98	7,350	73	20	4	3	—	0.45	115/85	—	—
108	1865	3/4	80	0.80	4'98	10,800	64	32	3	1	—	1.15	135/75	—	—
109	1868	16/20	83	0.87	5'35	11,700	70	22	6	2	—	1.30	120/80	—	—
110	1870	2/4	80	0.78	5'61	6,800	73	25	2	—	—	2.30	105/70	—	—
111	1745	2/3	82	0.78	5'23	7,200	67	22	7	4	—	1.15	110/75	—	+
112	1893	9/10	72	0.74	4'85	6,350	65	30	3	2	—	0.50	145/85	—	—
113	1828	6/10	81	0.81	4'83	4,650	68	27	4	1	—	1.00	120/80	—	—
114	1892	15/16	80	0.90	4'40	6,600	72	19	5	4	—	2.00	125/80	—	—
115	1902	9/10	85	0.82	5'20	7,800	68	22	8	2	—	1.00	130/90	—	—
116	1927	5/3	87	0.79	5'52	3,050	65	22	7	4	—	1.15	110/85	—	—
117	1929	3/7	82	0.83	4'92	7,000	70	25	5	—	—	0.45	120/85	—	—
118	1903	72/48	67	0.78	4'30	11,450	76	21	3	—	—	0.45	135/90	—	—
119	1950	12/11	80	0.88	4'53	5,900	65	27	4.5	3	0.5	1.00	115/70	—	—
120	1951	11/7	87	0.88	4'93	5,400	73	24	3	—	—	0.45	120/80	—	—
121	1975	5/3	82	0.80	5'12	6,200	63.5	29.5	5.5	1.5	—	1.15	120/90	—	—
122	1974	3/1	90	0.84	5'36	7,950	59	31.5	4	5	0.5	1.00	125/70	—	—
123	1979	4/2	79	0.82	4'82	4,800	69	29	2	—	—	1.15	125/95	—	—
124	1953	87/33	75	0.80	4'67	6,700	50.5	39	8,5	2	—	1.15	140/95	—	—
125	1980	5/5	84	0.83	5'09	4,200	60	29.5	7	3.5	—	0.45	105/70	—	—
126	2019	9/10	78	0.94	4'19	5,750	72	23	5	—	—	1.00	135/90	—	—
127	2029	7/10	84	0.84	5'02	5,500	55	32	7.5	4.5	1	1.00	110/85	—	—
128	2034	3/5	86	0.81	5'34	6,450	64	26.5	6.5	2.5	0.5	1.15	125/85	—	—
129	2036	7/9	91	0.85	5'38	10,400	61	29	5.5	4.5	—	2.30	125/90	—	—
130	2058	21/21	75	0.83	4'51	4,800	57	37	6	0	—	1.00	140/80	—	—
131	1905	23/7	60	0.72	4'15	5,700	65	27	3.5	3.5	1	2.15	145/105	—	—
132	2077	21/19	78	0.86	4'55	11,300	82	10	7.5	0.5	—	1.00	125/70	—	—
133	2018	1/1	83	0.74	5'62	4,200	63.5	27	7.5	1.5	0.5	1.00	105/65	—	—
134	2108	13/5	85	0.78	5'42	6,600	68.5	12.5	15.5	3.0	0.5	0.45	130/100	—	—
135	2128	2/2	95	0.83	5'75	6,500	68.5	21.5	9.0	1½	—	1.00	115/85	—	—
136	2130	11/6	80	0.77	5'19	9,450	57.5	29.5	8	5	—	1.30	120/85	—	—
137	2074	8/7	75	0.89	4'22	3.900	49	37.5	7	6.5	—	1.15	120/85	—	—
138	2090	6/2	95	0.86	5'57	9,150	70.5	23	8	0.5	—	1.00	145/100	—	—
139	2129	13/1	81	0.91	4'43	7,400	64.5	30	4.5	1	—	0.45	130/80	—	—
140	2131	14/6	85	0.78	5'52	10,450	62	25.5	10	2.5	—	1.15	140/90	—	—
141	2153	14/11	83	0.85	4'30	3,950	41.5	43	10	2.5	3	0.30	130/80	—	—
142	2162	18/28	87	0.92	4'72	10,100	68.5	25	6	—	1	0.45	145/100	—	—
143	2163	5/2	84	0.79	5'30	5,200	57.5	29.5	9	3.5	0.5	0.45	145/85	—	—
144	2144	16/26	84	0.82	5'12	7,350	72	18	5.5	4	0.5	0.45	145/95	—	—
145	4846/PKL	3/3	90	0.86	5'24	6,250	59.5	32.5	3.5	3.5	1	0.30	120/85	—	—
146	2203	5/5	93	0.85	5'22	4,750	66	24.5	7	1.5	1	0.45	135/95	—	—
147	2240	2/3	80	0.77	5'16	8,700	59.5	33.5	3	3	1	4.30	120/80	—	—
148	2232	6/6	87	0.78	5'56	9,650	71.5	20	6.5	1.5	0.5	0.30	135/90	—	—
149	2234	10/8	82	0.78	5'27	5,350	55	34	11	—	—	1.00	115/80	—	—
150	2363	9/8	88	0.80	5'52	8,500	67.5	25	4	3	0.5	1.15	135/82	—	—

