ON THE CAUSE
OF
VINCENT'S DISEASE
AS IT AFFECTS SAILORS IN SMALL SHIPS,
WITH OBSERVATIONS ON THE TREATMENT.

BY

SURGEON LIEUTENANT JAMES CUTHBERT, R.N.V.R.

I. INTRODUCTION.

Vincent's disease has been described in all parts of the world in times of peace, arising in certain circumstances, but it is in times of war that its incidence rises markedly. The present war is no exception.

I have been struck by the increasing number of cases presenting themselves for treatment and my endeavour has been to find some lesion or condition common to those cases and leading to the start of the infection. Perusal of the literature on the subject shows that different factors operate in different localities (84). My investigations were conducted amongst the sailors of our small ships, our destroyers, minesweepers, drifters and cargo boats. These men form a large and important part of the community in these stirring times and their manner of living is so different from life ashore that their inclusion in a class by themselves for the purpose of investigating this disease is justified.

I propose to give a full historical outline which will indicate the manner in which views on the subject have changed, and which will describe 'en passant' the typical clinical picture, the complications and the various methods of treatment which have been and are still used. It will be appreciated that opinions are divided as regards the aetiology and best methods of treatment of the condition.

Since recent authors have blamed dificiency in the diets of any one of vitamins A, B_1, B_2, C and D as being the predisposing factor in the infection, I have dealt chiefly with thataspect of the question, as will be described later. The investigation covers a period from April 1939 to August 1939 on a cargo boat in the tropics, a period of 4 months on one of H.M. destroyers in the North Sea from December 1939 to September 1940 and a further period of 3 months at a small R.N. Base

Hospital dealing with sailors from small ships operating in the North Sea under conditions of severe strain.

It is my intention, as the result of personal observation, to try to make some contribution, however slight, to our knowledge of the condition as it affects the seafaring classes, especially those on the small ships with whom it has been my privilege to serve.

II. ACKNOWLEDGMENTS.

I thank the Medical Director General of the Navy for his permission to publish this work and Surgeon-Captain Fergusson, R.N. for allowing me to carry out my researches on the cases in his hospital. I am also indebted to Surgeon-Commander (D) Patterson, R.N. for allowing me to work in his department.

III. HISTORICAL OUTLINE.

While infection, by the spirillum and fusiform bacillus, of the mouth and throat regions usually goes by the name of Vincent's Disease or Plaut-Vincent's Disease there is evidence that the disease and the organisms were described before Vincent wrote his paper.

W.D. Miller (1) an American dental surgeon practising in Berlin in Koch's laboratory appears to have first seen and recorded the typical organisms in 1882. Rauchfuss (2) described fusiform bacilli and spirilla in throat lesions in Petrograd in 1893. Hugo Carl Plaut (3 and 4), whose name is often linked with that of Vincent in naming the disease, in 1894 wrote an article entitled "Studien Zur Bakteriellen diagnostik der Diphtherie und der Anginen". In it he described fusiform-like bacilli found in cases of ulcero-membranous angina. Plaut (88 and 89) was a doctor and bacteriologist. Born in Leipzig on 12th October 1858 hewas appointed "professor" in 1919. He died in February 1928.

Jean Hyacinthe Vincent (87) now entered the field. Born at Bordeaux in 1862, he entered the Military School of Medicine and in 1889 became an "assistant" of bacteriology. In 1896 he was promoted to "professor". By 1902 he had been elected to the chair of bacteriology and epidemiology of Val-de-Grace. His activities were wide, including important work on typhoid in the army of France, work on the application of chlorine for disinfecting infectious localities, work on T.A.B. vaccines, the investigations of Typhus outbreaks in the French Army from 1914-18 and he worked with Stoedel on anti-gas gangrene sera. Articles were published on all those subjects. He claims our interest by describing more clearly than before, the infectionwhich bears his name. His original article appeared in 1896 (5).

That same year Vincent describing fusiform bacilli and spirilla present in hospital gangrene infections, entitled his article "Sur l'etiologie et sur les lesions anatomo-pathologiques de la pourriture d'hopital."

In 1897 Bernheim (6) reported 30 cases of ulcerative stomatitis and angina in which he found fusiform bacilli in association with spirilla. The following year Vincent (7 and 10) reported 14 cases of ulcero-membranous angina with the same organisms present. Since then the infection has been known as "Vincent's Angina".

Medical interest in the subject wasaroused and attempts were made to culture the organisms. Veillon and Zuber (8) seem to be the first workers to have isolated the B. fusiformis in pure culture in 1898. They used anaerobic methods and mention a list of sites from which they obtained the organisms.

That same year Moure of Bordeaux (9) described the typical case of ulcerative tonsillitis from its clinical aspects only.

Attempts were made to produce the infection experimentally and Veszpremi (11) was the first to succeed in 1905. Exudate was taken from a case of fatal fuso-spirochaetosis of the mandible with metastatic abscesses in the brain and lung. Pus from the lung abscesses was injected intraperitoneally into each of two rabbits and subcutaneously into each of two others. No disease resulted in one of the former pair but the other developed gangrenous peritonitis and Vincent's organisms were recovered. Both the rabbits which had been injected subcutaneously got foul gangrenous sores with the typical organisms in the exudate.

While this wasa successful experiment a number of failures was the lot of other workers in this field.

In 1905 Vincent (12) published an article in the Lancet. This was the first good description of the disease in English and was an exhaustive account of the clinical and pathological aspects of the malady. All the typical symptoms of sore throat, dysphagia, heavy breath, etc, were mentioned and will be described more fully later.

Wyatt Wingrave (13) in 1908 described the finding of spirochaetes in aural discharges along with many common organisms. These were Vincent's spirilla.

The next important observation was by J.D. Rolleston (14)

in 1912. Describing the ulceration and membrane he stated that when the slough separated a deep excavation of the tonsil might heal up with slight cicatricial contractions but the uvula and more or less of the faucial pillars might be destroyed.

Then came the Great War of 1914-18 and its attendant rise in the number of cases of Vincent's disease. Many workers wrote on the subject and I will now include the essential features of the more important advances in the knowledge of the condition at that period.

W. Wingrave (15) in 1915 again wrote on the condition and reviewed the position up to that time. He noted that much had been written in different countries but that there was no agreement as to the clinical constants of the disease. All workers he said agreed that the presence of spirochaetes and fusiform bodies was essential in diagnosis.

As early as 1915, as wesee in his paper, both officers and men in the British Army were widely affected and suffering from the malady.

He classified the disease into (1) acute and (2) chronic types and noted that constitutional symtoms bore no relation to the size of the patch of membrane. In his description of the throat lesions he noted that the patches came away "en morceaux" and not "en masse". Wingrave then stressed the importance of not confusing a case of Vincent's angina with diphtheria or syphilis or with "hospital" throats due to streptococci or staphylococci where ulceration and deep necrosis is rare. In discussing the aetiology he observed that most military cases were in recruits, both officers and men, who were "thoroughly exhuasted by the hard preliminary fitting up work". He stressed this factor of over-training.

Then followed a good and accurate description of the organisms. The spirocheate varies considerably in size and shape and is slender and undulating with 4 to 8 coarse unequal curves.

In a dense matrix it may be looped, coiled, slightly sigmoid or even straight, single or in bundles. Its length is 10 - 20/U. When seen by darkground illumination it is like an eel, or a whiplash. Vincent's spirillum attains to a much greater size and thickness than Treponema Pallida ever does (see later photograph).

The fusiform body is sluggishly motile and appears as a straight, bent, or "boomerang" shaped rod with tapering ends. The size varies but it is always thickest at the equator. Often it has deeply staining bands or beads adjoining the clear equator and it may occur singly or end to end in pairs. Both the spirilla and fusiform organisms are Gram-negative, the fusiform bacillus staining the more deeply.

Then followed a description of methods of staining and an observation that other elements must be overstained to show the spirilla well. The methods which had been used up to that time were (a) Giemsa, (b) hot carbol-fuchsin followed by a quick and weak alcohol bath, (c) diluted carbol-fuchsin (d) Burri's Indian ink method or 5% collargol in distilled water in the same fashion with no heat. He thought the Burri method was poor, (e) Levaditi for tissues.

Wingrave observed that the relative numbers of fusiform bacilli and spirochaetes varied in different stages of the disease, fusiform organisms being more numerous early in the disease and spirochaetes predominating later. This was strikingly evident in some of my own cases (see later description).

Discussing the distribution of organisms he stated that Vincent's spirochaetes unlike those of syphilis were essentially superficial and were confined to the surface of the tonsils or to their crypts. Even in noma they were rare in the deep structures.

Typical organisms had been discovered in the throat, nose, accessory nasal sinuses (when necrotic), the gums, decayed teeth, middle ear, meningeal and cerebral abscesses, c.s. fluid, gangrenous lung, venereal buboes and even in the cellular tissue of the neck and mediastina following tonsillectomy.

It is of interest to note that gum infections are mentioned now as they form the bulk of the cases in my own series. Wingrave observed that in noma the organisms had been demonstrated somewhat deeper than in Vincent's angina but limited to the necrotic zone, which suggests that they simply follow the death of tissues rather than precede or actually cause necrosis. All the lesions are associated with mixed infection by streptococci, staphylococci, B. pyocaneus etc..

The article went on to note that in the adult, if looked for in a tongue or fauces scraping in the morning, the organisms of Vincent's disease were constant saprophytes. The writer noted that they were undoubtedly symbiotic in their habits and that they favoured conditions associated with foetor and putrefaction. In his conclusions Wingrave made several statements which differ slightly from the findings in the class of men with which this thesis deals. For example he stated "the cause is in the individual rather than in the community" and also "in civil life no community, rich or poor, is especially selected and cases are chiefly sporadic".

In again mentioning the fact that no adult mouth or throat is free from the organisms even in health he said that any diminution of resisting power such as in cases of cerebral apoplexy, might favour spirochaetal activity. He noted the fact that the organism is very delicate and only lives with difficulty outside the body and he said that"contagiousness cannot be accepted for such an emphatic conclusion is not supported by clinical evidence".

We see in this article of Wingrave's the whole subject brought up to date and some of his conclusions e.g. as regards "carriers" or "contagion" are debateable from the point of view of my observations. A discussion will follow later.

Wingrave ended by summarising treatments used up to date. Iodine, silver nitrate, chromic acid, ferric chloride and phenol, he stated, were not thorough. He advocated 5% trikresol in alcohol, painted on the ulcer or formalin diluted in the form of "lysoform". He made no mention of the average time required to cure a mild or

moderate case, which is most important where a sick man is an essential part of a team in say a minesweeper and where his presence is required if the routine of work onboard has to go on smoothly. At the same period of time Harper (16) stressed the fact that Vincent's angina had not received the attention that its importance warranted. He, like Wingrave, pointed to the fact that the constitutional symptoms had hitherto been overlooked by many writers and also that the disease might take on different forms in different patients. He too described accurately the clinical aspects of the lesions and the appearances of the organism but thought that the bacteria had never been grown in pure culture till then, whereas Veillon and Zuber (8) had succeeded. After discussing the differential diagnosis with diphtheria and syphilis he mentioned a very important point, namely the tendency of the disease to persist for weeks and to recur unless adequately treated. (A full description of what I consider adequate treatment to prevent this recurrence is given later). At this point I might say that either local or general treatment, alone or combined, may be used and that Harper favoured local treatment which he stated was of first importance. He made no mention of general treatment. Remedies he had tried were iodine and sulphate of zinc which had only had limited success.

Cauterising with the galvanic cautery had little success either and often aggravated matters. His best results were obtained by painting the part once with 1/100 mercury perchloride and then painting with silver nitrate (30 gr. to the oz.) daily. If either fusiform bacilli or spirilla were still present in a week, then the throat was painted again with perchloride solution. If the treatment was stopped in a week and either organism was present, the condition, he stated, was liable to recur. He concluded that the presence of either of the organisms at the end of a week might be considered to be a positive proof of the continuance of the infection. There was no mention in the article of the possibility of gum infection. Three cases were described in which the throat was infected.

The first two were treated with local formalin and glycerine, and mercury perchloride, and were straightforward cases.

Case No. 3 had a superimposed staphylococcal infection and suppurative adenitis of the neck requiring incision. The throat condition had been present for 15 months. This case took a month to cure and had incisions of the abscesses in the neck, curettage of the throat ulcers and their bases swabbed with pure carbolic acid. Two days later the entire pharynx was swabbed with perchloride of mercury (1/100) and each day for two weeks with silver nitrate (60 gr. per oz.) and on alternate days for a month. This case had caries of the teeth and it shows how serious the malady can become if neglected. The application of carbolic acid seems to have been widely done, for in 1915 Bergeron (17) reported a case which ended fatally after the use of phenol. The latter had caused a gangrene of the throat. Another interesting case of infection of the cervix uteri was reported from America by McConnell (18) in 1916.

Taylor and McKinstry (19) who have done a large amount of work in this field now drew attention to the gum lesions in the disease in a paper entitled "The relation of peri-dental gingivitis to Vincent's Angina". Vincent himself had described an ulcero-membranous stomatitis and an ulcero-membranous gingivitis which might both be in the same patient and which both might have the typical organisms. Later writers had often overlooked the association between throat and gum lesions. Taylor and McKinstry described how they had found that throat and gum lesions often co-existed. These authors were keen on the use of salvarsan applied locally and the average time for the cure of the double infection of throat and gums was seventeen days.

They warn us to examine the gums in all cases presenting with throat infections and to treat infected gums, otherwise the throat will relax from re-infection from the gums. McKinstry (20) in 1917 later contributed again to the literature on the subject and reported on the incidence of fusiform bacilli and spirochaetes in the mouths of healthy recruits during the Great War. He found the organisms present in 95 out of 230 healthy men. In the same

year McKenzie (21) in discussing "Trench Throat" said that it had no connection whatever with trench life, as such, but that its dissemination was favoured by the collection in camps of large numbers of young men. He, too, had seen how it had become common with the war and he blamed this overcrowding. After mentioning, like previous writers, its resemblance to diphtheria and syphilis, he said that since it was an infectious disease the patient really should be isolated and he said that strict segregation in camp life was essential but not necessarily elsewhere as when in private life where the patient could easily keep his own feeding utensils to himself. Thus McKenzie believed in contagiousness as a factor in infecting others, which was in direct opposition to Wingrave's (15) statement. Just how varied the treatments used were, is evident by the fact that McKenzie mentioned his method. He believed in potassium iodide given internally. I quote the following lines to show how uncertain of a cure those workers were. "I suspect that the disease is self-limited in extent and duration and all we need to do is to clean up the ulcers as often as possible if only to subdue the appalling odour they diffuse. For the rest we may have to support the sufferer's patience, as the disease may drag on and on, sluggish but persistent, for as long as a couple of months without however inducing anything more serious than a little pain and a good deal of discomfort". He admitted that there were fatal or grave cases but considered their proportion to be so low that they were not worthy of thought.

Early lesions, he stated, could be aborted by the application of silver nitrate crystals or of pure phenol applied to the spots.

He described stubborn and resistant cases with extensive ulceration of mouths and throats and with typical gum infection. He said that powerful caustics were unwise for them but he advocated confinement to bed, gr. 10 of potassium iodide three times a day and personal attention to mouth toilet. All carious teeth were to be removed as they harbour infection. (I have seen alveolar abscesses arising from the extraction of teeth in such cases and think that it is better to temporise with the teeth if they are not

dead, by the application of a carbolised dressing which will kill the infection in the tooth cavity. If necessary the tooth can be extracted later).