Urine									Excr.			Ewald's test meal			Temp. ad.	Remarks
Alb.	Nyl.	G-t	L-l.	J-pr.	E-h	S-r	Reaction	C-vit. mg %	Wa.	We.	mae	Ko-o	Hcl	Ta.		
—	—	—	—	—	—	—	Neutr.	0.09	—	—	+	+	18	50	37.0°	Lues lat. Helminth.
—	—	—	—	—	—	—	Acid	0.22	—	—	+	—	—	45	36.5°	Helminth.
—	—	—	—	—	—	—	Acid	0.04	—	—	—	+	35	75	36.9°	
—	—	—	—	—	—	—	Acid	0.29	—	—	—	—	—	18	37.4°	
—	—	—	—	—	—	—	Neutr.	0.04	—	—	—	+	23	60	36.9°	Enteritis ac.
—	—	—	—	—	—	—	Weak acid	0.06	—	—	—	+	21	47	36.8°	
—	—	—	—	—	—	—	Acid	0.06	—	—	—	—	—	30	37.0°	
—	—	—	—	—	—	—	Acid	0.10	—	—	+	+	34	64	37.0°	Helminth.
—	—	—	—	—	—	—	Alk.	0.16	—	—	—	(+)	6	38	36.6°	
—	—	—	—	—	—	—	Neutr.	0.07	—	—	—	—	—	35	36.8°	
—	—	—	—	—	—	—	Acid	0.06	—	—	+	+	52	110	37.4°	Lues serop. Helminth.
—	—	—	—	—	—	—	Acid	0.04	—	—	—	+	10	58	36.8°	
—	—	—	—	—	—	—	Acid	0.18	—	—	—	+	37	64	37.5°	Commotio univ.
—	—	—	—	—	—	—	Acid	0.03	—	—	+	+	22	62	36.9°	Helminth.
—	—	—	—	—	—	—	Acid	0.07	—	—	—	+	57	65	37.2°	Dyspepsia
—	—	—	—	—	—	—	Alk.	0.06	+	—	+	—	—	29	36.8°	Helminth.
—	—	—	—	—	—	—	Alk.	0.12	+	—	—	+	30	63	36.8°	Furunculosis.
—	—	—	—	—	—	—	Acid	0.10	—	—	+	—	—	4	37.5°	Lues lat. Helminth.
—	—	—	—	—	—	—	Acid	0.07	—	—	—	—	—	25	37.2°	Lues lat.
—	—	—	—	—	—	—	Acid	0.04	—	—	+	—	—	33	37.1°	Helminth.
—	—	—	—	—	—	—	Acid	0.08	—	—	—	+	33	68	36.8°	
—	—	—	—	—	—	—	Acid	0.18	—	—	+	+	22	65	37.1°	Helminth.
—	—	—	—	—	—	—	Acid	0.09	—	—	—	—	—	23	37.0°	
—	—	—	—	—	—	—	Acid	0.10	—	—	—	—	—	22	37.0°	Cath. tub. audit. l. sin.
—	—	—	—	—	—	—	Weak acid	0.19	—	—	+	+	22	73	37.2°	Helminth.
—	—	—	—	—	—	—	Weak alk.	0.05	—	—	—	—	—	8	36.6°	
—	—	—	—	—	—	—	Weak alk.	0.15	—	—	+	+	47	72	37.1°	Helminth.
—	—	—	—	—	—	—	Acid	0.04	—	—	—	+	14	48	37.8°	Bronch. ac.
—	—	—	—	—	—	—	Acid	0.11	+	—	—	+	20	35	37.4°	Ot. med. supp. chron. l. sin.
—	—	—	—	—	—	—	Weak alk.	0.04	—	—	—	+	20	67	37.0°	
—	—	—	—	—	+	—	Weak acid	0.08	—	—	—	+	28	65	37.4°	Lues lat.
—	—	—	—	—	—	—	Acid	0.03	+	—	—	(+)	5	27	37.5°	
—	—	—	—	—	—	—	Acid	0.12	—	—	—	+	57	81	37.3°	Dyspepsia
—	—	—	—	—	—	—	Acid	0.23	+	—	—	+	36	47	37.1°	V. bomb. antebrach.
—	—	—	—	—	—	—	Acid	0.10	—	—	—	+	21	39	36.8°	l. dx ed. superfic.
—	—	—	—	—	—	—	Acid	0.08	+	—	—	(+)	7	47	36.8°	
—	—	—	—	—	—	—	Acid	0.03	—	—	+	—	—	23	37.2°	Helminth.
—	—	—	—	—	—	—	Weak acid	0.10	—	—	—	—	—	41	37.6°	
—	—	—	—	—	—	—	Alk.	0.07	—	—	+	+	37	59	37.7°	Lues lat.
—	—	—	—	—	—	—	Acid	0.11	—	—	—	+	37	60	36.8°	
—	—	—	—	—	—	—	Acid	0.15	—	—	+	+	24	52	36.8°	Lues lat. Helminth.
—	—	—	—	—	—	—	Acid	0.10	—	—	—	+	40	60	36.8°	
—	—	—	—	—	—	—	Acid	0.12	—	—	—	+	45	61	37.3°	
+	—	—	—	—	—	—	Weak acid	0.09	—	—	+	—	—	13	36.8°	Lues lat. Albuminuria.
—	—	—	—	—	—	—	Acid	0.03	—	—	—	+	80	114	36.5°	
—	—	—	—	—	—	—	Acid	0.10	—	—	—	—	—	12	36.5°	
—	—	—	—	—	+	—	Acid	0.05	—	—	—	—	—	34	36.8°	
—	—	—	—	—	—	—	Acid	0.05	—	—	—	+	30	57	36.8°	
—	—	—	—	—	—	—	Acid	0.06	—	—	—	—	—	20	37.0°	
—	—	—	—	—	—	—	Acid	0.14	—	—	—	+	27	46	37.6°	V. bomb. buccae sin.

We now go to examine the results of the urine tests and find that in 15 cases the Urobilin reaction was slightly or completely positive and one case only (No. 2) showed simultaneously a slightly positive Ehrlich. But as Urobilin is constantly present in normal urine a positive Schlesinger reaction should not be considered important especially as Urobilinogene reactions were negative in all cases, excepting the one just mentioned. As regards this single case nothing has appeared either anamnestically or by clinical investigations that might suggest a pathological state in the organism. No importance should be attached to this positive reaction. A positive Urobilinogene reaction was furthermore stated in two cases, however combined with negative Schlesinger. — Slight positive Gerhardt in one case (No. 67) and positive albumin reaction in one case (No. 144), this one, however, due to other causes (Neosalvarsan). Besides the above mentioned facts nothing abnormal was observed in the urine. — In the reaction of the urine nothing worth noting was stated.

In the haematological tests it was observed that the sedimentation reaction showed an increase in 70 cases (46.7 ± 4.07 per cent), the limit having been fixed at the value of 7 mm/h which WESTERGREN considers the extreme limit in normal blood in men. In the blood picture of erythrocytes nothing pathological was stated excepting in one case (No. 72) which will be treated in another connection.

As a slight Leucocytosis was observed in many of the blood tests the work was enlarged in that from cases No. 77 onwards the complete picture of leucocytes was determined as well; regrettably a differentiation of the neutrophile leucocytes was not worked out. This was carried out for the purpose of discovering whether, in the state of the disease, any changes in the mutual relationship of the leucocytes could be combined.

The 74 cases examined in this way show, however, that the mutual per centage values of the leucocytes do not present any notable changes. Yet, the relative number of the eosinophile cells has, in several cases, been larger than normally which is in all probability due to tape-worm in large quantities. The percentage values of the monocytes seem to be somewhat increased as well.

In a few cases clearly reduced leucocyte values were observed. The number of neutrophiles was less than usual while lymphocytes and monocytes appeared in abundance. A blood picture

of this description may, however, be due to other causes. For instance, in case No. 137 the patient had been given a rather large dose of Streptolycin on account of symptoms of inflammation in the throat, and No. 141 was undergoing antiluetic treatment at the time of the investigation.

The result of this detail is that in connection with Stom. ulc. a slight Leucocytosis was stated in 53 cases (35.3 $\pm$ 3.89 per cent) the limit being fixed at 8,000, which, according to the II Medical Clinic of the University at Helsingfors, may be said to be the highest number of blood-corpuscles per mm^3 normal blood in healthy men in Finland.

The time of bleeding was not exceptional in any of the cases.

At the same time as the blood picture of leucocytes was included in the series of investigations the dichlorphenol-indophenol reactions for the purpose of discovering C-vitamin deficits in the organism were added. This was done on account of some modern authors having maintained that Stom. ulc. is a state of disease of a C-hypovitaminosis course. Not one of the 74 tested cases showed a negative reaction and no other avitaminose symptoms were observed.

A special group was made up of 14 cases of lues. 7 of these cases showed a typical vismuth-intoxication. In 5 cases Stom. ulc. did not develop until a long time after an antiluetic treatment. The remaining two cases had not been treated and it may be supposed that the luetic infection may have reduced the power of resistance of the organism in the case of secondary infections.

When interpreting all the above mentioned calculations and reactions, carried out by the same experimentarian, I have been able to collaborate with a physician of internal medicine attached to the hospital. He has also made notes of the general state of the patient. Nothing exceptional was derived herefrom nor did the blood pressure show any abnormalities.

Summarizing the results obtained and adding the information that a general and odontological state afford, observations were made which may be collected in the following main groups.

Table VI.

Anaciditasis	58 cases	= 38.7 $\pm$ 3.97	per cent
Subaciditasis	32 »	= 21.3 $\pm$ 3.34	» »
Helminthiasis	34 »	= 22.7 $\pm$ 3.42	» »

St. post morb. inf.	a) Enteritis ac.	9 cases	28 cases = 18.7 ± 3.18 per cent	
	b) Dysentery	4 »		
	c) Various ...	15 »		
Causa localis		15 »	= 10.0 ± 2.45 »	»
Causa ignota		9 »	= 6.0 ± 1.94 »	»

Excepting these the 14 cases or 9.3 ± 2.37 per cent of lues should be remembered, of which 7 cases or 4.7 ± 1.72 per cent were Intox. c. Bi.

The above mentioned observations support the conception that Stom. ulc. may be related to general disturbances of internal origin in the organism — manifested in the cavity of the mouth — of which the cause should be sought deeper than what the habitat of the disease suggests, even if the local causes play a dominating rôle in a few cases (15 cases or 10.0 ± 2.45 per cent). The supposition is not quite excluded that the symptoms have been caused by reduced general resistance, due to various reasons among which disturbances in the normal functions of the gastro-intestinal system of the organism come into prominence. This way of reasoning brings us to an explanation of the 134 cases which were examined, including those in which local causes may have influenced the course while the causal connection in 9 cases (6,0 ± 1,94 per cent) remains obscure. In 7 cases the etiology was, as known, connected with Intox. c. Bi. This way of reasoning may also be supported in 13 finds in the Stat. post morb. inf. group in which the stomatitis was caused by infectious diseases in the intestinal tube. The probability of this conception becomes still greater if we bear in mind the results of the investigations of the frequency.

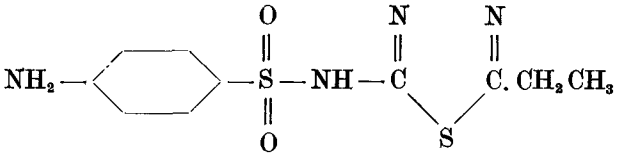
Apart from the etiology we may note that the clinical picture has shown, besides the increased sedimentation reaction treated above, and a slight Leucocytosis, a rise in the temperature of the body — calculated from + 37.0° in the axillary — in 63 cases (42.0 ± 4.03 per cent).

Therapeutics.

As already mentioned in the Introduction a test of a therapeutical method was included in the investigations, based upon simultaneous local and internal treatment. A reason for such proceedings being undertaken is evident from the introduction. The results of the etiological investigations form a further in-

dication that combined therapeutics should lead to quick and certain cure; other possible diseases occurring at the same time should, of course, be taken into consideration.

Examinations and treatment of the patients were carried out according to the following programme. After the anamnesis and general odontological state had been noted the blood pressure was taken, urine and excrements were tested, and the sedimentation reaction was stated. The following morning the blood picture was determined and Ewald's test meal served. The actual treatment was started at the same time; tampons soaked in 19 per cent chromic acid were placed in the decayed parts for about 2—3 min. and the whole cavity of the mouth was rinsed with 10 per cent H_2O_2. Corrosion is justified because the patient is delivered from all pain in a very short time. Immediately after the use of tampons with chromic acid the sulfonamide cure was started; 1 gr sulfetylthiodiazol was administered orally.



The compound is known in the market as Globucide. Every fourth hour, day and night, the dose was administered whereby a constant sulfonamide mirror was maintained in the blood which is particularly important on account of the influence of the remedy. LINN, inter alia, stresses that Globucide should be used entirely on the lines true abortive therapeutics presuppose. For the purpose of avoiding toxic complications and possibly a precipitation of sulfetylthiodiazol in the nephritic channels 1 gr of bicarbonate of sodium was administered at the same time as the sulfonamide which increases the pH value of the urine to 8 whereby a precipitation of the sulfonamide is prohibited (KORHONEN). The patients rinsed their mouths three times a day with a solution of 0.5 per cent NaCl. NaCl is indifferent, as known, and possess the capacity of excluding the corpora aliena from the cavity of the mouth (EKENSTEN).

On the second day of treatment, when the symptoms were abating somewhat, a cleansing of the cavity of the mouth was started, the sulfonamide cure was continued analogously, and of course the rinsings of the mouth with a salt-solution as well.

On the third day of treatment the cleansing of the mouth was completed, if possible, the Globucide cure and rinsing with NaCl was continued.