McKenzie now mentioned that the best cleansing solution was hydrogen peroxide frequently sprayed on the ulcers and followed by a mouth wash containing potassium chlorate and tincture of myrrh. He mentioned tincture of iodine or 2% silver nitrate in water as being good local agents.

Bouty (22) had also by 1917 noted the increase in Vincent's angina in the troops in France in the past two years. He stated that in peacetime Vincent's angina constituted 2 to 3% of all cases of throat complaints in the French Army. Recent statistics in a British Military Hospital showed the proportion now to be 23%. This article shows us how serious the problem was becoming. Bouty described the symptoms of the disease and discussed its aetiology. He had been discussing the matter with his friend Vincent who at that time believed that excess of smoking was the chief predisposing factor, plus the presence of decaying organic matter and any irritant fumes such as those given off from exploding shells. Though Bouty admitted that the organism was normally present in the mouths of healthy individuals he thought it favoured irritated conditions of the mucosa. Two types of disease, namely an "ulcerative" and a "pseudo-membranous" were described and it was stressed that secondary streptococcal infection often caused serious complications. An excellent list of complications compiled from previous descriptions was given and they included, 1.painful adenitis of the cervical glands, nearly always present, 2. pyorrhoea alveolaris, 3. stomatitis and gingivitis, 4. gastro-enteritis cases with abundant Vincent's organisms post mortem (Vincent), 5. nephritis and albuminuria (Bouty quotes a case), 6. ulceration of the pharynx with perforation of the carotid artery (Vincent), 7. bronchitis, laryngitis with ulceration of the vocal cords, pleurisy, empyema, otitis media, cachexia and endocarditis, 8. gangrene of the vulva and perineum (Spillman), 9. Vincent's organisms complicating a syphilitic chancre of

phagedaenic tendency (Laimois), 10. thrombosis of the left saphenous vein with gangrenous ulceration of the leg (Guinsberg), 11. osteomyelitis (Gilberti) and 12. Mayer observed an associated pernicious anaemia.

Deaths from Vincent's disease had occurred by this time in French hospitals.

Still more treatments were mentioned by Bouty. e.g. calomel in repeated small doses; but Vincent had informed the writer that he considered that mercury aggravated the condition when the patient was not a syphilitic. Local applications of arsenic and a mixture of sodium-salicylate and potassium chlorate combined with tonics were used too, as were mercury perchloride (1 in 500) in glycerine, silver nitrate, methylene blue and neo-salvarsan (solution or powder applied locally for 2 or 3 days) and frequent gargles of hot hydrogen peroxide. Vincent himself was at this time finding the most efficacious treatment to be a thorough painting with 6% tincture of iodine after well scrubbing with a tampon to remove the membrane. Anti-diphtheritic serum was said to have been of benefit in some cases.

It was important according to Bouty to treat the unaffected side to prevent its outbreak there. His final words were "one dose of salvarsan intravenously has been said to modify the disease and prevent recurrence". This statement is important as it shows when arsenicals began to be used for the condition by the parenteral route and I include it as intravenous arsenic plays a big part in the treatment I have adopted.

Eagleton, Mercer and Hudson (26) now, in an article in the British Medical Journal, stated that they had read articles on Vincent's angina and had noted its prevalence in the troops and had swabbed cases but that they were surprised to see no mention of the condition of the gums in this infection. In several of their cases the throat had cleared up in 8 to 10 days while the organisms had easily been demonstrated in pus around the teeth; and vice versa, in more than one case the patient had come up for treatment for a sore throat (? Vincent's angina) and they had found the organism in peri-dental pus

in great quantity whenever a swab from the tonsil had been indeterminate. These workers stressed the importance of looking for dental trouble in all cases of sore throat and they warned against discharging a patient who had had a sore throat till a swab from around the teeth was negative.

Early in 1918 an interesting episode occurred which shows how eager some of the workers in the field of Vincent's disease were to get credit for their views on the subject. Taylor and McKinstry (23) published an article in reply to the one by Captains Eagleton, Mercer and Hudson of the R.A.M.C. (26) who had written to the British Medical Journal on January 5th 1918 expressing surprise that in the various articles on Vincent's angina both in our tropps overseas and in the home service units they could find no mention of the condition of the gums in this infection. Taylor and McKinstry (23) drew attention to the fact that they had noted the matter in a paper entitled "Fuso-spirillary peri-dental gingivitis" read before the odontological section of the Royal Society of Medicine on November 27th 1916 and published in the "Proceedings" of the Society of that date. They also stated that the condition of the gums was fully dealt with in a further paper entitled "The relation of peri-dental gingivitis to Vincent's angina" (19). This paper has been described and articles such as the above serve to show how the disease was beginning to be fully investigated. McKinstry (24) investigating the subject further examined for organisms the throat smears of 1,320 soldiers and found the fuso-spirillary organisms in 32 or 2.43%. On examining the gums of 230 healthy men not yet in military life he found that 95 were positive i.e. 41.3%.

Colyer (27) while noting in 1918 that attention had been drawn to Vincent's disease of the gums and tonsils during the Great War deprecated the idea that some writers had that it was a disease discovered during the war. Hestated that dental surgeons had recognised the condition for 20 years and that in John Hunter's famous work on the teeth the condition was well described in the section on "scurvy of the gums" (this is the first reference to the lack of vitamin theory, as a cause). Colyer then stated

that J. S. Marshall of Chicago should get the credit for describing the condition and for being the one of the recent writers to draw attention to the subject. Marshall's clinical description was quoted and Colyer stated thatwwe had yet to find the real cause and mentioned that one writer had suggested it was to be found in the constant inhalation of putrescent matter. This was probably Bouty (22) to whom he referred. Colyer's article makes one important point. He noted that the disease was seldom found in a clean mouth and that it often occurred around badly fitting crowns and in the deep pocket posterior to the lower third molar or in any position where there was preexisting periodontal disease e.g. where there was stagnation. (I have found this to be true). His treatment was directed towards the irrigation of the pockets and the local application of drugs considered to have a specific action on the organism. He made a statement that he had not found that arsenical preparations had any advantage over iodine. He believed that areas that could not be easily irrigated should be eradicated.

The war was now over, but articles on the disease continued to be published. In 1919 Semple, Price-Jones and Digby (28) published a report for the Pathological Committee of the War Office of an inquiry into gingivitis and Vincent's disease occurring in the Army. They brought previous reports together and the salient features were to date, a. that gingivitis was present in the Army in soldiers of all ages and categories to the extent of 12% of hospital admissions. b. That soldiers' teeth conditions were not satisfactory, c. that fusiform bacilli and spirochaetes were invariably present in large numbers in gingivitis and Vincent's disease of the mouth and throat, d. that in the absence of clinical symptoms the presence of fusiform bacilli and spirochaetes on the gums, mouth or throat did not mean that a person was suffering from Vincent's disease and, e. that B.fusiformis amboceptors were present in convalescent patients' serum and in patients with fusiform bacilli in their mouths.

A definition of "Vincent's disease" was given and it was classified thus:- 1. acute ulcerative, 2. sub-acute, and 3. chronic. Only three cases of No.1. were seen during the investigation while

the majority of cases were in 2 and 3. The following signs were found in the cases of the series:- 1. swollen and inflamed gums (local and general), 2. gums bleed readily on touch or pressure or had a history of having bled easily for some time, 3. pain was variable and the patient was often unaware of his condition, 4. there was a frequent association with pyorrhoea or the discharge of pus from the teeth sockets, 5. all conditions of the teeth were possible i.e. clean and healthy or dirty and decayed.

An interesting and important report of the percentages of gingivitis in the troops was given. In one unit of 3,000 men 33% had gingivitis and another unit had 29.5%.

Large numbers of men were examined and the percentages of gingivitis and Vincent's agina found. Thus in 184 with unclean mouths Vincent's organisms were present in 139 or 75.5% and gingivitis in 59 or 32%. In all those with gingivitis, fusiform bacilli and spirochaetes were present in large numbers.

The workers took at random 512 men of all ages from 18 to 35 years and upwards in hospital. Fusiform bacilli were found in the gums of 489 men or 95.5%. Spirochaetes were found on the gums of 488 men or 95.3%. Gingivitis was present in 128 men or 25%. Of 234 of the men whose tonsils were examined too, fusiform bacilli were found on the tonsils of 78 or 33.3% and spirochaetes were found on the tonsils of 108 men or 46.1%. (I mention those figures as they have a bearing on my investigation which will come at the end of this historical outline). The Committee finally gave a detailed account of the growing of the organisms and a suggested method for a complement fixation test. Their exact words re the findings in this test are interesting i.e. "These facts suggest that a man's resistance, as indicated by the presence of immune bodies, is his greatest safeguard against the disease".

This report which brings the work of Vincent's disease in the Army up to date in 1919, makes no reference to dietary deficiency as a possible cause.

Most of the work on the illness had hitherto been confined to

the Army. In 1919 a Naval surgeon, W.N. Pickles (29) described the disease as it affected sailors. He had seen large numbers of cases in a depot ship in the previous months and he too described the clinical aspects of the condition and reviewed past literature. His own observations were that it was difficult to determine what the predisposing causes of the affections were in view of the fact that the majority of the patients he had seen were healthy specimens and he concluded that it did not confine itself to the debilitated. Oral sepsis was present in half his cases but he was inclined to think that there was a question whether there was not a similar condition in all patients who came to the sick bay for treatment; and on the other hand he had seen the disease in well cared for mouths where not one single carious tooth could be found. He thought that in infection by this organism preparedness of the soil was not of great importance and that healthy individuals with clean mouths were frequently affected. The disease, he noted, could co-exist with pyorrhoea alveolaris with its atrophied gum papillae but he again stressed the fact that he thought that an unhealthy mouth was far from being the invariable antecedent of Vincent's disease.

His treatment consisted of a mouth wash of potassium chlorate t.d.s.. The gums were cleaned with pledgets of cotton wool moistened, then mopped with dry wool and painted with tincture of iodine. Later instead of iodine he used a solution of liquor arsenicalis, vin.ipecac. and glycerine, similar to that described by Beaumont (51). On this treatment the bleeding and tenderness went in two to three weeks but two months was the average time for the organisms to vanish from smears. With the treatment used by me the average time taken for cure was less than this but the patients were confined to bed as will be described. Pickles described two cases of the acute and more chronic types and discussed the differential diagnosis of the disease on the lines of previous writers. He found that with throat cases his average time for cure was a fortnight (it is much less in my experience) and he stressed the importance of treating the gums to prevent relapses of the throat lesion, like Taylor and McKinstry (19),

Eagleton, Mercer and Hudson (26) and others. Pickles (29) made no reference to diet deficiency as being a possible cause of the condition and I will include here notes of another Naval surgeon's work which was published at this time on scurvy, not mentioning Vincent's disease, i.e. F.C.B. Gittings (31).

He believed that scurvy on shipboard was due to the overcooking of the meat and food and he noted that green vegetables onboard were supplied by the canteen and were subject to the laws of supply and demand; in many cases the demand was not made by the mess caterers. (I mention this because the same conditions prevail to-day in small ships). Gittings dealt with the question of lime juice and stated that in the quantity supplied it was "Merely a pleasant summer drink, nothing more". (See my later work).

At the same time in 1919 Hamilton (30) dealing with a suggested improved diet on the East Indies Station said "There is, I think, very strong evidence that the Naval lime juice is of little or no value as an anti-scorbutic".

The important point in these references is that Gittings (31) while he described cases of scurvy makes no mention whatever of Vincent's infection in the mouths of the cases. (I describe one case of true scurvy with Vincent's disease and another case of scurvy which I saw at Durban and which had no signs of gum or tonsil infection).

We are now, in this historical outline, in the post-war period and I have endeavoured to show at what stage our knowledge of the subject was at that period. The vitamins began to be widely studied and I will now include a description by A.F. Hess (32) of the symptoms he believed existed in a person with hypovitaminosis (subscurvy). His article was entitled "Scurvy past and present". The subscurvy patients, he said, suffer from weakness, lack of stamina, and general ill health and had a sallow complexion. In more serious cases haemorrhages were severe and often extensive. There were vague pains in the joints and limbs and anaemia (normocytic or slightly macrocytic in type) was always a salient feature. (While my contention is that subscurvy plays a large part in causing the lesion, along with other necessary factors, I have not

found the subscurvy state to have such a definite clinical form as Hess describes).

C.H. MOrris (33) in 1921 discussed diet deficiency in Vincent's angina and made several new observations. His statement that Vincent's angina was an affliction of the masses and in civil life was most commonly found among the poorer classes of society was directly opposite to the statement of Wingrave (15) who stated that "In civil life no community rich or poor is especially selected". It will be appreciated that the factor of diet deficiency is creeping into the discussion. Morris further noted that in Military service it was prevalent where large numbers of men were assembled together, dwelling in close proximity to each other and subsisting on the same rations. While rarely seen in civilian dental practice it was daily seen by the Military dental surgeon. Morris proceeded to look for such differences as do exist in the classes affected and those not and he observed that the difference was in the diets. The class prone to be affected lacked the food with the"water soluble" vitamins. The writer by no means claimed that the infection of Vincent's angina was made possible solely because of diet deficiency but he merely desired to call attention to the obvious fact that this disease was most prevalent where diet deficiency existed and he expressed the belief that correction and maintenance of a properly balanced diet would go far towards its prevention. Thus, to the theories of previous writers, of bad teeth, oral sepsis, lack of amboceptors, offensive odours from putrescent matter and a host of others we have added a new theory by Morris.

In the very same journal at the same date as Morris, J.B. Goodall (34) wrote about Naval cases he had seen as opposed to the military ones described by Morris. Goodall moreover emphatically said "the diet does not seem to play any part in the aetiology". As all my cases are sailors this is an important statement.

The article described the typical clinical picture and stressed that the lesions of Vincent's organisms were not entirely confined to the throat but might also affect the gums and buccal

mucosa and that the lesions were more frequent on the lower jaw and in the folds behind the last molar. His treatment was more on the lines as carried out in my series, with the exception of curettage, and was :- stage 1. a good antiseptic spray with an atomiser to all crevices, 2. salvarsan applied to the teeth and gums, and 3. in 3 or 4 days the gums were thoroughly curetted with a spear-shaped excavator. He noted the tendency to recurrence unless the disease was eradicated and in more chronic cases he injected salvarsan twice a week for 3 weeks plus the arsenic paint as described by Beaumont (51) locally. There was no mention of "scaling" the teeth which I consider to be most essential.

David and Hecquet (90) in 1921 were also working on the lines of diet deficiency in Roumania and were struck by the large numbers of ulcerative bucco-pharyngeal lesions labelled "scurvy" which turned out to be due to Vincent's spirillum. They stated as a general rule that Vincent's ulcero membranous lesions coincided with a very unsubstantial nourishment though it was better than that distributed during the months that scurvy ran riot and this fact was so evident to the writers that they could not admit that the supervention of Vincent's organisms was due to insufficient food. Most of the gum cases they saw showed Vincent's fuso-spirillary symbiosis plus the rich but commonplace flora of mouth satrophytes. As will be described later, the criterion of a positive diagnosis is the presence of many Vincent's organisms in any one field. There came a time when ulcero-membranous lesions almost ceased to occur in the Roumanian Army and the civil population, but French companies began to develop it. These French troops were all healthy and had no overstrain or previous morbid strains, (c.v. Wingrave (15) and others), and who were well nourished. Yet in 20 days 12 cases of Vincent's disease appeared.