On the fourth day, when the patients had received 18 doses of sulfetylthiodiazol in all, the cure was stopped. This quantity has been proved to be sufficient, as the parts of the gingiva which previously displayed necrotic parts were, at this time, as a rule just slightly red due to the formation of the epithelium having been started. In a few cases a slight inflammatory reddishness has been observed. The treatment was continued only with salt rinsings until the fifth day, when a new sedimentation reaction was measured (see Table V, the second value in column SR) and with few exceptions (6 cases) the patients were discharged cured. In those cases only in which a vermifuge was still to be administered or other treatment was required (high SR, anemia, etc.) the stay in hospital was extended, for instance in case 72, as the patient's blood picture was not good (Hgb 56, Er 2 60, Le 5950 I 1.10) and increased SR (24 mm) on account of suffering from worms for a long time (anemia hyperchrom. botr.). The stomatitis was cured in normal time despite the patient's other ailments.

For the purpose of giving the reader a clear and more detailed picture of the examinations and treatment of the patients the clinical report of two of the ordinary cases and one of the most malignant character will be given below.

Patient No. 59, J. No. 1,399, T. H., labourer, 25 years.
Admitted: 8. V. —44.

Anamnesis: Family healthy. Measles as a child. Never visited dentist, yet daily care of mouth. Healthy until wounded in the summer 1943. About a fortnight ago the patient felt pains in the left side of his lower jaw, the gums began to swell and the slightest touch caused them to bleed. All through the war stationed in front line, living in timber cottage, food from field kitchen; no milk, smoker. Morb. ven. neg. During last winter pains in stomach after meals.

Stat. pr. The gingival, especially in left side of lower jaw, swollen, inflamed and showing coatings. At —8 spreading along the mucous membrane of the cheek. Foetor +. Pains in gums. Regional lymphal glands on left side swollen and tender. Oesophagus 0. Tartar, no pockets in gums. All teeth intact excepting 6— (extracted) and —6 (Radix).

General condition good. Colour of skin usual. Subcutaneous tissue mediocral, slightly reduced. G. Thyr. normal. Pulm. 0. Cor 0. Abdomen 0. Patellar reflexes normal. Pupils react to light.

D g. Stomatitis ulcerosa.

8. V. 1944. SR 13 mm. Ton. art. 115/75 Hg.

0.5 per cent NaCl rinsing × 3.

9. V. 1944. Clinical picture unchanged. Hgb. 80. Er. 5'40, Le. 9,400, I 0.74. Time of bleeding 1.30. WaR —, Kahn —, Alb. —, Nyl. —. Acid reaction, Gerhardt —, Legal —, Iodine test —, Ehrlich —, Schlesinger —, Wagner —, Weber —, mae —, Ewald's test meal: Kongo —, Ta. 32. Temp. ad 36.5°. Chromic acid tamp. + H_2O_2, rinsing. Globucide 2 × 6, $NaHCO_3$ × 6, 0.5 per cent NaCl rinsings × 3.

10. V. 1944. Symptoms reduced. Coatings disappeared, excepting at —8. Foetor +. Pains continue. Lymphal glands tender. —8, —6 extracted without anaesthesia. Temp. ad. 36.5°.

Globucide 2 × 6, $NaHCO_3$ × 6, 0.5 per cent NaCl rinsings × 3.

11. V. 1944. Symptoms further reduced. Foetor +. No pains. Lymphal glands reactionless. Slight coatings at extraction wound. Tartar removed. Temp. ad. 36.3°.

Globucide 2 × 6, $NaHCO_3$ × 6, 0,5 per cent NaCl rinsings × 3.

12. V. 1944. Formation of epithelium started. Gingival practically normal. Temp. ad. 36.1°.

0.5 per cent NaCl rinsings × 3.

13. V. 1944. Gingival and cheek normal. Temp. ad. 36.5°. SR 7 mm. Discharged from hospital.

13. VI. 1944. Mouth and oesophagus 0.

13. VII. 1944. Mouth and oesophagus 0.

28. VIII. 1944. Mouth and oesophagus 0.

Patient No. 102; J. No. 1,751; M. T., farmer, 26 years.

Admitted: 15. VI. 1944.

Anamnesis: Nothing noteworthy in family. Whooping-cough as a child. Has not visited dentist nor has he cared for his mouth. Healthy, until gums became inflamed about a week ago. Guard post during the war, lodgings underground, no milk, smoker. Morb. ven. neg. No disturbances in stomach.

Stat. pr. Gingival in upper and lower jaw highly inflamed and rich coatings. Foetor +. Pains in gums. Difficulty in eating. Oesophagus reddish. Regional lymphal glands tender. Distally round —8 pocket, not inflamed. Tartar.

V̄	—	V̄	+	V̄	+	+	+		+	+	+	+	+	—	V̄	—
8	+	7	+	6	+	5	+	4	+	3	+	2	+	1	+	+	1	+	2	+	3	+	4	+	5	+	6	+	7	+	8
8	—	7	—	6	—	5	—	4	—	3	—	2	—	1	—	—	1	—	2	—	3	—	4	—	5	—	6	—	7	—	8
+		—		—	+		+		+		+		+				+		+		+		+		+		—		+		+

General condition good. Colour of skin ordinary. Subcutaneous tissue mediocre, slightly reduced. Gl. Thyr. normal. Pulm. 0. Cor 0. Abdomen 0. Patellar reflexes ordinary. Pupils react to light.

D g. Stomatitis ulcerosa. Helminthiasis (Botr. lat.).

15. VI. 1944. SR 8 mm. Ton. art. 130/75 Hg.

0.5 per cent NaCl rinsings × 3.

16. VI. 1944. Clinical picture unchanged. Hgb. 86. Er. 5'18, Le. 8,200 I 0.83. Ne. 62 per cent, Ly. 29 per cent, Mo. 7 per cent, Eo. 2

per cent. Ba. —, WaR —, Kahn —, Time of bleeding 1 min. Alb. —, Nyl. —, Reaction acid, Gerhardt —, Legal —, Iodine test —, Ehrlich —, Schlesinger —, C-vit. 0.22 mg per cent, Wagner +, Weber —, mae. +, Ewald's test meal: Kongo —, Ta. 45. Temp. ad. 36.4°.

Chromic acid tamp. + H_2O_2 rinsings. Globucide 2 × 6, $NaHCO_3$ × 6, 0.5 per cent NaCl rinsings × 3.

17. VI. 1944. Symptoms much reduced. No coatings. Foetor —. No pains. Lymphal glands still tender. 8+, 6+, 4+ extracted without anaesthetics. Temp. ad 36.3°.

Globucide 2 × 6, $NaHCO_3$ × 6, 0.5 per cent NaCl rinsings × 3.

18. VI. 1944. Slightly reddish. Formation of epithelium started. Lymphal glands not tender. Temp. ad. 36.3°. Extr. —8 and +7, no aneasthetics.

Globucide 2 × 6, $NaHCO_3$ × 6, 0.5 per cent NaCl rinsings × 3.

19. VI. 1944. Practically no symptoms in gums. Anthelmintic treatment.

0.5 per cent rinsings × 3.

20. VI. 1944. Mouth and oesophagus 0. Discharged from hospital.

21. VIII. 1944. Mouth and oesophagus 0.

19. IX. 1944. Mouth and oesophagus 0.

Patient No. 24, J. No. 1,047, U. K., son of small farmer, 23 years. Admitted: 11. IV. 1944.

Anamnesis: Nothing special in family. Measles as a child. Has not visited dentist regularly, has not cared for mouth. Healthy until sore throat 5. IV. The following day the patient noticed that the whole of his mouth was inflamed, even the tip of his tongue. No medicine used. Stationed at transport battalion, food from field kitchen, quartered in timber cottage, no milk, non-smoker, Morb. ven. neg. No stomachal troubles.

Stat. pr. Gums swollen, deep red and coatings plentifully. Tip of tongue inflamed. Foetor ++. Difficulty in eating. Slight pains in gingival. Reg. lymphal glands enlarged and tender. Tonsils inflamed, coatings in the craters. Distally around —8 deep, inflamed pocket, No tartar. General condition good. Has not lost weight. Skin normal. Dermografismus +. Reflexes ordinary. No tremor. Gl. Thyr. slightly enlarged, soft. Cor 0. Pulm. 0. Abdomen 0.

—	+	√	+				Protes				√	+	√	+	+	
8	+	7	+	6	+	5	+	4	+	3	+	2	+	1	+	+
8	—	7	—	6	—	5	—	4	—	3	—	2	—	1	—	—
+	√	√	+	+	+	+	+		+	+	+	+	+	√	+	+

1	+	2	+	3	+	4	+	5	+	6	+	7	+	8		
1	—	2	—	3	—	4	—	5	—	6	—	7	—	8		

D g. Angina Plaut-Vincenti et Stomatitis ulcerosa.

11. IV. 1944. SR 30 mm. Ton. art. 135/80 Hg.

0.5 per cent rinsings × 3.

12. IV. 1944. Clinical picture unchanged. Hbg. 82, Er. 5·33, Le. 4,000, I. 0.77. WaR. —, Kahn —, Time of bleeding 2 min. Alb. —, Nyl. —, Acid reaction, Gerhardt —, Legal —, Iodine test —, Ehrlich

—, Schlesinger +, Wagner +, Weber —, mae. —, Ewald's test meal: Kongo —, Ta. 22. Temp. ad. 37.4°.

Chromic acid tampons + H_2O_2 rinsings. Globucide 2 × 6, $NaHCO_3$ × 6,

0.5 per cent rinsings × 3.

13. IV. 1944. Symptoms in tongue and oesophagus distinctly less. In gingival still coatings, plentiful. Foetor +++. No symptoms in lymphal glands. Taking food more difficult. Temp. ad. 37.2°. +4 and +6 extracted, no anaesthetics.

Globucide 2 × 6, $NaHCO_3$ × 6, 0.5 per cent NaCl rinsings × 3.

14. IV. 1944. Symptoms in gums and oesophagus still less. Coatings still in Palatum velum, less in tonsils. Foetor hardly noticeable. No pains. No difficulty in eating. Temp. ad. 36.7°. 6+ and —6 extracted.

Globucide 2 × 6, $NaHCO_3$ × 6, 0.5 per cent NaCl rinsings × 3.

15. IV. 1944. Coatings have disappeared from mouth and throat. Gums reddish only at extraction wounds and remaining roots. Fetoro —. Tongue normal. Temp. ad 37.0°. 7—, 6— extracted.

0.5 per cent rinsings × 3.

16. IV. 1944. Reddishness still around extraction wounds. No symptoms left. —8 extracted.

0.5 per cent NaCl rinsings × 3.

17. IV. 1944. Mouth and oesophagus norm. Temp. ad. 36,9°.

0.5 per cent NaCl rinsings × 3.

18. IV. 1944. SR 10 mm.

24. IV. 1944. Mouth and oesophagus 0. SR 5 mm. Discharged from hospital.

25. V. 1944. Mouth and oesophagus 0.

20. VI. 1944. Mouth and oesophagus 0.

20. VII. 1944. Mouth and oesophagus 0.

All the 150 patients were treated in detail as described above. The method seems above all to have the advantage of curing the disease quickly — in 3 days — and surpasses in this respect all results of treatments mentioned in the literature. It should further be observed that even those cases which originate from intoxation with Bi, abated in the same time. Apart from a few cases with vomiting on the third day of treatment and one case in which the patient had attracted Urticaria no complications caused by the physics were stated.

Percentage of Relapses.

The reliability of the therapeutical method remains to be stated, as relapses are apt to occur in the disease according to various sources in the literature. The question became more interesting as a rather large number of the treated cases, $57 = 38.0 \pm 3.96$

per cent were old, stubbornly relapsing cases which had been treated for a long time according to different local and internal methods but relapses recurred frequently, in most of the cases with very short intervals.

On this account the patients were advised to appear for examination once a month for three months; it was not, however, possible to follow the cases noted for more than one or two months. Various reasons prevented all patients from being examined monthly. I have anyhow been able to control, in all

126 cases, divided as follows:

I month,	126	cases,	of which	8	relapsed
II months,	106	»	»	1	»
III »	68	»	»	—	»

The percentage of relapses is 7.1 ± 2.25 per cent.

The time of control may seem too short but if you take into consideration that all of the comparatively few relapses occurred in the first month, excepting one case, after the treatment was completed and that the majority of the patients were controlled for 3 months, the value of the reliability of the therapeutical method is increased in a high degree.

A closer study of the relapses reveal no special characteristics which might refer the patients to any of the mentioned 7 main groups. On the contrary, representatives of achylists and one former worm-carrier as well as cases in which local causes collaborated were found. In two cases relapses occurred on account of bodily overstraining and in one case on account of gastric catarrh.

Reflections on the Results.