David and Hecquet classified the malady into two groups and described 1. acute rapid cases, and 2. forms running a slower course where the gums were affected. They noted that the trouble started often round the lower wisdom tooth and spread towards the mucous membrane in the inter-dental line as did Goodall (34), Colyer (27) etc. . They mentioned too, the great loss of substance of the tonsil and the high incidence in which the upper pole of the

tonsil was affected. Some of their cases had bad teeth and others had excellent teeth as was similarly found by Semple, Price Jones and Digby (28).

David and Hecquet favoured arsenobenzol and glycerine applied locally and did not favour internal arsenobenzol which they found was less efficacious than the local treatment. They first cleansed the mouth with 10% silver nitrate and always removed the false tonsillar membrane in a few days and found permanent loss of substance. They advocated a milk diet and gargles of sodium bicarbonate. As a general rule recovery of the patients took place in 12 to 20 days.

Interest in the subject did not fade with the passing of the war and researches continued. Apostolides (35) working on the experimental transmission of the disease to fresh hosts in 1922, observed a similarity in the bacteriological picture of tropical ulcer and Vincent's angina and bandaged pieces of pseudo-membrane from 2 cases of angina over the scarified skin. In two instances he succedded in producing ulcers containing fusiform bacilli and spirochaetes. That same year Keilty (36) continued work on the subject, being chiefly interested in focal infection as a cause and he conducted an exhaustive bacteriological study of the gums. In 200 patients whom he examined fusiform bacilli and spirochaetes were present in almost every case.

The following year Tunnicliff (37), who has done a vast amount of work on Vincent's organisms, observed the organisms in the normal tonsil. In 1924 Davis and Pilot (38) made an exhaustive study of fusiform bacilli and spirochaetes and went into their role as invaders in pulmonary conditions. They succeeded in demonstrating typical Vincent's organisms in the lung cavities of bronchiectasis and pulmonary tuberculosis.

Nichols (39) in that same year was concerned with the sites in which the organisms existed and showed that they could practically always be demonstrated in the sub-gingival crevices of apparently healthy mouths.

Like Davis and Pilot (38), Chevalier Jackson (40) in 1924 published an account of two cases in which there was bronchial infection. Vincent's organisms were demonstrated to be present

and the interesting point is that the bronchoscope was used and the lesions actually viewed.

In 1925 Goodall (41) described a case with extension to the larynx.

In 1927 P. Varney (42) was concerned with the study of fusiform bacilli and approached the problem from a new angle by classifying them serologically. He dealt in his introduction with a short history of early reports on Vincent's organisms and he gave a list of the sites in which the organisms had been obtained including the normal mouth and throat, angina cases, noma, carious teeth and pyorrhoea, leg abscesses, the antrum of Highmore, pulmonary abscesses and bronchiectasis, and in erosive and gangrenous balanitis. He concluded that fusiform bacilli most often attacked mucous membranes but under special conditions might attack almost any organ of the body often producing a rapidly advancing necrosis which unless quickly checked might terminate fatally.

Then he described how 18 pure cultures of fusiform bacilli were isolated and how he had identified four different types by serological and morphological studies. He considered that types 3 and 4 could often be identified by morphological appearances alone but the organisms of types 1 and 2 which vary greatly in size and shape could be safely differentiated from each other only by serological tests. Varney was of the opinion that a classification of fusiform bacilli upon cultural and morphological grounds only, should not be attempted. He isolated a wavy type of fusiform bacillus but he had no evidence that it had any relationship to a true spirillum. R. Tunnicliff (64) believes that fusiform bacilli and spirilla forms are different stages in the life history of a single organism.

The next important step in the study of the disease was by D.T. Smith (43) in 1932, when working on experimental Vincent's infection. He fed guinea pigs on a diet deficient in vitamin C and inoculated them with material rich in fuso-spirillary organisms, between the mucous and cutaneous layers of the cheek. The result was a spreading gangrene analogous to noma. Only local abscesses developed in 8 out of 10 normal animals with similar injections. It will be seen how vitamin deficiency comes to be mentioned more and more as the articles come nearer the present date and how a deficiency of practically all

the vitamins is blamed by subsequent writers and their findings are evidently true for the particular part of the world in which their work was done.

Smith (44) writing again in the following year, 1933, said that the intravenous use of arsphenamine in chronic trench mouth did not produce good results unless it was supplemented by dietary measures and thorough dental treatment.

Two years later in 1935, Gay (45) stated that Vincent's stomatitis was well known to occur particularly in mouths traumatized by chronic irritants such as tartar or faulty dental restorations or accompanying nutritional disturbances such as scurvy, or intoxications such as mercurial poisoning. Noma occurred most often in children's institutions, especially following epidemics of exanthematous fevers or otherwise in undernourished children.

So far dietary deficiency as a whole has been mentioned and only Smith (43) mentioned vitamin C specifically in connection with the disease, in his experimental work. However T.D. Spies (46) in 1935 gave evidence concerning the implication of the vitamin B complex in a paper on the treatment of pellagra. He showed how large numbers of fuso-spirochaetal organisms were found in pellagrous stomatitis and how the lesions cleared as the pellagra improved with the administration of the vitamin B complex. If a subscurvy state exists there may be a subpellagra state too, and many workers, King (80), McLester (70) and others believe in it.

Dalldorf and Russell (47) do also believe that those so-called "sub-clinical vitamin deficiency states" exist. In an article in 1935 on the effect of ascorbic acid injections on capillary resistance they tell how they gave intravenously, to eleven inmates of a county home, 100 mg. of ascorbic acid, and two 3 others 50 mg. All the 14 patients showed a marked and prompt rise in capillary resistance. This they concluded, showed that in one institution at least vitamin C deficiency was practically universal.(This reference is to show how sub-clinical forms of vitamin deficiency can exist).

In 1936 the Australian Clements (49) produced evidence that infections by the fusiform bacillus and the spirillum of Vincent

were predisposed to by a deficiency in the vitamin B complex in the diet. His work was experimental in nature and consisted of feeding rats on a diet deficient in the vitamin B complex and of inoculating them with fuso-spirillary material under different circumstances. Cutaneous ulcers from which he recovered the organisms, only resulted after the part had been traumatized as well. He considered the lack of the vitamin B complex to be vital.

That same year 1936 May Mellanby (50) showed that pyorrhoea developed readily in dogs whose diets were deficient in vitamin A, while on similar diets with this vitamin added the gums remained normal.

Sinclair (65) himself believes that the predisposing factor in fuso-spirillary infection is a combined deficiency of vitamins A and B.

At this period, 1937, current text-books of medicine dealing with "Trench Mouth", cancrum oris or noma, or with Vincent's angina proper, merely mentioned the common clinical signs. Beaumont (51) advised painting of the ulcers with liquor arsenicalis (1½ drachms), tinct.ipecac. (1½ drachms) and glycerine (1 drachm) plus a mouth wash of Milton. The arsenical paint is similar to that used by Goodall (34) and many earlier writers. Beaumont makes no mention of the question of there being an associated vitamin deficiency. For cancrum oris he advised intramuscular sulpharsenol. Local treatment with arsenic solutions and Milton is all that Beaumont mentions for trench mouth and he makes no mention of scaling the teeth and removing tartar which I will show later is as important as any other factor in the treatment.

In 1937 also, another vitamin was mentioned by Lee and Sure (52) who said that there was a possibility that a diet deficient in vitamin B_1 might be reflected dentally. They worked on albino rats and found that when animals were on a diet deficient in vitamin B_1 then the most regular and marked degeneration of myelin was found in the trigeminal and sciatic nerves. The degree of degeneration corresponded in most cases to the number of days the rats were on the deficient diet. The soft parts and the dentine became unduly sensitive to instrumentation. In humans too

it is claimed that persons who have extreme sensitivity of the dentine to instrumentation and thermal changes and who are unduly sensitive to the hypodermic needle, can often have this undue sensitivity cured by large doses of the vitamin B_1.

It is seen from the above references that deficiency of several of the vitamins is important in the study of gum lesions and since fuso-spirillary organisms are in most mouths, (Bouty (22), Semple, Price Jones and Digby (28), Nichols (39), Varney (42) and many others) how easily "trench mouth" could be started and mask the original lesion. This is one of the lines on which I base my study of the seafaring class amongst whom it has been my privilege to work.

There are many instances of workers finding that classes of persons they had examined were deficient in a certain vitamin. Thus Jeghers (53) in 1937 examined 162 medical students at Boston with a special photometer and found 55 or 34% deficient in vitamin A.

In 1937 also, St. Clair, Thomson and Negus (55) published an up-to-date article on the condition and their description of the throat lesion is one of the best I have seen. I include it here in order to bring the salient features up to date. "The local features are fairly typical. On the first day one tonsil shows an easily detachable exudation; on the second day this membrane is found to rest on an ulcerated surface; and on the third and fourth days it becomes thicker and softer. The membrane may become detached at its edges and is expelled or swallowed, leaving a slightly ulcerated surface on which new membrane forms. The so-called membrane is correctly speaking, simply formed by the necrotic tissue from the surface of the ulcer. It is soft and grey, yellowish grey or greenish in colour. When picked up with forceps it comes away in soft easily torn fragments (c.v. Wingrave (15)) leaving an infractuous, eroded area dotted with small bleeding points. The ulcer has an irregular, indolent, flattened base, the edges of which are abrupt or sloping. The surrounding tissue may be reddened or oedematous. After 4 to 10 days the pseudo-membrane ceases to re-form and the ulcerated surface soon gets clean and heals over. But in more pronounced cases the tissues

are involved more deeply and the process extends over the whole tonsil, the adjoining faucial pillars and gums and rarely the side of the pharynx. The destruction of tissue occurs 3 or 4 days after the onset of the disease."

The authors noted that in the great majority of cases Vincent's angina was a unilateral affection. They summarised all previous methods of treatment which have been previously mentioned but considered that the best method was to apply salvarsan powder on a throat swab moistened with glycerine to the lesion, or better still to give intravenous or intramuscular injections of arsenic. This is the opposite opinion of Colyer (27), Cecil (72) in 1940, who thinks that intravenous arsphenamine is not particularly efficacious, David and Hecquet (90) in 1921, and others. St. Clair, Thomson and Negus (55) also advocated cleansing and disinfection of the mouth, with rest, fresh air and tonic treatment. They believed that when the ulcerated gingivitis known as "trench mouth" occurred it was unwise to extract teeth until the general condition had improved with rest, purging and tonics and cleansing the mouth with some such preparation as eusol. They advocated treatment of the ulcerated part with arsenical paint as described by Beaumont (51). There was no mention of vitamins in this work although it was an up to date article otherwise. They said, however, that the disease was most frequently seen in debilitated subjects who were over-worked or in insanitary surroundings; and they said it was but feebly contagious. The writers stressed the fact that increase in the frequency was noted in countries outside the war area. Thus in a clinic in Prague in 1914 the number of cases treated was 7 while in 1919 this had increased to 57.

Again in 1937 more work was done on the mild degrees of vitamin deficiency. Elsom (56) regarding vitamin B deficiency said it was well established through the prolonged study of patients subsisting on a diet of known composition that there were symptoms and signs of mild vitamin B deficiency. These were referable chiefly to a. the gastro-intestinal tract, b. the nervous system, c. to the blood and d. to the cardio-vascular system in certain circumstances. I mention this because I have sought for those symptoms in my cases in an endeavour to find a

shortage of vitamin B in the patients.

Also in 1937 Smith, Persons and Harvey (57) worked to study the identity of the so-called "Goldberger" and "Underhill" types of black tongue in dogs. Their work led them to conclude that vitamin B deficiency was responsible and furthermore that a deficiency of vitamin B in the dog's diets led them to develop fuso-spirochaetal infection in each type of black tongue. They were very definite about this point and even stated that "Dogs on the Goldberger diet may be used with confidence in assaying the pellagra-curative value of certain substances because the appearance of secondary fuso-spirochaetal infection is a reliable indicator of the nutritional status". This is a very important pronouncement.

In 1938 Stitt, Clough and Clough (58) mentioned in connection with the sites in which fuso-spirillary organisms could be found that they were very numerous in Vincent's angina and Vincent's stomatitis but that they might complicate other types of ulceration such as diphtheria, syphilis or carcinoma. This is to be expected in view of the universal presence of the organisms in most mouths. The first writers on the subject, Harper (16), McKenzie (21) and many others, stressed how important it was to differentiate those three conditions, but did not mention that they could co-exist with a Vincent's infection.

T.W. Ross (62) in 1938 continued the research into the disease and advocated treatment with antimony and potassium tartrate and with an antimony compound used for bilharziasis called "fuadin". This was administered intramuscularly but it was very toxic and death was liable to occur with no warning.

In 1939 T. Rosebury and G. Foley (63) worked on experimental Vincent's infection because they stated that in many of its aspects it remained an "obscure problem", and they cited cases of successful transplantation of the organisms to fresh hosts. Failures were mentioned and the workers thought that the negative findings suggested that, while a mixture of members of the fuso-spirochaetal flora might be pathogenic, its virulence was ordinarily not high and hence conditions in excess of simple transfer of the infectious mixture might be required for a successful result.

(This was my own idea and my chief work was to try to find such a condition which was laying the gums of the patients open to infection by the microbes.) The authors mentioned how R. Tunnicliff had induced a state of intoxication in dogs by scillaren-B injections and how easy it was to get a successful transplant of Vincent's organisms in such cases.

Many cases were quoted where mechanical traumatization had been successfully used to aid the transplant of the organisms. Cases were mentioned where infection had followed human bites (c.v. Hennessy and Fletcher (91)) or a blow on the teeth and they noted the rarity with which dentists got infection in comparison with the number of times they injured their hands on teeth. This led them to conclude that the organisms have a comparatively low virulence.

An entirely new idea was evident from their work viz. that complex mixtures were necessary to produce lesions in man, e.g. a. treponema microdentium and b. fusiform bacilli along with c. a small actively motile organism regarded as the "vibrio viridans" of Miller and d. an inadequately defined anaerobic streptococcus. (On reading of this mixture the parallel appeared to me to be the association of the streptothrix actinomyces with the bacillus actinomycetum comitans). Guinea pigs were chiefly used in Rosebury and Foley's work as they had fuso-spirillary organisms in their mouths in health. The great value of experimental fuso-spirochaetal infection, as they saw it, lay in the opportunity that it gave for the study of predisposing or modifying factors in the disease. They carried out an experiment which is important from my point of view, as they studied the effect of neo-arsphenamine on experimental fuso-spirochaetal infection. They too, cited a number of conflicting reports on its efficacy in the disease. Arsphenamine's chief value was to lessen the discharge of the lesions in guinea pigs and to cause a great diminution of the Vincent's flora in the discharge when injected into the lesion. Other experiments with it followed and they concluded that although intravenous administration of neo-arsphenamine under the conditions of these experiments was ineffective in altering the course or character of experimental Vincent's infection, when the same drug

was applied in direct contact with the flora there was a distinct emeliorative effect.

Another very important discovery was that "passage" was possible through various guinea pigs as often as 20 times and that there was "exaltation of virulence". No immunity was conferred on guinea pigs by recovery from the lesions. It will be noted that there was no mention of vitamins in the work of Rosebury and Foley.

In 1939 the American workers seem to have taken a new interest in fuso-spirillary infection for R. Tunnicliff (64) gave a masterly report on the organisms. She described their morphology and the manners of culturing them anaerobically. Her strongest evidence of an aetiological relation between fusiform bacilli and the associated spiral organism and the necrotic processes with which they were associated was furnished by their presence, unmixed with other bacteria, in the area of advancing necrosis.

The main theme of her work was on the question as to whether the fusiform organisms and the spirilla were two distinct organisms or whether they were parts of the life cycle of one organism. (She believed the latter theory). Varney (42) thought in 1927 that they were distinct organisms. He had no evidence with which to conclude that they were otherwise related. Various authors writing of the organisms, mention Tunnicliff as believing that the two organisms are different stages of the same life cycle. Her views were based on cultural evidence mostly. She thought that impaired vitality of tissues was necessary to cause the lesion and she conducted experiments on dogs which she intoxicated with scillaren-B injections and then found that they developed infections with Vincent's organisms. She quoted the work of Miller etc. who did feeding experiments to cause dog black tongue by reducing the vitamin B complex component of their diets.

Dudley Buxton (97) said that the spirochaete is in the deep tissue layers and the B fusiformis is on the surface; and that same year, 1939, another American worker J.A. Sinclair (65) investigated vitamin A and B deficiency as an aetiological factor

in acute, sub-acute and chronic Vincent's infection. His experiments at first were conducted on dogs and other animals. He concluded that epithelial changes developed with vitamin A deficiency when epithelial growth was stimulated by mechanical irritation. This could happen in persons through irritation by fillings, illfitting crowns and carcareous deposits resulting in a slow, non-healing or recurring Vincent's infection. (I may state here that I noted no such keratinisation in sections of gums from cases). He noted that Vincent's infection very frequently occurred along with pellagrous lesions as had T.D. Spies (46) in 1935 and others. Sinclair observed how, after nicotinic acid, there was fading of the mucous membrane lesions, blanching of the erythema of the cutaneous lesions when present and a tendency towards the return of normal gastro-intestinal function. Usually within 24 hours there were striking objective changes in the mucosa of the mouth, throat and tongue and in the rectum. The abnormal redness and the Vincent's infection vanished. His main work, however, was with dogs and his experiments convinced him that vitamin A and B deficiency either alone or combined as a poly-avitaminosis would cause fuso-spirillary infections to arise. He thought too, that vitamin C given along with the vitamins A and B would effect cures, but vitamin C deficiency was in his eyes a minor matter, compared with the lack of vitamins A and B.

Sinclair (65) quoted E.J. Wright as having described the disease which had been reported in various parts of the world and which expressed itself in dimness of vision (day-blindness) sore mouth with smooth, glazed tongue and excematous lesions of the vulva, scrotum or anus. I mention this as it is important in this thesis to consider later those points with reference to the particular class of patients I have observed. From Sinclair's conclusions we see that vitamin A and B deficiency is especially important either alone or as a combined deficiency (poly-avitaminosis). His evidence was backed by the findings of Kirkpatrick (66).

In a field survey in New Guinea, Kirkpatrick examined about 2,000 natives and analysed the data obtained by statistical methods.

The findings indicated that Vincent's infection and suppurative periodontitis too, were associated with a partial deficiency of vitamins A and B.

The majority of writers and workers in the field of Vincent's infection now believed that vitamin deficiency might play a part in predisposing a person to infection. It must not be forgotten that within the last few years vitamin deficiency has been blamed as causing dozens of different diseases just as other causes for diseases had been postulated at different times formally and have since proved to be false with the lapse of time; thus there was the vogue for diagnosing "colitis" and for blaming it on a host of vague symptoms as is described by Axel Munthe in his "Story of Sans Michele". Now, however, there are more accurate chemical and clinical tests with which to study such conditions and the vitamin deficiency theory has been very scientifically studied and conclusions are justifiably made.

As an instance of how far this vitamin aspect of the question has become generally recognised I quote Cecil (72) who in his up to date text book of medicine in 1940 advocates a rich diet, especially one rich in vitamins. No special vitamins are mentioned. The treatment he advocates is local treatment with 10% chromic acid, after hydrogen peroxide had been applied first to the ulcer. Copper sulphate is also mentioned as is 10% neo-arsphenamine in glycerine. He considers that intravenous neo-arsphenamine is not particularly efficacious.

Tidy (69) did not mention vitamin deficiency in his text book but he considered that lesions were probably only produced in the presence of the factor, debility. He said that some authorities believed that agranulocytosis and other blood changes could be produced by Vincent's infection but that the evidence was inconclusive.

The blood picture had not been thoroughly investigated and in July 1940 D.G. Stine (73) working in America, gave his report on 128 cases. The changes in the blood presented a great variety of pictures in Vincent's disease of the mouth and throat. There was no uniformity in either the total leucocyte counts nor in the

differential counts and there was no relation between total leucocyte count, the differential count and the degree of febrile reaction or in the duration of the illness. All his patients were young adults of about the same age, and all were university students and recovered with no complications.

The highest total leucocyte count was 40,000 per cubic millimetre and the highest neutrophil count was 93%. The lowest leucocyte count was 3,450 per c. mm. and the lowest neutrophil count was 12%.

There was no relation between the height of the leucocyte count and the neutrophil count.

The highest lymphocyte count was 88%. Five cases, only, showed a small number of myelocytes.

Red cell counts varied between 2,560,000 per c. mm. and 6,000,000 per c. mm. and the haemoglobin estimation varied between 58% and 115%.

He concluded that the blood picture was of no help in making a diagnosis of Vincent's infection of the mouth and throat. In fact the wide variations possible and the lack of any definite type of blood picture might lead to confusion with leukaemias, agranulocytic angina, "monocytic angina" and aleukaemic leukaemia in all of which a superimposed Vincent's infection in the necrotic lesions might occur. Other clinical evidence had to be used to exclude the latter conditions from Vincent's infection only.

In connection with the blood picture Fuller (92) in 1941 published 5 cases which resembled the anginose type of glandular fever. Four of the cases had a heavy infection of Vincent's organisms and as arsenic caused a rapid improvement in three of them he suggested that these organisms were responsible for the throat infection. However the whole illness was not attributable to a throat infection in his opinion, for he stated that Vincent's organisms are found repeatedly in undoubted cases of glandular fever. The Paul Bunnell test was positive in most of this series and there was a slight monocytosis.

At that time too, Stuart-Barber (93) described anginose glandular fever which had to be differentiated from diphtheria with its more severe constitutional symptoms, definite pseudo-membrane, leucocytosis and the presence of Klebs-Loeffler bacilli

from true Vincent's angina with the absence of characteristic blood changes of glandular fever and the spirilla in a direct smear, and from lymphatic leukaemia with its typical blood picture and steady downward course. He quoted A. Lemierre (94) as having said that "it was very probable that a great many of the so-called cases of Vincent's angina were nothing else than monocytic angina". Perusal of the works of the last two workers, Stuart-Barber (93) and Fuller (92) leaves one in doubt as to whether the cases were truly "mononucleosis" afflictions.

As late as 1940 Breazeale and Greene (74) carried out a further investigation into the incidence of spirochaetes and fusiform bacilli in throat and gum smears although it had been done before by many workers e.g. Semple, Price Jones and Digby (28), McKinstry (20) and others. Breazeale and Greene observed both organisms in small numbers in the mouths of most normal adults especially around gum margins and in tonsillar crypts. They quoted Rosenau's findings of a few Vincent's organisms in 50% of all swabs taken from the throats of troops at Bramshott, U.S.A., Other workers found only one carrier in 50 normal individuals but fusiform bacilli and spirilla in 90% of all smears from diseased teeth in a dental clinic. Breazeale and Greene made slides from throat and gum smears of different groups of patients and after fixing them with heat stained them with crystal violet and ammonium oxalate.

Positive results meant several organisms in a field, while one or no organisms per field meant a negative result. In throat smears only 44 or 6% were positive, in gum smears only 127 or 18% were positive, and in both smears 83 or 11% were positive.

The lowest incidence occurred from school children of superior social and financial levels, while the highest incidence was from Mexican children from homes of low social and economic levels. Organisms were found in 41% of a group of coloured children and Indians at a boarding school.

Among young adults, enlisted men in the Arizona National Guard were 27% positive, college students (girls) were 25% positive and male college students were 36% positive. The results indicated

that among school children and young adults one might expect to find significant numbers of spirochaetes and fusiform bacilli in approximately 35% of throat and gum smears examined.

No historical outline of Vincent's disease and the work that has been done on it would be complete without a reference to the work of Bennett (75) published in 1940. A reception service was established at the Creedmoor State Hospital in May 1936 and it was decided to find the most frequently recurring remediable physical disease noted on admission. There was to be preliminary observation on ways of early detection, methods for control and determination of the possible effects of this disease on the mental course of the patient.

After observation for one year it was decided that ulcero-membranous stomatitis headed the list of remediable diseases. Bennett's criteria of a positive diagnosis included the demonstration of ulcerative lesions of the mucous membrane of the mouth or throat with the finding of both fusiform bacilli and spirochaetes in the stained smear.

His "cure" was constituted by the healing of these lesions and by the finding of negative smears on two consecutive examinations taken 24 hours apart. It is of interest to study Bennett's experiences in treating the condition. The gamut of oral therapeutic agents was run and this included local applications of chromic acid (5%), tincture of iodine, 5% silver nitrate, Fowler's solution, 2% sodium perborate, hydrogen peroxide, tincture of Benzoin, 8% zinc chloride solution, and powdered copper sulphate. N.A.B. was given intravenously and a 10% solution in glycerine applied locally. A small control series got alkaline sodium bicarbonate mouth washes plus a high vitamin C intake. No improvement was noted in this series. Bennett noted that patients with Vincent's disease tended to reject foods rich in vitamin C such as the citrus foods, salads and tomatoes etc, and thought it probable that scurvy in various degrees existed in the patients and that by producing an ulcerated state of the gums it created a favourable medium for infection. Bennett gave all cases of Vincent's disease a high vitamin C intake but he noted that patients in the hospital previously free from Vincent's

infection had developed it while under a high vitamin diet supplied in the form of concentrates. This is an important reference from the point of view of aetiology.

In treatment Bennett's best results were with the anti-syphilitic remedy "Maphersen". Half of the dose was given intravenously and the other half painted on the gums. The teeth were scaled as a routine.

The psychomatic relationships of Vincent's disease were determined. The incident rate of all admissions was 6-5%. Difficulty of feeding was noted in 92 of 121 positive Vincent's patients admitted, and 85 of these required either tube feeding or forced spoon feeding. All patients, save 7, began eating voluntarily as soon as the infection had subsided. One case of affective psychosis contracted Vincent's disease in hospital and had a delusion that she was being poisoned. The delusions subsided after treatment of the mouth affection. Bennett noted the tendency in patients afflicted with Vincent's disease, especially in the trend reaction group, to fortify the delusional system, particularly regarding poisoning by food. He said that refusal of food was an important symptom of this group. When painful oral lesions were relieved very often delusions were ameliorated or depressed and the patient's total load tension was lessened.

Thus in this historical outline we see the fresh aspect of Vincent's disease in mental hospitals.

In 1940 the question of Vincent's organisms in association with vitamin B_2 deficiency cropped up again when Spies, Hightower and Hubbard (79) investigated the effects of nicotinic acid on cases of pellagra. If adequate amounts of nicotinic acid were given to persons with acute or relapsed pellagra there was striking improvement of the Vincent's infection associated with it as well as improvement in the other symptoms of the disease. This is similar to the finding of Sinclair (65) and many others.

An important piece of work was done by J.D. King (83). A medical friend of his mentioned to him that the Nyasaland natives suffered severely from Vincent's disease. Pellagra was common in the region and the two maladies were believed there to be due to lack of the P.P. factor.

King investigated Vincent's disease from this angle and concluded, that as it did not respond to vitamin C, that that ruled out a scorbutic origin. King like Manson-Bahr and Ransford in 1938, believed in the presence of glossitis and stomatitis not only in true pellagra but also in what they termed the "pre-pellagrous state". They said that in temperate climates the skin lesions of pellagra do not become apparent but that the deficiency of the P.P. factor shows itself by stomatitis, characteristic desquamation of the tongue and chronic diarrhoea. King has treated over 34 cases with nicotinic acid and published the results of 4 of them in his first paper. He gave 250 mg. of nicotinic acid in water daily by mouth for 10 days and cured the cases. Only very light local treatment in the form of gargles and paints was used. King firmly believed in the deficiency of nicotinic acid as the predisposing cause of Vincent's Disease. He also attempted to inoculate his own gums but failed. He injured his gums before applying the infective material and there was still a negative result. He concluded that it would appear that factors other than the passage of infected material into mechanically injured tissues may in man at least be necessary before Vincent's gingivitis or stomatitis can be induced in a previously uninfected mouth. King quoted several former writers. McKinstry in 1918 had found no association with scurvy or other dietetic errors. Grieves (1919) mentioned that oral filth and not oral sepsis was the main predisposing cause and that mercury, lead or zinc, could cause similar lesions in some cases. (see later.). Clewer thought in 1919 and 1923 that a mild form of scurvy might be the cause in soldiers on active service. He only went on dietary histories. Harris and Raymond (67) found increased excretion of nicotinic acid in the urine of heavy smokers. Goadby in 1923 blamed deficiency in vitamin C or of fat soluble vitamins A and D as contributory factors to the establishment of the disease.

King (83) said that no convincing evidence had been brought forward to show that in true scurvy the numbers of fusiform bacilli and spirochaetes in the human mouth are unduly increased.

King (80) in a survey of 1,530 school children in the isle of Lewis found only 10% free from gingivitis whereas about 20% of 470 London and Sheffield children were free. There were no definite cases of Vincent's Disease seen in Lewis but no swabs were taken. Only clinical evidence was used.

Roff and Glazebrook (82) worked in a naval training ship situated in the one locality all the time. The boys there were divided into groups and controlled experiments were conducted in them. (Roff was the dental surgeon and Glazebrook the medical specialist in the establishment). They found that those cases with marginal gingivitis and gingivo-stomatitis showed a deficiency of ascorbic acid in the urine. They used the method of Harris and Abbasy (54). Glazebrook informs me that those cases cleared up very well with ascorbic acid. They were not necessarily Vincent's infections but the greatest majority were ordinary gingivitis with swollen, bleeding gums and not cases of trench mouth.

Cathcart, Murray and Beveridge (81) in 1940 studied the diet of the inhabitants of the isle of Lewis from a quantitative aspect. They found that in this island the vitamin C intake was lower than in many other parts of Great Britain. The work made no special reference to Vincent's Disease however, so does not tell us of the relationship of vitamin C deficiency to the disease.

In 1940 another American, H. Field (84) did a lot of work in connection with treatment of the disease and pulmonary infection by the organisms. He noticed that there were grades of infection of the throat and lungs much less severe than those usually described and which were often missed. He had observed the large numbers of drugs used in treatment and stressed that many of them caused more harm to the tissues than good. He thought that more attention should be paid to the general treatment of the patient, particularly nutrition and dental measures, where gums are involved. He endeavoured to find what conditions permitted the fuso-spirillary organisms which were ordinarily harmless saprophytes to become invasive. He thought that possibly there were some more virulent strains than others and that this would seem to justify infectious precautions which would not otherwise

be indicated. He was not absolutely definite on this point however. Field was aware that local lesions like tuberculosis, neoplasms, syphilis or diphtheria might facilitate the entry of a superimposed Vincent's infection (as had been observed by Stitt, Clough and Clough (58)). He also said that certain blood diseases such as acute leukaemias , aleukaemic leukaemia, aplastic anaemia, granulocytopenia and infectious mononucleosis were apt to be complicated by Vincent's infection as is stated by D.G. Stine (73) and others.

Poisoning from heavy metals benzine or its derivatives might be the antecedent to such infection.