Reviewing the results of the investigations we cannot avoid attaching importance to the numerous internal disturbances that influence the origin of ulcerous inflammation of the gums, and to the very small part represented by dental causes. As already mentioned, only 19 finds were made in which the teeth, in connection with other factors, or, the teeth alone, may be considered to be the origin of the disease. In all these cases the cause evidently lay in unnaturally deep pockets in the gums around the wisdom tooth and not in the roots, badly made fillings, artificial teeth, etc., as maintained by some authors. This intensifies the impression that the general causes should be sought deeper in the organism and CORIOT's statement of dental causes repre-

senting 94—96 per cent seems rather disputable, at least if we consider the material at disposal. Furthermore, in 31 cases, completely intact sets of teeth were stated and yet some of the most malignant cases were found among these patients. In the relapses 2 cases had a perfect set of teeth which strenghtens the fact that local processes in the cavity of the mouth are of secondary nature. In a third case, with a faultless set of teeth, a relapse occurred notwithstanding that a wisdom tooth with an infected pocket was extracted during the treatment. Similarly, in all cases in which the patients had fixed or detachable partial substitutes for teeth (5 cases), no symptoms were observed in clasps, bridge pillars, etc.

There is also some difficulty in accepting the opinion expressed by STURM; he considers Stom. ulc. an allergic disease in which milk or products of milk represent the irritating factor. In the present material only one patient was able to obtain milk regularly. All the others had milk irregularly or at long intervals.

Contrary to the statements of TRETTER, STANLEY, PRINZ, and others, no facts appeared in my material which would support the opinion of a C-vitamin deficit in the organism. Dichlorphenolindophenol reactions were positive, as mentioned, in all the 74 cases, and, besides, the results of the frequency investigations speak against such a conception. It is difficult to connect a lack of C-vitamins with an increase of the morbidity in September.

The investigations of frequency reveal another circumstance which diverges from opinions expressed by authors previously. DALEY and MORELLI mention a summit of the morbidity in the beginning of the year, while NEHSE considers the transitionary months the critical ones. My observations revealed a decided increase after the hottest period of the year. A critical study of a large number of yearly curves would have been most interesting, partly for the purpose of finding a reliable basis for the results obtained, partly for the purpose of comparing whether the same "recurrence every second year" is found in Stom. ulc. as PELKONEN was able to observe in the infectious diseases investigated by him, but statistical material required for this purpose has not been at my disposal.

In connection with the clinical picture LILLY and ROCHETTE mention a transitory positive Wassermann reaction. Anything of this kind has not appeared in the present series of investigations but the positive reactions which have been stated, were

represented by actual lues cases. Neither has anything appeared that would strengthen TIETZE's and ZEMSKY's opinions that the urine contains protein.

On account of the results obtained by these investigations a few words should be said about the values received in connection with Ewald's test meal. Table VI gives a remarkably high percentage of anacide and subacide cases; many of the latter border, however, upon anacidity. For the purpose of obtaining material for comparison between the percentage of anacidity in the material and the true achyli percentage, a series of controls comprising 150 persons who had not complained of stomachal troubles or disturbances in the digestive apparatus were carried out. The following results of the test meals were obtained: Anacidity 21.3 ± 3.34 per cent; Subacidity 29.3 ± 3.72 per cent; the remainder, i. e. practically the half, normal. Contrary to the number of subacide cases in the series of Stom. ulc. a striking majority bordering on normal values are included in these cases. If the above mentioned numerical values are compared with the corresponding values in Table VI, and if the parallelism between the frequency curve on the one hand regarding Stom. ulc. and the gastrointestinal disturbances on the other hand are taken into consideration, a further support of the opinion that persons with a reduced power of resistance on account of disturbances in the digestive channel are those likely to be subjected to Stom. ulc.

An analogous series of controls as regards the presence of tapeworm were also carried out, the 150 patients being chosen at random. A final value of 18.0 ± 3.14 per cent worm-carriers was obtained. The figures given in Table VI are 22.7 ± 3.42 per cent. The difference in this control is not so remarkable as in the previous one.

By the side of the actual investigations very interesting observations were made on the relationship between Ulc. stom. and Angina Plaut-Vincenti. The identity between these two diseases has been pointed out earlier by several authors, DUBOIS, WINBERGER, MORELLI, GRYTHE, FABER, and others. This side of the question was, of course, considered to a certain degree in the present investigations and it may be mentioned that Angina Plaut-Vincenti appeared simultaneously with the stomatitis in 12 cases, or, 8.0 ± 2.22 per cent. A microscopical examination of the coating on the craters of the tonsils showed the same, typ-

ical characteristics as appear in the necrotic parts of the gums; they have been described before by some authors. It should further be pointed out that the angina was cured by the same therapeutics on the whole, and as quickly as the inflammation of the gums. Yet, the impression remains that the throat-affection is more difficult to cure. Excepting the mentioned widely prevalent anginas, a deep redness, stating an inflammation in the oesophagus was noticed in 52 cases, or, 3.47 ± 3.88 per cent.

Another observation, which concerns Stom. ulc. direct, has become fairly clear by these investigations. In many cases the first symptoms and coatings have arisen around the front teeth of the lower jaw. It is difficult to decide whether this is incidental or connected with the manifestation of the disease. This part of the mouth may be considered a locus minoris resistentiae originating in local irritations, such as tartar, etc., and that the bacteria, on this account, here find a first secure position. But as similar conditions were noticed in complete and well cared for sets of teeth this hypothesis seems hardly probable. Another possibility is found in that the saliva might not be normal in some way in this state of the disease. A change in the composition of the saliva in one direction or another or an alteration of the pH value may have taken place.

If this way of reasoning proves to be sound it might be considered that the profuse flow of saliva from the sublingual salivary gland represents a constant irritation in the region in question. A counter-indication is found in the fact that nothing special has been observed around the exit of the parotid gland; on the contrary, a remarkable power of resistance was noticed in these parts. This is undoubtedly an interesting province for further research but there have been no possibilities to undertake such doubtlessly complicated investigations.

I have unfortunately been obliged to set aside other areas of investigation which might have been useful for the purpose of the etiology of the disease and which would have given a more firm hold on the causal connection. I now think, in the first place, of various clinical and medical-clinical methods of investigation, principally those concerning the importance of vitamins, which should have been desirable for the purpose of an all-round treatment of the question. I very much regret to say that there were no possibilitites of carrying out detailed investigations at the hospitals assigned to my work.

Summary.

In my attempt of a detailed explanation of the etiology of ulcerous stomatitis I have striven to make clear the causal connection by internal-medical and frequency investigations. A combined therapeutical method was included in the programme of investigations. Remedies of 10 per cent of chromic acid, 0.5 per cent NaCl-solution, and Globucide, a sulfonamide preparation, were used.