Vitamins were discussed again by Field who considered that the reason that Vincent's infection occurred in acute infections and chronic debilitating diseases was because there was an increased requirement for, or a decreased intake of, vitamins in those cases. Field made a statement, very important in connection with this work, that experimental and clinical evidence showed that a deficiency of any one of three vitamins, either A, B or C, might result in fuso-spirillary infections. Field summarised the evidence incriminating those three vitamins. Thus vitamin C was incriminated by 1. finding the mouth organisms invading gingival tissues in scurvy, (Hess (32)) 2. by noting a correlation between gingivitis and capillary fragility in children and by noting that ascorbic acid cured the capillary fragility, (W. Nordenmark (95)) 3. by the finding of low blood levels of ascorbic acid in patients with gingivitis (Weisberger, Young and Morse (96)), and 4. by observations on guinea pigs already mentioned (Smith (43)).

In connection with experimental black-tongue in dogs . 2 types were found to exist. One was cured by the vitamin B_2 complex alone and the other required vitamin A as well to cure it. Fuso-spirillary infection always arose when a dog developed black-tongue. Field, however, concluded that there was still a doubt about the whole question of vitamins and deemed it wise to give supplements of them amounting to at least a daily maintenance dose in treatment. A great deal of Field's work

on treatment covered ground which has already been fully mentioned. One important pronouncement was that arsphenamine is not a specific remedy for fuso-spirochaetosis to the degree that it is for syphilis or that quinine is for malaria. Most of Field's pulmonary cases had fuso-spirillary gingivitis. He thought that smoking lowered local resistance. Local arsenicals were considered to have a specific action on the fuso-spirillary organisms e.g. 10% neoarsphenamine in glycerine. He advocated the introduction of 1% solution of acriflavine to the sub-gingival tissues using a blunt pointed syringe. Dealing with complications of the disease he did not mention any that have not been mentioned before. Field found that the most commonly serious fuso-spirillary infection was pulmonary i.e. lung abscesses and bronchiectasis and he stressed the importance of prophylaxis by attending to the teeth and nutrition.

Another American, Leigh (85) working at the same time as Field investigated the disease and described its epidemiology in America, its clinical course and treatment. The first time quarantine measures against the disease were taken was by Oliver in Bishop's Palace in Manila in 1902. An outbreak of epidemic ulcerative gingivitis occurred in headquarters troops there and half of 85 men were affected. The epidemic only ceased when the afflicted were quarantined and the individual cooking utensils were boiled.

Leigh was emphatic that Tunnicliff's (64) former claim that the spirillum was a late phase in the life cycle of B. fusiformis was no longer tenable and also that trauma or infection injured the tissues often and the organisms were secondary invaders.

The disease had occurred in America in epidemic form from 1914 -18; latent cases always exist in many parts of the country and it was not clear to Leigh just what caused the epidemic to spread. He observed that any article contaminated by saliva could carry infection and that "kissing" was the most positive mode of propagation. He thought this might be the basis of speaking of Vincent's infection as the "fourth venereal disease".

His findings in regard to treatment do not coincide entirely with those I have described later. Thus he waited till the subsidence of all acute symptoms before scaling the teeth. His other local measures have been mentioned but he stated that "while chromic acid might have a therapeutic action on the ulcerating gingivae, its use should be positively discontinued". I quote this because I used it as a means of treatment in all my gum cases with no ill effects. I have found it to be an excellent local remedy and ideal for firming the gums. Leigh said that its use decalcifies the teeth. However this is not noted in a case treated with other therapeutic agents as well, as will be described. Citrus fruits were advocated in the acute stage at least but the reason they were used by Leigh was that they locally prevented salivary stasis by stimulating the salivary flow and thus they indirectly reduced the bacterial count.

CONCLUSION.

In the preceding historical outline I have accomplished that which I set out to do, namely to cover the ground on the most important aspects of the work which has been done on Vincent's Disease. Perusal of the above historical outline will show how the views on the subject have changed since the end of the nineteenth century, how it has been treated at different times and above all the uncertainty which exists with regard to its relation to vitamin deficiency. There are literally hundreds of articles on the condition, to read all of which ~~it~~ is impossible while on active service, but I have in the above summary taken a sample representative of all views on the subject.

IV. GENERAL LINES OF THE INVESTIGATION.

All the work I have done on the condition of Vincent's disease of the gums and throat has been conducted amongst the seafaring classes. I have experienced the life on board ship and have seen the conditions under which the sailors live and work, both in times of peace and in war. The cases written up were all "in-patients" in a small R.N. base hospital, where patients were mostly from small ships, with the exception of 3 cases which I mention. Many cases, not written up were treated as out-patients and I saw them with the kind permission of Surgeon-Commander (D) Patterson,R.N.. It became evident to me that Vincent's Disease in this as in the last war was becoming commoner amongst the personnel of the small ships, i.e. destroyers, drifters, minesweepers, etc, as it had been noted amongst the soldiers during the war of 1914-18 by Wingrave (15), McKinstry (20), McKenzie (21), Bouty (22), Eagleton, Mercer and Hudson (26), Colyer (27) and many others, and amongst the sailors by Pickles (29).

To prove it really was on the increase I hunted through the records of the hospital and found the numbers of bed cases of the disease as far back as April 1939 before the war broke out. I noted the numbers admitted as "in-patients" each quarter up to December 1940. In addition I noted the total numbers of all cases, of tonsillitis, and of diseases of the teeth and gums, and of scurvy, beri-beri and pellagra admitted in each quarter. The accompanying table A. shows the results.

Table B. shows the percentage in each quarter of Vincent's Disease, tonsillitis, scurvy, beri-beri, pellagra, and all diseases of teeth and gums respectively, to the total number of bed cases in the hospital during the quarter. If we study table B. we see that the total number of cases rose after September 1939 when war broke out. This is the natural result of an increased Naval strength and the consequences of service afloat and is to be expected.

During the quarter April to June 1939 there was no dentist and therefore no diseases of the teeth and gums were treated. The percentage of those latter diseases has risen with each ensuing quarter and gingivitis, not classed as Vincent's disease, has been

common in this class. As regards tonsillitis it will be seen that those two quarters in which it was at its highest percentage were the two quarters before war broke out. There is no question of missed Vincent's disease in those figures for my friend Dr. Glazebrook was in charge of the wards at that time and alive to the condition.

TABLE A.	ALL CASES	TONSILLITIS.	DISEASES OF TEETH AND GUMS.	VINCENT'S DISEASE.	SCURVY ×	BERI-BERI OR PELLAGRA
April to June 1939	141	16	0	0	0	0
July to Sept.1939	253	25	1	0	0	0
Oct. to Dec. 1939	586	22	14	0	0	0
Jan. to March,1940	756	32	26	7	1	0
April to June 1940	700	20	31	9	5	0
July to Sept.1940	758	23	49	19	2	0
Oct. to Dec. 1940	899	35	47	27	1	0

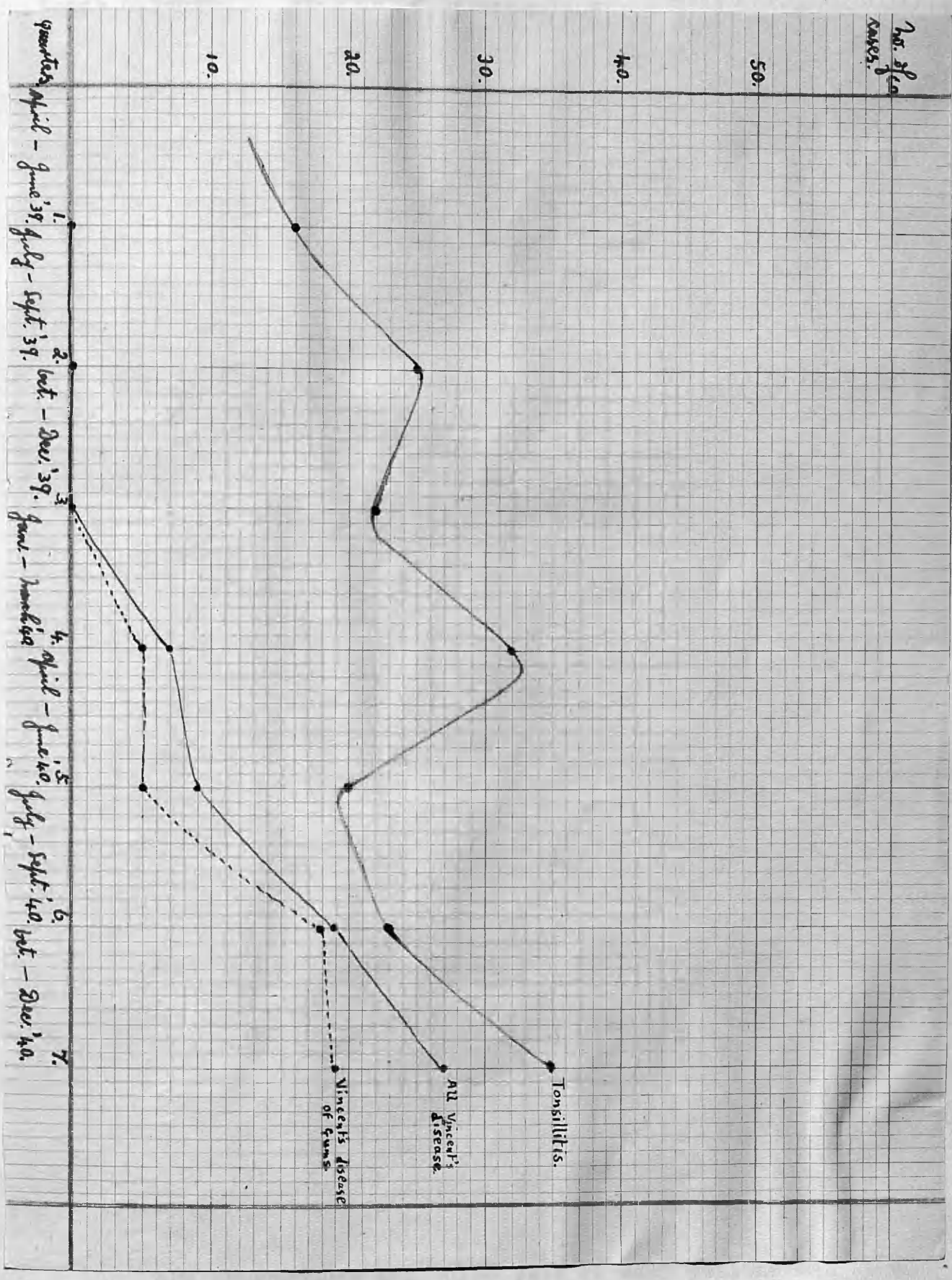
× None of these passed to the "Scurvy" at all Glazebrooks time.

TABLE B.	ALL CASES	% TON-SILLITIS	% DIS-EASES OF TEETH AND GUMS	% VINCENT'S DISEASE	SCURVY	BERI-BERI OR PELLAGRA
April to June 1939	141	11.3	0	0	0	0
July to Sept. 1939	253	9.9	0.4	0	0	0
Oct. to Dec. 1939	586	3.8	2.4	0	0	0
Jan. to March, 1940	756	4.2	3.4	0.93	0.13	0
April to June 1940	700	2.9	4.4	1.3	0.7	0
July to Sept. 1940	758	3.1	6.5	2.5	0.26	0
Oct. to Dec. 1940	899	3.9	5.2	3.0	0.1	0

Tonsillitis percentages rose slightly in the first winter of war to fall in the summer of 1940. Then they showed a slight rise again last winter. This is to be expected. Tonsillitis is common in ship's companies in very cold weather when everything is battened down and the bulk-heads "sweat" and the sailors and officers turn on electric heaters to try to heat the mess-decks and ward-rooms alike. The dryness of the confined atmosphere is extreme, droplet infection abounds, and the nasal and throat mucous membranes suffer as a result.

The fourth and the fifth columns interest us. They show a steady rise in the percentage of Vincent's disease. The first cases were evident in the fourth quarter, i.e. after the war had been in progress for four months. This is the time taken for the body to be depleted of its stores of vitamin C. McLester (70) states that scurvy appears in adults after 2 to 4 months of complete or almost complete lack of vitamin C. This fact of Vincent's disease coming on at this period in the sailors of our small ships has not been mentioned before to my knowledge. The accompanying graph shows the rate of increase of Vincent's disease as the war goes on. Table B. in column 5 bears out this statement of McLester (70) if there is any lack of vitamin C in the diet of the crews of the small ships. The first case of scurvy was found in the same quarter of the year as the first case of Vincent's Disease. This is no mere coincidence as I will endeavour to show later. The fact that the scurvy percentage has not risen like the Vincent's infection percentage is, in my opinion, due to the fact that scurvy is caused by diet deficiency alone, while in Vincent's disease there is the additional factor of infection by the fuso-spirillary organism. In suitable hosts there can be an exaltation of virulence of this organism, (Rosebury and Foley (63), Leigh (85) and others). Such an increased virulence of the organism would cause an increasing number of cases if there was an abundance of suitable"soil". The scurvy numbers were greatest in the second quarter of the year, that is after the winter months with their attendant lack of fresh vegetables.

GRAPH SHOWING THE RATE OF INCREASE IN CASES OF VINCENT'S DISEASE AS THE WAR GOES ON.



It will be seen that there have been no cases of beri-beri or pellagra. If a deficiency of vitamin B_1 or the B_2 complex had been concerned in the sailor class with which I deal one would have expected at least a mild case or two of those diseases to crop up. That vitamin B_1 or B_2 deficiencies can predispose to Vincent's disease in some communities has been very well proved by Spies (46), Clements (49), Smith, Persons and Harvey (57), Tunnicliff (64), Sinclair (65), Spies, Hightower and Hubbard (79), King (83), Field (84) and others.

But short of actual pellagra the so-called "pre-pellagrous state" might be present. That such a clinical state does exist is shown by King (83), Elsom (56) and many others. In my study of the cases I looked into this question and A. sought clinical signs of such a state, and B. carried out the latest urinary test for nicotinic acid excretion in the urine as done by Harris and Raymond (67), and C. tried the effect of nicotinic acid on several cases of Vincent's disease.

As regards the relationship of vitamin C deficiency to the disease, I approach the question along the following lines:- A. A urinary excretion test of ascorbic acid was done on the principle of that used by Harris and Abbasy (54), B. the signs of scurvy or sub-clinical scurvy were sought for in each case, C. a "tourniquet test" was performed on each case, D. in some of the cases a saturation with ascorbic acid was maintained as the sole form of treatment or in addition to the routine method used by me, and E. the diet history of each case was taken.

At the outset I thought that perhaps anaemia was the predisposing factor and accordingly did a full blood count when the patient was admitted.

The temperature and pulse rate of each patient was taken as a routine and the urine was tested.

Finally I endeavoured to find out the best method of treatment with the remedies at hand. These included Beaumont's

paint (51) already mentioned, N.A.B. injections, Eusol (1 in 4 with water), hydrogen peroxide, 10% chromic acid, potassium permanganate and potassium chlorate gargles and M. & B. 693. Ascorbic acid and nicotinic acid were also at hand.

What I found to be the best treatment is described later.

A histological study of normal gum, a gum from a case of sub-scurvy and one from a patient with Vincent's Disease was made.

In small ships each man is part of a team and his absence from duty throws extra work on those remaining on board, hence it is important to be able to treat those cases quickly and to discharge them as soon as possible. The results of those investigations will be given.

All the cases were bed cases when admitted, and were diagnosed by having a "positive swab".