1) The results obtained cause a temptation to consider ulcerous inflammation of the gums as being caused by general disturbances of infectious origin in the organism — manifested in the cavity of the mouth — the cause of the disease having to be sought deeper than suggested by the habitat of the disease. There is an impression of the symptoms possibly having arisen on account of a reduced power of resistance, due to various causes, among which disturbances in the normal functions of the gastro-intestinal system of the organism seem to be the principal ones. This conception is supported by the following facts:

a) An investigation of the frequency comprising one year and based upon a monthly statistical material of about 6,000 persons showed a summit of the morbidity in September (0.89 ± 0.12 per cent) i. e. following the hottest time of the year. By reproducing, also graphically, all the gastro-intestinal diseases occurring in the same material and during the same period, a summit of the morbidity was found in August, (2.07 ± 0.19 per cent), one month before that of the stomatitis, which should be noted. There is a possibility that a pathological state in the digestive channels might be a causal factor of the greatest importance.

b) The internal-medical investigations, comprising various haematological tests, test of urine and excrements and determining the acidity value of the gastric juice, showed, that in a material of 150 persons, observations were made which may be brought together in the following main groups:

Anacidity	58	cases	$= 38.7 \pm 3.97$	per	cent
Subacidity	32	»	$= 21.3 \pm 3.34$	»	»
Helminthiasis	34	»	$= 22.7 \pm 3.42$	»	»
St. post morb. inf. (a) Enteritis ac. 9 cases; (b) Dysentery. 4 »; (c) Various ... 15 »	28	»	$= 18.7 \pm 3.18$	»	»
Causa localis	15	»	$= 10.0 \pm 1.94$	»	»
Causa ignota	9	»	$= 6.0 \pm 2.45$	»	»

Besides these the following should be remembered: 14 cases, or, 9.3 ± 2.37 per cent of lues of which 7 cases were Intox. c. Bi as well and 2 earlier not known. The results show that a considerable majority, 124 finds, originate in the stomachal or the intestinal tube, 13 finds may to some degree be included in the group Stat. post. morb. inf. in which the stomatitis has arisen following inflammatory disturbances in the functions of the bowels. Local finds, 10.0 ± 2.45 per cent, were observed, the majority of them being combined with general causes. The origin of 9 cases was not stated by this method of investigation.

c) 53 cases (35.3 ± 3.89 per cent) of slight Leucocytosis, 70 cases (46.7 ± 4.07 per cent) of increased speed of sedimentation reaction of the erythrocytes, and 63 cases (42.0 ± 4.03 per cent) of increased body-temperature are included in the clinical picture.

2) Examinations concerning the therapeutical method proved that a local treatment with a 10 per cent chromic acid and 0.5 per cent NaCl rinsings combined with a Globucide-cure comprising a total dose of 18 gr during 3 days and nights have brought about a reliable cure. The effective time of treatment has been surpassed in such cases only when the patient has been kept on in hospital for other reasons. The 7 cases originating from intoxication with Bi and cured by the same method of treatment, and in the same time, are extremely interesting.

3) For the purpose of testing the reliability of the method of treatment the patients were examined once a month. 126 patients in all were under observation, the longest period of control being 3 months (67 patients), 2 months (106 patients), and 1 month (126 patients). All the relapses, excepting one, occurred within the first month after termination of treatment. The final percentage of relapses appeared to be 7.1 ± 2.25 per cent.

Zusammenfassung.

Bei meinen Versuchen, die Ätiologie der ulzerösen Stomatitis eingehend zu erklären, habe ich mich bemüht, durch innermedizinische Untersuchungen und Frequenzbestimmungen die ursächlichen Zusammenhänge klarzulegen. Das Programm für die Untersuchungen umfasste u. a. eine kombinierte therapeutische Methode. Als Heilmittel kamen 10 %ige Chromsäure, 0.5 %ige NaCl-Lösung sowie ein Sulfonamidpräparat, Globucide, zur Verwendung.

1) Die erzielten Resultate legen die Annahme nahe, dass die ulzeröse Entzündung des Zahnfleisches durch allgemeine Störungen infektiöser Natur im Organismus bedingt ist, die sich in der Mundhöhle manifestieren, so dass also die Krankheitsursache tiefer zu suchen ist als was der Sitz der Krankheit angibt. Man hat den Eindruck, dass die Symptome vielleicht durch eine Herabsetzung der Abwehrkräfte entstanden sind, die durch verschiedene Ursachen bedingt ist, unter denen Störungen der normalen Funktion des Gastrointestinalsystems des Körpers die wichtigsten zu sein scheinen. Die Auffassung wird durch folgende Tatsachen gestützt:

a) Eine Frequenzuntersuchung, die ein Jahr umfasste und auf einem monatlichen statistischen Material von etwa 6,000 Personen fusste, zeigte eine Anhäufung der Krankheitsfälle im September (0.89 ± 0.12 %), also nach der heissesten Jahreszeit. Durch Darstellung, auch graphisch, sämtlicher gastrointestinaler Krankheitsfälle, die im gleichen Material und derselben Zeitspanne vorkamen, fand man eine Anhäufung der Morbidität im August (2.07 ± 0.19 %), einen Monat vor derjenigen der Stomatitis, was zu beachten ist. Es liegt die Möglichkeit vor, dass ein pathologischer Zustand im Verdauungskanal einen sehr bedeutungsvollen kausalen Faktor darstellen kann.

b) Die innermedizinischen Untersuchungen, die verschiedene Blutuntersuchungen, Harn- und Stuhluntersuchungen sowie Bestimmung der Säurewerte am Magensaft umfassten, ergaben an einem Material von 150 Personen Beobachtungen, die in folgende Hauptgruppen zusammengestellt werden können:

Anazidität			58 Fälle	$= 38.7 \pm 3.97$ %
Hypazidität			32 »	$= 21.3 \pm 3.34$ %
Helminthiasis			34 »	$= 22.7 \pm 3.42$ %
St. post morb. infect.	a) Enteritis acuta	9 Fälle	28 »	$= 18.7 \pm 3.18$ %
	b) Dysenteria	4 »		
	c) Verschiedene Inf.	15 »		
Causa localis			15 »	$= 10.0 \pm 1.94$ %
Causa ignota			9 »	$= 6.0 \pm 2.45$ %

Im übrigen ist noch folgendes zu bedenken: 14 Fälle, oder 9.3 ± 2.37 % Lues, darunter 7 Fälle von Bi-Vergiftung und 2 bisher nicht bekannte Fälle. Aus den Ergebnissen geht hervor, dass eine bedeutende Majorität, 124 Fälle, ihren Ursprung im Magen oder Darmkanal haben, und zwar sind 13 Fälle in gewis-

sem Masse zur Gruppe Status post. morb. infect. zu rechnen, in der die Stomatitis im Anschluss an entzündliche Störungen der Funktion des Darmkanals aufgetreten ist. Lokale Befunde wurden in 10.0 ± 2.45 % beobachtet, und zwar war die Mehrzahl derselben mit allgemeinen Ursachen kombiniert. In 9 Fällen konnte die Ursache mit dieser Untersuchungsmethode nicht festgestellt werden.

c) 53 Fälle (35.3 ± 3.89 %) von leichter Leukozytose, 70 Fälle (46.7 ± 4.07 %) von beschleunigter Senkung der roten Blutkörperchen und 63 Fälle (42.0 ± 4.03 %) von erhöhter Körpertemperatur sind in dem klinischen Bilde vorhanden.