This "positive swab" meant that when a slide of a smear from the gum or tonsil lesions was examined, the organisms met the eye straightaway, i.e. there were many fuso-spirillary organisms in each field and a minute hunt for them had not to be made. This was the criterion for a positive diagnosis by Breazeale and Greene (74).

I stained the slides, after fixing with heat, with 1 in 10 carbol fuchsin for five minutes, then they were washed in water

and dried. The fusiform organisms always stain more vividly than the spirilla. Staining by 1 in 10 carbol fuchsin did not give a sufficient contrast for photography so experiments were made with the few simple stains available. It was found that the best method of staining was to use Gram's crystal violet stain for three minutes, then to add Lugol's iodine for three minutes and then to wash the slides lightly with water and to allow to dry. Both the organisms stand out well by this method as the preceding photograph of a "positive swab" shows. (Film 1000X).

Before concluding this introductory chapter I will mention that the attendances at the dental out-patient department in each quarter were as noted in table C.

TABLE C.	TOTAL ATTENDANCES	VINCENT'S DISEASE	% VINCENT'S DISEASE.
Jan. to March 1940	368	17	4.62
April to June 1940	581	18	3.1
July to Sept. 1940	460	15	3.62
Oct. to Dec. 1940	462	40	8.66
Total for all quarters:	1871	90	4.81

The biggest percentage of Vincent's diseases fell in the winter months and there was a striking rise in the last quarter.

Neither this figure nor the figures for the bed cases in Table B. were anywhere near the high figure of 23% in a British Military Hospital towards the end of the last war in 1917, (Bouty (22)), or of the figure of 32% in one unit, given near the end of the war by Semple, Price Jones and Digby (28).

This may be due to the fact that A. my figures are at a comparatively recent date from the onset of war and dietary factors etc have not had time to operate properly, and B. the Naval dental service has functioned efficiently for many years past.

V. HISTOLOGICAL STUDY TO GET IDEAS FOR TREATMENT.

At the outset of this study in the R.N. base hospital I thought that it would be an excellent plan to fix a piece of normal gum and of diseased gum in 10% formalin and to prepare and stain these in order to compare them. The gums in these cases were anaesthetised by a lingual nerve block so that no local anaesthetic would be injected into the tissues to be removed, and a small V-shaped peice of gum was excised with a Bard-Parker knife. In the case of Vincent's disease a piece was removed from a portion of gum which was swollen and red but which had no obvious ulceration or white membrane over it. Another piece was removed from a part of the gum with white membrane on it. This latter piece of tissue was stained by Dobell's method for spirilla, as an ordinary routine smear was "positive".

Dobell's is a bulk method, i.e. the whole tissue is stained en masse as follows. The tissue is first fixed in 10% formalin, then thoroughly washed in distilled water. It is then placed in 60, 80 and 90 % alcohol respectively for three hours in each. Then it is washed thoroughly and left in distilled water for 24 hours. It is now placed in 1.5% to 3% silver nitrate in the dark, in an incubator at 37° for three days. Then it is washed thoroughly in distilled water and placed in 1% hydroquinone for 24 hours in the dark. It must be thoroughly washed before entering the hydroquinone solution. It is finally thoroughly washed again and dehydrated in ascending grades of alcohol up to absolute alcohol. Then it is cleared in chloroform for 4 hours, placed for 12 hours in paraffin, embedded and cut.

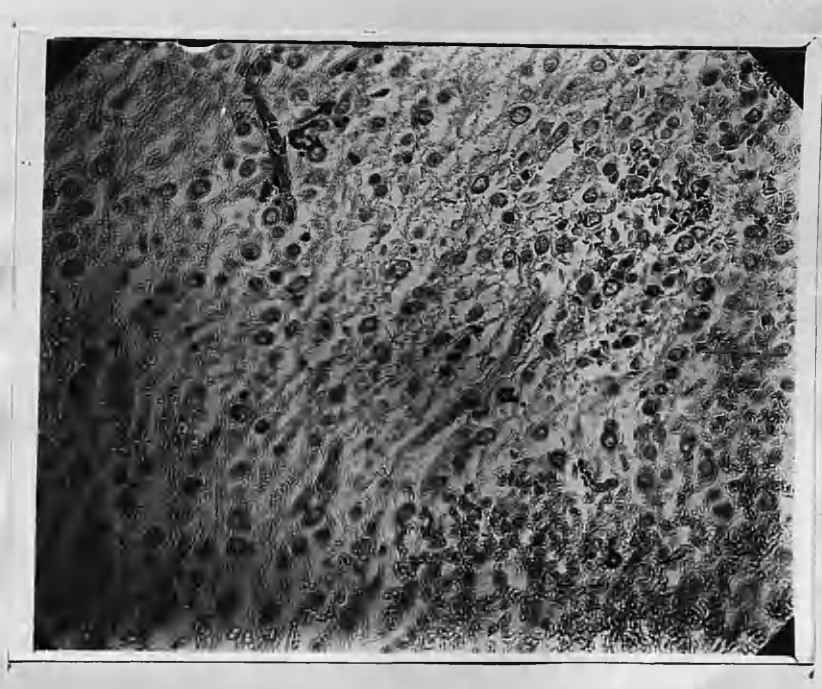
Normal gum and the tissue from the red swollen gum with no obviously diseased surface were fixed and stained in the usual way with haemalum and eosin. Photographs of the three sections were made.

1. Normal gum. L.P. H.& E. 100X

The surface epithelium is similar to that of the skin with papillae but no glands and no hair follicles.

There is no inflammation in the deep layers and the sub-epithelial layer is not diseased.

2. H.P. Swollen gum with no membrane over it.
H.& E. 250X.



The layer of tissue beneath the epithelium i.e. the area marked with an X in the preceding photograph, was focussed and photographed. Pathological changes are at once evident. The capillaries are distended with blood and at one point (a.) a capillary rupture is evident. Red blood corpuscles are free in great numbers in the tissues (b.) and have escaped by rhexis from the capillaries. The typical signs of acute inflammation are absent, i.e. the presence of numbers of polymorphonuclear leucocytes or pavementing of the arterioles or capillaries with these cells. There are large numbers of histiocytes (c.) in the tissues however. This type of swollen gum liable to bleed on the least friction is similar to that found in scurvy.

3. H.P. Diseased tissue stained by Dobell's method. 1000X

Dudley Buxton (97) says that the fusiform bacilli are on the surface layers and that the spirilla are in the deeper layers. Although the routine swab was strongly positive in this case no spirilla were found in the deep layers. However a necrotic area was present on the tissue's surface and a few fusiform bacilli were evident in it. One (a.) is shown in the accompanying photograph in the midst of the necrotic surface layers. In the excision of the gums and during subsequent preparation most of

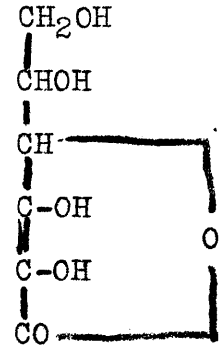
the surface membrane has been removed. Tags remain at places.

I had hoped that spirochaetes might be found deep in the tissues but they evidently lack the penetrating power of treponema pallidum. My findings agree with those of Dudley Buxton (97) as regards fusiform bacilli. However it was felt that in treatment it would be logical to give intravenous arsenic in preference to local application as the tongue of the patient automatically licks round the gums and removes most of a local drug as soon as it is applied. Also with an intravenous medicament the drug would be slowly released into the tissues shown by the previous photographs to be swollen and hyperaemic and there would be a concentration of it exuding at the damaged surface layer and available for a longer period than if it were locally applied. Results with general and local N.A.B. seem to bear out this theory as will be shown later, but local treatment, not necessarily arsenic, is required in addition.

Rosebury and Foley (63), and others, believed that intravenous arsphenamine was not very efficacious. I have seen many cases, resistant to local measures, which cleared up with intravenous N.A.B.. This is especially true of tonsillar affections.

VI. THE URINE TEST EMPLOYED FOR VITAMIN C.

Vitamin C is water soluble and is found in animal tissues and in the juice of fresh fruit especially in oranges. In 1928 Szent Gyorgyi showed that the suprarenal cortex had in it a carbohydrate derivative with the formula $C_6 H_8 O_6$. This was hexuronic acid and was found to be a powerful reducing agent. In 1932 Szent Gyorgyi suggested that hexuronic acid should be named ascorbic acid and he said the substance was probably identical with vitamin C. (Coward and Morgan (48)). Gyorgyi's conclusions were drawn from feeding experiments on animals whose diets were deficient in vitamin C. It is now definitely accepted that ascorbic acid is the actual vitamin. Ascorbic acid whose formula is shown, is sold in the form of small white tablets, each tablet containing 50 mgms..



Harris and Abbasy (54) in 1937 described a simplified procedure for the vitamin C urine test and communicated the method to the Biochemical Society at the Birmingham meeting on December 10th 1937. The method previously described by them for the measurement of vitamin C under-nutrition had involved collection of a 24 hours specimen of urine to determine 1. the "resting level of excretion" and 2. the response after test doses. It is often inconvenient to collect the 24 hours specimen in examining large groups of subjects. Measurements of mere concentration were not recommended since the result depended too largely on the degree of dilution of the urine. They therefore began the following procedure.

At 9 a.m. the patient emptied the bladder. This urine was discarded. There was no more urination till 12 noon when a specimen was taken and titrated. This was repeated on the second and third days. Then at 10 a.m. on the following day 70 mg. of ascorbic acid per stone of body weight were given and that same afternoon the urine was collected at 2 - 5 p.m. and titrated.

Control tests on large numbers of volunteers have proved

that on graded levels of intake the three hours morning specimen represents $\frac{1}{8}$th. of the total day's excretion and is therefore a record of the "resting level". Harris and Abbasy (54) maintain that the collection of the 2 or 3 hours afternoon specimen is adequate to show whether or not there has been any marked response to the successive day's test doses. The morning period is chosen for measurement of the "resting level" since it represents approximately the fasting value. Working on several hundreds of controls, Harris and Abbasy had shown that the "resting level" of excretion as well as the response to the standard test dose are closely graded in proportion to the past intake. The consumption of reputed minimal optimum of vitamin C is 25 mg. per ten stone of body weight and it causes the excretion at equilibrium of 13 mg. per day (resting level) and a response to the standard test dose generally on the first and certainly on the second day.

The firm of Roche Products Ltd. (86) have brought out a dye, dichlorophenol-indophenol, which is used as the indicator in testing for ascorbic acid in urine and other watery solutions. Their method for the quantitative estimation of vitamin C in aqueous solutions is as follows.

One indicator tablet is dissolved in 50 c.c. of water and 5 c.c. of the blue solution (equivalent to 0.1 mg. of vitamin C) is pipetted into a small beaker and about 1 c.c. of glacial acetic acid is added. The colour changes to red.

The liquid to be analysed is run directly from an ordinary burette into the dye solution till the colour is just discharged.

In calculating, if u c.c. of the liquid analysed are required to discharge the 5 c.c. of dye solution then u c.c. contain 0.1 mg. of vitamin C. 100 c.c. will contain $\frac{0.1}{u} \times 100$ mg. In other words 10 divided by u will give the ascorbic acid content in m.g.%.

This analytical method of Roche (86) depends on the fact that the dye dichlorophenol-indophenol is transformed to its colourless leuco-derivative by the reducing action of ascorbic acid (vitamin C). Other reducing substances found in normal urine also decolourise the indicator, but if the method is used in conjunction

with test doses of ascorbic acid it is sufficiently accurate for diagnosis. Thus in diagnosing vitamin C deficiency Roche Products Physicians recommend the following procedure:-

Ordinary urine is taken and its reduction value in mg. % of ascorbic acid is determined. A test dose of 300 mg. of ascorbic acid (6 tablets) is given. The reduction value of the urine passed 3 to 5 hours after the intake of the test dose is now determined and from the two possible alternatives conclusions are drawn. i.e. (a) the reduction value of the urine, expressed in mg. ascorbic acid per 100 c.c., can have risen to double the initial figure but at least to 5 mg. %. In this case deficiency is improbable or so slight that a single test dose sufficed to correct it, or (b) the reduction value of the urine expressed in mg. ascorbic acid per 100 c.c., has not been doubled as compared with the figure found before administration of the test dose. In this case there is vitamin C deficiency.

All the tests for diagnosing vitamin C levels by urinary analysis depend on the fact that supplements of vitamin C are stored by the system and are not excreted until an existing deficiency has been made good, whereupon an excess is promptly excreted in the urine. It is thus possible to recognise the point of saturation which coincides with the correction of the deficiency, by the sudden rise of the reduction value of the urine. Pemberton (77) proved this principle by his experiments with schoolboys. One group of schoolboys (31) received 35 mg. of vitamin C per head per day in their food, while those in another group received 63 mg. The minimum daily intake of vitamin C is put at 40 mg. a day by Pemberton. The boys with an intake of only 35 mg. a day did not respond to the test dose. Those with an intake of 63 mg. did. Pemberton (77) found that the urinary excretion of vitamin C in a well nourished subject showed a sharp rise within 4 to 8 hours of administering a test dose of 50 mg. per stone of body weight.

The test used by me is based on the one by Roche and Pemberton, but most workers agree that 300 mg. of ascorbic acid is too small an amount for a test dose, so 700 mgs. are given. Dr.

Thompson of the Royal Infirmary, Edinburgh, informs me that 700 mg. is the dose now used and my results are calculated as they do in that institution.

The apparatus required is in the accompanying photographs.

1. Three dark stoppered bottles to hold the urine samples.
2. Bottle of glacial acetic acid.
3. Burette stand with 25 c.c. burette.
4. Small white titrating bowl and stirring rod.
5. Conical flask for indophenol solution and 10 c.c. pipette graduated in c.c.s.
6. Tube of dichlorophenol-indophenol tablets.
7. Bottle of ascorbic acid tablets.
8. Microscope and 9.throat swab.(The latter two articles are for diagnosing the condition.)

It is essential to have strict uniformity in the method used in each test. Accordingly I adopted the following procedure in each case.

The patient was in bed and on being awakened at 6 a.m. emptied his bladder and discarded the urine. At 9 a.m. the bladder was emptied into a dark stoppered bottle. Immediately afterwards 700 mg. of ascorbic acid (14 tablets) were given with a drink of water and no more urine was passed until 12 noon. The bladder was now emptied and the urine put in a stoppered bottle. At 3 to 5 p.m. a third emptying of the bladder was done and the urine was again

put into a dark stoppered bottle. To each of the three samples of urine passed 10% of glacial acetic acid was added immediately. This and the dark colour of the bottle prevented the ascorbic acid from being oxidised. Each specimen was taken down to the laboratory immediately and analysed as follows.

One tablet of dichlorophenol-indophenol was dissolved in 50 c.c.s of water in the conical flask. A blue solution resulted. 5 c.c.s of this were pipetted into the titrating bowl and 1 c.c. of glacial acetic acid was added. The solution became red at once. (It is important to wash the pipette after measuring acetic acid otherwise the blue solution in the flask goes red when next measuring out 5 c.c.s of dye.) The urine to be analysed was run into the dye solution from the burette till the colour just became clear. The numbers of c.c.s of urine used was noted. Each specimen was titrated 3 times and the average was taken. (Thre is enough urine in a burette to do 3 titrations as a rule). Calculation was made as advocated by Roche (86).

It is essential to do the titration rapidly and within two minutes and to keep each patient on the same amount of fluids during the day of the test. The first specimen, before the test dose of ascorbic acid, is usually low. The 3 hours specimen can be ignored but the 6 hours specimen is the important one and if the result of it shows 8 mg. % of ascorbic acid or over there is no deficiency. Many persons who have been on a rich fruit diet show 50 to 80 mg.% after the test dose. Persons below 8 mg.% are deficient in vitamin C.

The blood ascorbic acid investigation shows that the normal level is 1 mg. per 100 cc.s of plasma. Ungley (59) showed that a deficiency of vitamin C causes the resting level of ascorbic acid to be low in the plasma and that a test dose only slightly increases it. In a person deficient in vitamin C any extra taken into the body is stored up by the tissues. Only when the body has sufficient does the excess overflow to the urine.

I lacked the necessary apparatus to do blood estimations and when any are mentioned in connection with my cases, they were done in other institutions.

To see how quickly vitamin C deteriorated in urine the following tests were done. I put clear urine (S.G. 1015, acid in reaction, albumin, bile, blood and sugar-nil.) into 3 samples. This urine was rich in vitamin C and represented the urine of a healthy person who had been saturated with vitamin C and who had, 5 hours previous to collecting the sample, taken 700 mg. of vitamin C.

Sample 1. was placed in an ordinary, clear urine jar in the dark.

Sample 2. was placed in a dark bottle which had recently contained hydrogen peroxide and in which there was a damp piece of cork which had been floating in the hydrogen peroxide.

Sample 3. was placed in a black, stoppered bottle and 10% of glacial acetic acid was added.

The three samples were tested at intervals and the results are shown in the accompanying table D.

TABLE D. TIME OF TESTING.	SAMPLE 1. mgs.% vitamin C	SAMPLE 2 mgs. % vit.C.	SAMPLE 3 mgs.% vit.C.
9 a.m. (urine passed)	50	50	50
11 a.m.	33.3	33.3	50
1 p.m.	33.3	16.67	50
3 p.m.	33.3	9.3	50
5 p.m.	33.3	1.19	50
7 p.m.	33.3	0.95	50
9 p.m.	28.3	0.5	50
9 a.m. (next day)	25	0.4	50

Fourteen days later a random sample of urine contained 4.2 mgs. % of ascorbic acid, none of which had been taken during this period. The 6 hours specimen of urine after 700 mgs. of vitamin C rose however to 35 mgs.% ascorbic acid, showing that there was really no deficiency of vitamin C in the body. All this time the subject (myself) was on a diet which was poor in vitamin C and contained no fresh vegetables. Sample No. 3 was kept in the dark bottle with 10% glacial acetic acid in it and

on the 14th day contained 25% of ascorbic acid. These results show that ascorbic acid slowly deteriorates in ordinary urine in the dark. When stoppered and kept in the dark with 10% glacial acetic acid added, the deterioration is not so marked and certainly not on the first day. Dirty receptacles i.e. the bottle with the hydrogen peroxide cork in it, cause a rapid oxidation of the vitamin C content even if the bottle is dark in colour. The results also justify my collecting of urine in dark bottles, the addition of 10% glacial acetic acid and the testing of it immediately a sample was obtained.

VII. THE METHOD USED TO DETERMINE THE LEVEL OF VITAMIN B_2 IN THE BODY.

Many workers, as has been shown in the initial historical outline, believed that in some localities vitamin B_2 deficiency was responsible for the commencement of Vincent's disease. Spies (46), Clements (49), Smith, Persons and Harvey (57), Tunnicliff (64), King (83), Field (84) and many others were among this lot.

While vitamin B_1 is the anti beri-beri vitamin and while it is suggested that alcoholic and other forms of neuritis are predisposed to by deficiency of vitamin B_1. (Coward and Morgan (48)) it is well known that vitamin B_2 has no anti-neuritic properties. Vitamin B_2 was known to protect against pellagra with its gastro-intestinal upsets, severe dermatitis and chronic dementia and vitamin B_2 was after its isolation found to be identical with lactoflavin and to be present in whey, egg-albumin, egg-yolk and liver.

The latest work on the subject shows that "vitamin B_2" is a complex of at least three factors, i.e. lactoflavin, vitamin B_6 the so-called "rat pellagra factor" and P.P. the human pellagra and canine black-tongue factor.

The most recent work indicates that the P.P. factor is related to or identical with nicotinic acid (? $C_6 H_5 NO_2$.) (Leslie Harris (60)).

It is now established that nicotinic acid is concerned in the prevention of pellagra. A short history of the splitting of vitamin B into B_1 and B_2 will not be out of place.

Funk in 1912, discussing pellagra, thought it might be a deficiency disease but his views were not recognised from 1916 to 1926 owing to the doctrine of Goldberger, an American, who believed it was due to poor quality protein in the diet. However by 1926 Goldberger had himself realised that pellagra was a vitamin deficiency disease. At this time a factor called "vitamin B" was known and Goldberger showed that the factor concerned in curing pellagra had a distribution similar to "vitamin B". Goldberger concluded that ordinary preparations of "vitamin B" had two factors at least in them, i.e. 1. the old anti-neuritic factor and two the new anti-pellagra vitamin. One reason for Goldberger's conclusions was that yeast was found to be both an excellent anti-neuritic remedy and an excellent anti-pellagra supplement. Yeast however, after autoclaving was no longer anti-neuritic though it still had anti-pellagra properties. Also he noted that some maize meal extracts were rich in the anti-neuritic vitamin but poor in the anti-pellagra factor. Further tests on dogs and cats confirmed the dual nature of "vitamin B".

Thus to date vitamin B was found to be split into a heat-labile, anti-neuritic principle and a heat-stable component or complex as we now believe it to be. This heat-stable complex had four distinctive properties

1. It prevented pellagra in man.
2. It prevented black-tongue in dogs (a disease apparently the same as pellagra).
3. It was necessary in a rat's diet for growth and health.
4. It prevented a pellagra-like dermatitis in rats.

Goldberger wrongly assumed that the four properties were all due to the"pellagra-preventing factor".

It is important to note that nicotinic acid which we now know to be part of the B_2 complex is heat-stable. Nicotinic acid

according to McLester (70) has the formula $C_5 H_4 N (COOH)$ and is the beta-monocarboxylic acid of pyridine. The important thing from the point of view of this thesis is that it can be boiled without loss of potency, unlike vitamin C with which 60° C. for one hour kills 80% of the vitamin. (Tidy (69)). Also vitamin C, soluble in water, is rapidly destroyed by alkalis and drying.

Harris and Raymond (67) have done a great deal of work on the excretion test for nicotinic acid. Some of my cases had their nicotinic acid estimations done by the method these workers have elaborated. The normal nicotinic acid excretion per day is 4 to 7 mgs., with the average excretion per day as 6 mgs.. The figure rises with a high meat diet. Some cases were examined where the persons were very heavy smokers. The output was 7 to 13 mgs. per day, but Harris and Raymond were inclined to think that as a general rule, smoking had very little effect in raising the output of nicotinic acid.

These workers examined persons deficient in nicotinic acid and found that cases of pellagra, persons on low diets, and cases of myasthenia gravis had 2 or under, mgs. per day, of an output.

The test has been proved to be reliable from a vast amount of experimental work with guinea pigs. As a general rule about one-fifth of a person's total daily intake of a vitamin is excreted in the urine. Working on this general rule one would expect that 25 to 35 mgs. of nicotinic acid would be ingested daily in the average case.

In doing the test all the subjects, who were kept in the ward, were put off smoking for 3 days previously. A 24 hours specimen of urine was taken and the amount measured. The test is as follows:-

"25 ml. of urine are heated with 5 ml.of 20% sodium hydroxide solution for 30 minutes in a steam bath. 2 ml. of 4% sodium bicarbonate solution are added and the solution is carefully neutralised with concentrated hydrochloric acid to pH 6 - bromo-thymol blue is used as an external indicator. The whole is quantitatively transferred to a 50 ml. graduated flask

and made up to 50 ml. with distilled water. Four labelled 15 ml. volumetric flasks are prepared into the third and fourth 20 µg. and 40 µg. of nicotinic acid are pipetted accurately, using a standard solution containing 100 µg. of nicotinic acid per ml. To all of the flasks 10 ml. of the neutralized urine are added; the concentrated nicotinic acid solution in the third and fourth flasks is carefully washed down with the urine. The flasks are immersed in an opaque sided water bath at 80° C. for ten minutes; 2 ml. of a freshly prepared solution of cyanogen bromide (made daily by adding a 10% aqueous potassium cyanide solution drop by drop to a saturated bromine water until it is just decolourized) are added to all except the first flask, mix by rotation and keep at 80° C. for 4 minutes. Remove to a cold water bath in dim light and cool for 4 minutes. Add 0.2 ml. of p-aminoaceto-phenone solution (5 gm. p-aminoaceto-phenone, 14 ml. of 10% hydrochloric acid and distilled water to 50 ml.) to all four flasks, mix and leave in a dark cupboard for 15 minutes. To each flask is than added 0.4 ml. of 10% hydrochloric acid solution and water to 15 ml. and the flasks are returned to the dark cupboard for 15 minutes. The colour is measured in a Pulfrich photometer. If a turbidity develops at any stage it should be removed by centrifugation before proceeding. Urines may be preserved with sulphur-free redistilled toluene. "

In the photometer we use the blue filter S47. The purple one, S 43, is too dark.

PULFRICH PHOTOMETER.

Two 3 cm. cells are used in the instrument. One called "x" has the urine with no cyanogen bromide nor nicotinic acid, and it is placed in the left hand compartment. In the right hand compartment the other cells are placed, one at a time, and the scale is set at the 100 mark. These other cells have:-

(a.) Just cyanogen bromide (0).

(b.)0.2 c.c. of nicotinic acid plus cyanogen bromide ("the 20 cell").

(c) 0.4 c.c. of nicotinic acid plus cyanogen bromide ("the 40 cell").

i.e. we put the 0, 20 and 40 microgram cells in, one at a time, into the right hand side, with the drum at 100 and take the readings after balancing the colours with "x".

Suppose the readings are 82 with "0", 44 with "20" and 25 with "40".

Harris and Raymond calculated their results by plotting the extinction coefficient against the nicotinic acid added and obtaining a straight line which when produced to transect the abscissae gave the concentration of nicotinic acid in µg. of the urine.

I work it out as follows. For easy comparison the results are written as:-

X = 82 with 0 at 100

X = 44 with 20 at 100

X = 25 with 40 at 100.

Now take the logarithms of the readings 82, 44 and 25 by a log-table. i.e. they are 1.9138, 1.6435 and 1.3979.

Now subtract each of these from 2 to get it to a decimal i.e. 0.0862, 0.3565 and 0.6021.

Now subtract the 0.0862 (i.e. the cell with no nicotinic acid in it) from the other two readings (i.e. the 20 and 40 microgram cells).

The results are 0.2703 (for the "20" cell) and 0.5159 (for the "40" cell).

Now multiply 0.0862 (the cell with no nicotinic acid) by 20 and divide by 0.2703.

Also multiply 0.0862 by 40 micrograms and divide by 0.5159. These two results are expressed as micrograms per 5 c.c. i.e. "20" had 6.378 and the "40" had 6.683. The average is taken and is 6.53 micrograms per 5 c.c.

Now we calculate the percentage and the total amount excreted per day.

VIII. FINDINGS FROM THE CASES OF VINCENT'S DISEASE.

Having described the ways in which I have approached the problem of Vincent's Disease let us now turn to the findings in the actual cases. A brief description of each case is given later.

VITAMIN B DEFICIENCY.

Let us consider the question of vitamin B deficiency as a predisposing factor in this class of patient.

Spies (46), Clements (49), Smith, Persons and Harvey (57), Tunnicliff (64), Sinclair (65), Spies, Hightower and Hubbard (79), King (83), Field (84) and many others all state that in certain localities a deficiency of vitamins B_1 or B_2 exists and lays the gums open to invasion by the fuso-spirillary organism.

King (83) emphatically states that C deficiency is not the cause and even describes the "pre-pellagrous state". Furthermore he states that true pellagra can exist in temperate climes and that it is characterised by an absence of skin lesions. All writers on pellagra or the pre-pellagrous state agree that diarrhoea is a constant accompaniment. Of my 55 cases not one had this complaint, indeed, 22 tended to be constipated. Other findings which are described in the pre-pellagrous state are stomatitis and glossitis and a characteristic desquamation of the tongue. The latter two signs were absent in all of the 55 cases. Stomatitis is a sign of the malady itself.

Elsom (56) talking of vitamin B deficiency in general, stated that its symptoms and signs were referable to (a.) the gastro-

intestinal tract, (b.) the nervous system, (c.) the blood, and (d.) to the cardio-vascular system in certain circumstances. McLester (70) confirms this fully. It has already been shown that there was no diarrhoea in any of the cases. Four had "gastritis" and one had a history of "indigestion" on admission. This so-called gastritis consisted of ascending flatulence and heartburn and a feeling of discomfort after meals and could have been accounted for by the constant swallowing of pus and foul material from the gums. One case was admitted with a perforated peptic ulcer which could likewise be accounted for.

No one of the cases had peripheral neuritis or vague aches and pains such as one gets in vitamin B_1 deficiency. The blood pictures were variable and not characteristic of vitamin B deficiency and there was no clinical evidence of dropsy or myocarditis.

Thus from the cases seen there was no clinical evidence of a lack of vitamin B.

An interesting report appears in the Journal of the R.A.M.C. (25). An outbreak of several hundred cases of true pellagra occurred in Turkish prisoners in prison camps during the last war. Germans in neighbouring camps on the same rations escaped because they received extra money from their consulate and supplemented their rations with it. None of the British troops or hospital orderlies took it. The Observers concluded from the wealth of clinical material available, that the causes of pellagra are in operation for 4 to 6 months and that there ensues a clinically pre-pellagrous stage during which apparently healthy persons gradually approach the extreme limit of possible adjustment between normal energy expenditure and abnormal malnutrition. Soon any disturbance of the precarious balance, e.g. by extra work or further digestive derangement rapidly converts the subject into a clinically definite pellagrin.

Let me again state that in the sailors of the small ships there were no signs of the pre-pellagrous state and no cases of pellagra itself came into the hospital. Nor were there cases of beri-beri seen.

In the R.A.M.C. (25) investigation, very full reports of the bacteriological section, of the protozoal section and of the section who carried out the post-mortems all failed to mention the findings of fuso-spirillary infection. Thus it could not have been there with any constancy or it would have been mentioned. Indeed the protozoological section states that "There is no evidence of infection by any protozoal, spirochaetal or ultra-microscopic organism standing in aetiological relation to pellagra."

While this committee was hunting for the cause of pellagra, nevertheless it serves to show us that Vincent's disease is not a constant accompaniment of pellagra by any means.

Chlorotic anaemia was also a constant accompaniment of those cases of pellagra. A study of the blood pictures of the 55 sailors will show that this is not the case here.

THE DIET. One naturally turns to the diet of the sailors and more will be said of it later in connection with vitamin C.

Nicotinic acid guards against pellagra, (McLester (70)), and deficiency of it has been chiefly blamed for laying the way open to the invasion by the Vincent's organisms. Spies, Grant, Stone and McLester (61) have demonstrated the effectiveness of nicotinic acid in preventing pellagra. The acid is found naturally in yeast, liver, lean meat and milk, and it is quickly absorbed from the gastric tract even in the presence of violent gastritis and stomatitis (McLester (70)). Although the diets were monotonous, meat was plentiful in them all and I can vouch for this from personal experience. There is no lack of meat in the Naval diets. Moreover McLester (70) states that nicotinic acid is unusually heat-stable and can be boiled without loss of potency. Thus there is no deficiency of nicotinic acid in those sailors' diets.