2) Untersuchungen inbezug auf die therapeutische Methode zeigten, dass örtliche Behandlung mit 10 %iger Chromsäure und Spülungen mit 0.5 %igem NaCl kombiniert mit einer Globucide-Kur, die eine Gesamtdosis von 18 Gramm im Laufe von 3mal 24 Stunden umfasste, zuverlässige Heilung gaben. Die effektive Behandlungszeit wurde nur in denjenigen Fällen überschritten wo der Kranke aus anderen Gründen im Krankenhause blieb. Die 7 durch Bi-Vergiftung bedingten Fälle, mit derselben Methode und in der gleichen Zeit geheilt, sind äusserst interessant.

3) Um die Zuverlässigkeit der Behandlungsmethode zu prüfen, wurden die Kranken monatlich untersucht. Es standen im ganzen 126 Kranke in Beobachtung, und zwar war die längste Beobachtungszeit 3 Monate (67 Fälle), 2 Monate (106 Fälle) und 1 Monat (126 Fälle). Sämtliche Rückfälle, ausser einem, traten während des ersten Monats nach Beendigung der Behandlung auf. Die endgültige Zahl der Rückfälle erwies sich als 7.1 ± 2.25 %.

Résumé.

Dans sa tentative de donner une explication détaillée de l'étiologie de la stomatite ulcéreuse, l'auteur s'est efforcé d'éclairer le lien causal par des recherches portant sur la médecine interne et la fréquence de l'affection. Il a inclu dans son programme une méthode thérapeutique combinée. Il a employé comme remèdes des solutions d'acide chromique à 10 %, de NaCl à 0.5 % et une préparation de sulfonamide, le Globucide.

1) D'après les résultats obtenus, on est tenté de chercher la cause de l'inflammation ulcéreuse des gencives dans des troubles généraux d'origine infectieuse se manifestant dans la cavité buccale mais dont il faut chercher la cause plus loin que ne le sug-

gère la localisation buccale. On a l'impression que les symptômes pourraient manifester une diminution du pouvoir de résistance qui serait due à des causes diverses, dont les troubles fonctionnels du système gastro-intestinal paraissent être les principales. Cette conception s'appuie sur les faits suivants:

a) Une enquête concernant la fréquence de la maladie s'étendant sur une année et basée sur un matériel statistique mensuel d'environ 6,000 personnes a démontré un paroxysme de la morbidité en septembre (0.89 ± 0.12 %) c'est-à-dire à la suite de la période la plus chaude de l'année. En experiment par un graphique tous les cas de maladies gastro-intestinales survenus dans le même matériel et durant la même période, on trouve un paroxysme de morbidité en août (2.07 ± 0.19 %) précédant d'un mois celui de la stomatite. Fait important à noter. La possibilité existe que l'état pathologique du tube digestif soit un facteur causal de la plus grande importance.

b) Les recherches de médecine interne, comprenant divers tests hématologiques, urinaires et coprologiques ainsi que la détermination du degré d'acidité du suc gastrique, ont démontré chez 150 personnes la présence de symptômes pouvant être répartis dans les principaux groupes suivants:

Anacidité	58 cas	$= 38.7 \pm 3.97$	%
Subacidité	32 »	$= 21.3 \pm 3.34$	%
Helminthiase	34 »	$= 22.7 \pm 3.42$	%
Etat post-infectieux: a) Entérite 9 cas			
b) Dysenterie 4 »	28 »	$= 18.7 \pm 3.18$	%
c) Divers 15 »			
Cause locale	9 »	$= 10.0 \pm 1.94$	%
Cause inconnue	9 »	$= 6.0 \pm 2.45$	%

En outre il faudrait se souvenir que 14 cas ou 9.3 ± 2.37 % étaient des cas de syphilis dont 7 atteints d'intoxication au bismuth ainsi que 2 ignorés antérieurement. Ces résultats démontrent que dans une majorité considerable des cas, 124, il faut chercher l'origine de l'affection dans le tube digestif et que 13 cas peuvent être compris dans le groupe des états post-infectieux, dans lequel la stomatite s'est produite à la suite de troubles inflammatoires intestinaux. Des causes locales, 10.0 ± 2.45 %, ont été relevées dont la majorité étaient combinées avec des causes générales. Dans 9 cas, la cause de l'affection n'a pas été déterminée par cette méthode de recherche.

c) Une légère leucocytose dans 53 cas (35,3 $\pm$ 3,98 %), une augmentation de la vitesse de sédimentation des érythrocytes dans 70 cas (46.7 $\pm$ 4.07 %) et une élévation de la température dans 63 cas (42.0 $\pm$ 4.03 %), font partie de l'image clinique.

2) Des recherches concernant la méthode thérapeutique ont démontré que le traitement local avec une solution d'acide chromique à 10 % suivi de rinçage au moyen d'une solution de NaCl à 0.5 %, combiné à une cure de Globucide (dose totale diurne et nocturne) de 18 gr. en 3 jours composent un ensemble sur lequel on peut compter. La durée effective du traitement n'a été dépassée que dans les cas où le malade a été gardé à l'hôpital pour d'autres motifs. Les sept cas, suites d'intoxication par le bismuth, traités et guéris par le même procédé, sont intéressants.

3) Dans le but de contrôler l'efficacité de la méthode de traitement, les malades ont été examinés une fois par mois. 126 cas en tout ont été contrôlés, la période la plus longue de contrôle étant de trois mois (67 cas), deux mois (106 cas) et un mois (126 cas). Toutes les récidives excepté une se sont produites dans le premier mois qui a suivi le traitement. Le chiffre final des récidives a été de 7.1 $\pm$ 2.25 %.

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