CHEMICAL TESTS. The next thing to consider is the table of results obtained from the analysis of the urinary excretion of nicotinic acid. As it is a long process and time is limited

23 of the 55 cases were examined thus. The results are shown in table E.

TABLE E. CASE NO.	Milligrams of Nicotinic Acid excreted per day.	Deficiency or otherwise.
1.	6.31	No
2.	9.3	No
3.	8.8	No
4.	2.87	Doubtful deficiency
5.	3.83	No
6.	13.13	No
7.	9.6	No
8.	8.34	No
9.	11.15	No
10.	8.75	No
11.	8.2	No
12.	6.3	No
13.	5.1	No
14.	4.14	No
15.	8.0	No
18.	6.3	No
19.	2.87	Doubtful deficiency
21.	5.0	No
23.	6.38	No
24.	4.75	No
40.	5.14	No
46.	4.35	No
57.	3.42	No

Harris and Raymond (67) state that the normal nicotinic acid excreted per day is 4 to 7 mg.. Pellagrins and people on a low diet have a value of 2 or under. While two of the cases have the value of 2.87 mg. excreted per day only two others are below the 4 mg. figure. The majority of the cases (82.6%) are well within the normal limits and some (cases 9 and 6) show very high values.

From these results we may take it that the sailors of the small ships have ample vitamin B_2 in their diets and that in this class of men deficiency of nicotinic acid can be ruled out as a predisposing factor in Vincent's Disease.

The results of using nicotinic acid in treatment will be described later but here it will suffice to say that it does not cure the disease if used alone in the absence of local treatment, and that with local treatment there is no acceleration of cure using nicotinic acid in this class of patient. This is in direct contrast to the findings of King (83).

VITAMINS A AND D.

In the Great War of 1914-18 the Danes and Swedes exported most of their butter to belligerent powers and left themselves short of that food. As a result there were outbreaks of xerophthalmia and keratomalacia in children's schools and the disease rapidly healed on giving vitamin A. It is well known that deficiency of those vitamins is reflected in the eyes and I have recently seen in the West Coast, a case of a man whose corneal ulceration cleared up with "Adexolin", though it had been resistant to local remedies. He was on a shore station and he had no Vincent's Disease. On the other hand none of the 55 cases written up had any signs of eye trouble.

Furthermore at the time when the cases were seen, butter was very plentiful and in addition many of the men had fish in their diets and there was fat on the meat. Carrots are common on shipboard too as any sailor will agree. Even now, with an admixture of margarine (which contains vitamins A and D) in the butter, the diet has sufficient vitamins A and D. Carrots, fats and butter are all rich in vitamin A (Beaumont and Dodds (68)).

Indeed with the open air life the sailors lead and the abundance of sun in the summer months, ample vitamin D would be manufactured in their bodies. There is definitely no cause for supposing that shortage of vitamins A and D was the reason for the occurrence of Vincent's disease on our small ships as

is claimed by Field (84), Kirkpatrick (66), Sinclair (65), and others in other communities. Indeed Jeghers (53) has shown us that it is possible for different classes of people to lack vitamin A but this has no bearing on this case.

VITAMIN C.

Let us now turn to the question of vitamin C deficiency. In the first place it is significant that the hospital had 9 true cases of scurvy from January to December 1940. Those men were all from the small ships with which I am dealing and beyond doubt there must have been other cases on board in a low state of vitamin C as they of necessity were on the same diets as the scurvy cases.

My next point is that there is no real, clinical "subscorbutic state" with constant symptoms. The tourniquet test for capillary fragility is probably concerned with vitamin P (hesperidin), although Bell, Lazarus and Munro (76) reduced raised petechial counts in eight weeks with doses of vitamin C. Of the 55 cases I mention, though almost all showed a deficiency of vitamin C by the urinary excretion test previously described, only 7 had a"positive tourniquet test". This test is of no value in my opinion, in estimating the subscorbutic state, for other patients with negative tests had the same or lower figures for ascorbic acid excretion as the "positives".

The figures from the urinary excretion tests show that 46 out of 55 cases were definitely deficient in vitamin C on admission. Of the remaining 9 cases, numbers 10, 18, 24, 33, 41 and 47 had been found deficient in, and were treated with, ascorbic acid before admission and thus were not deficient when seen by me. However at the outset they were really deficient because the blood ascorbic acid values of cases 33 and 41 were done at another sick quarters and were .26 and .21 mg.% respectively.
Only cases 26, 34 and 45 had no previous vitamin C administered and were not deficient on admission. Thus of 55 cases, 52 or 94.55%, were deficient in vitamin C by the urinary excretion tests and of

these the values of vitamin C in the urine specimens were so low for the most part that there is no doubt of the sub-scurvy state in which the patients were.

However the matter cannot be dismissed so lightly. Could it not be that it is the presence of infection that causes the vitamin C to be so low in those cases? In my opinion the answer is "no". It is obviously difficult to have a person under observation on a constant daily rate of excretion of vitamin C and then to see the result when Vincent's infection developed; on the other hand there are many other cases described where true gingivitis only, has occurred due to vitamin C deficiency and where there was no question of Vincent's Disease superimposed which might lower the vitamin C level. Indeed those cases respond to vitamin C only. (H. Gordon-Campbell (98), Roff and Glazebrook (82) Weisberger, Young and Morse (96) and others). Then again we do see cases, as witness numbers 26, 34 and 45, where there is no deficiency of vitamin C in spite of the oral sepsis but where the local trauma in the form of caries and tartar has itself caused the initial lesion which allows the organisms to enter. Thus oral sepsis does not always lower the vitamin C level. Also I have seen out-patients with Vincent's Disease who have had a low level of vitamin C and who, when all sepsis had been abolished by the 10% chromic acid and intravenous N.A.B. treatment, went back on board ship and were still deficient in vitamin C two or three weeks later. This shows that we are concerned in this class of patient with a true diet deficiency since there is now no sepsis to deplete the vitamin C after treatment.

DIET. The dietary history is of paramount importance in this connection. Perusal of the diets of the cases shows that many lack the articles of food which contain vitamin C. Some diets appear plentiful but a knowledge of conditions aboard is necessary to see that what vitamin C is originally in these apparently good diets is soon largely destroyed. McLester (70) says that vitamin C is easily destroyed by contact with heavy metals, exposure to air, high temperatures, disruption of cell structure alkulinity and exposure to light. Tidy (69) states that 60° c. for one hour kills 80 % of vitamin C and that it is rapidly destroyed by

alkalis and drying.

In most ships the mid-day meal is prepared and cooked immediately after breakfast and is kept at a high temperature several hours before it is served. Usually the food is overcooked and is served up so hot that it is uneatable.

Any person who has been many weeks at sea in a small ship will tell of the monotony of the diet as a rule. As a result appetites flag and the maximum amount of benefit is not derived from the food. It is usual to be able to say "this is Sunday, there will be pork for dinner; or to-day is Wednesday, there's tinned herring for breakfast". This regularity, with limited food, and little variation, gets very monotonous.

In a small ship too, one usually finds a system known as "canteen messing". Each man in the mess subscribes say 1/8d. per day and one of the Mess Committee buys the food at the canteen. Often his choice is limited. Naturally the members of the mess try to save from the "messing" and some messes each save as much as 10s. per head, per month. Obviously the more they save the less food they get.

Then again with this system there may be regular cooks who are good at their jobs but very often the members of the mess take it in turn to do the cooking for their shipmates. I quote the saying of case number 25, "If a fellow was good enough to make a duff, there was a duff," to show to what extent they depended on each other's capabilities to get a good meal.

Since November 1940 it has been increasingly difficult to get apples or oranges and indeed fruit of any kind was rarely eaten by the ratings unless they were supervised. Some ships get an issue of limejuice. Hamilton (30) as long ago as 1919 carried out experiments with guinea pigs and Naval limejuice. All his control guinea pigs fed on bran and heated milk only, died of scurvy in just under 60 days. Guinea pigs fed on bran and heated milk plus 1 c.c. of Naval limejuice died of scurvy in an average of just over 50 days. The same applied to guinea pigs fed on bran and heated milk plus 1 c.c. of "Supply and Transport" limejuice one year old.

Guinea pigs fed on bran, heated milk and freshly prepared, pure limejuice lived at least 70 days and two lived to and were well on the 84th day. Hamilton said "There is I think, very strong evidence that the Naval limejuice is of little or no value as an anti-scorbutic".

Gittings (31) in 1919 also stated that, in the quantity supplied onboard, limejuice was merely a pleasant summer drink. His remarks too, that green vegetables onboard were supplied by the canteen and were subject to the laws of supply and demand and that often the demand was not made by the mess caterers, apply to-day as has been shown above. He was not considering Vincent's Disease at all.

I took several samples of Naval limejuice as supplied onboard and tested it against 5 c.c. of dichlorophenol-indophenol as in the urinary excretion test.
The average number of c.c.s necessary to discharge the red colour was 23. Thus Naval limejuice contains about 0.43 mg.% of ascorbic acid, which is not sufficent in the amounts of limejuice taken.

Perhaps the most important factor in lowering the vitamin C content of the diets is the manner of cooking. In most ships the mid-day meal, the main meal of the day, is cooked immediately after breakfast and is kept hot for about three hours before being served up. Sometimes the food is even cooked overnight and reheated for the next day's meal. This custom prevailed in 1919 (Gittings (31)) and is still evident to-day.

Then again many of the vegetables are tinned and are all heated for a long time before use. Even tinned fruit is used and in many of the small ships this too is absent on most days.

It is rare to get fresh milk on shipboard. Greens, such as cabbages and sprouts, are often two weeks or so old when cooked and have largely dried up, thus further impairing their vitamin C content. (Tidy (69)). The nutrition information bulletin (99) stresses how vitamin C deteriorates with storage and drying of vegetables.

Taking it all over and on considering that 9 true cases of scurvy cropped up in the hospital it is certain that many of the small ships' companies lack the necessary supervision of their

diets and in consequence are in a low state of vitamin C concentration. Indeed I have seen officers and men with swollen, bleeding gums which responded to ascorbic acid only, in a matter of 2 weeks and in those cases there was no question of Vincent's disease. Roff and Glazebrook (82), and H. Gordon Campbell (98) have seen similar cases, the former on shipboard.

In contrast to the majority of cases numbers 26, 34 and 45 showed no deficiency of vitamin C. The latter two had meals ashore and all had fuller diets than the majority of the cases. All had fruit too, in their diets. Other factors as will be seen later operated in causing the disease.

Harris (78) examined 35 schoolboys from a poor district by the test dose method and found 14 to be below the standard level of vitamin C nutrition (below a daily intake of 40 to 50 mg.). Five showed a relatively severe deficiency. By contrast, at a home where the diet was exceptionally good, none of the 29 boys examined was below standard. I quote this to show the parellel to my experience of ship cases. Cases 26, 34 and 45 correspond to the 29 boys from homes where the diet was exceptionally good. The reason the sailors in the small ships lack the good diet is largely through ignorance. When they are properly guided as is shown later, they remain healthy.

BLOOD PICTURE.

TABLE F.	HIGHEST COUNT	LOWEST COUNT
1. R.B.C.	5,250,000 per c mm.	3,280,000 per c mm.
2. H B%	103	64
3. W.B.C.	16,000 per c mm.	3,200 per c mm.
4. Polymorphs.	86%	40%
5. Monocytes	7%	0%
6. Lymphocytes	53%	11%
7. Eosinophils	3%	0%
8. Basophils	1%	0%

I will state briefly that I found no constant feature in

the blood counts of my cases which might be classed as a cause or as an effect of the disease. Stine (73) had similar findings. Table F shows the upper and lower limits of the various counts.

While some of the cases had a slight degree of anaemia the great majority could not be said to be anaemic. Also, there was no constant calling forth of leucocytes in this type of infection. Again it will be seen that the cases were not those of the anginose type of glandular fever described by Fuller (92) or Stuart-Barber (93).

OTHER STATISTICS.

In each of the cases a careful note was taken of whether or not they had caries or tartar, as in my opinion this has a very important bearing on the cause.

Table G shows the results.

TABLE G.	TARTAR.	CARIES.	TARTAR + CARIES	STAINING ONLY.	TOTAL.
	17	10	27	1	55

The case marked "staining only" had a dark brown, basal staining associated with unbrushed teeth. Many of the cases as will be seen from the case histories, had fillings, closely crowded teeth, and dentures. Thus tartar and caries as we see from the above picture form a constant accompaniment of our series.

It has been said that smoking predisposes to Vincent's infection. Table H. gives the figures of the smokers and the drinkers of beer or spirits.

TABLE H.	SMOKER	DRINKER	BOTH	NEITHER	TOTAL
	12	12	26	5	55

Many of those cases as will be seen from the histories, smoke and drink in moderation only and in my opinion the figures are about equal to the figures in the same class of men who are not affected with Vincent's disease. There is no ground after reviewing the table for saying that smoking or drinking are predisposing factors when, as has been stated, this is only in moderation.

IX. PROBABLE MODE ON ONSET OF THE DISEASE.

Having observed the many out-patients who arrived with Vincent's disease and the in-patients with the malady, I drew conclusions as to how the disease started in this class of seafaring men.

McKinstry (24) showed that the Vincent's organisms were in the throat smears of 2.43% of soldiers and in the gums of 41.3% of healthy civilians. Again McKinstry (20) found the organisms in 95 out of 230 healthy men i.e. 41.3%.

Semple, Price Jones and Digby (28) found that in random hospital cases 95.5% had fusiform bacilli while 95.3% had spirochaetes in their mouths.
Breazeale and Greene (74) in 1940 found the organisms in 35% of throat and gum smears from ordinary individuals.

I swabbed the gums of 100 random patients, not with Vincent's disease and not with oral conditions, taken at random from the sailor class with which I deal and found that 63% had the organisms present. These of course were not present in the numbers found in a "positive swab" but on careful searching one or two of the organisms could be found in the oral debris.

The next point is that these organisms cannot all be virulent otherwise there would be more cases of the disease in the community as a whole. Field (84) stresses the possibility of strains with different virulence while Rosebury and Foley (63) believe in the possibility of exultation of virulence by passage. Certainly with other organisms there are varying strains of virulence as a rule e.g. streptococci, K.L.B. etc.; so there is no reason to doubt that organisms of the fuso-spirillary class are otherwise placed.

In one trawler-minesweeper there were 21 out-patients with Vincent's disease within the space of a few weeks. Three of them were officers in a separate mess. The ratings had one cup between 40 men. The other cups had been stolen by dockyard workers when the ship was refitted or had been broken in action. This one cup was passed round and often was not washed between

individual drinkers. The officers became infected through their steward who contracted the disease in the ratings' mess and who had also been using the officers' utensils. This was an ideal case for observation and the measures taken to stamp the disease out were to get individual cups and to ensure that these and other utensils, were boiled before use. Then the men were mustered three times a day and made to gargle with potassium permanganate and to brush their teeth, under the eye of the First Lieutenant. No fresh cases appeared. Existing cases were meantime vigorously treated.

This was evidently a case where virulent organisms attacked susceptible persons and proves without a doubt the contagiousness of the complaint. There may also have been exaltation of virulence by passage.

Wingrave (15) said that the belief in contagiousness was not supported by clinical evidence although Leigh (85) stressed contagiousness by fomites. Many small ships never had a case of the disease on board.

In my cases paddle minesweeper (X) had three cases, paddle minesweeper (Z) had three cases, paddle minesweeper (Y) had three cases, trawler (Z) had two cases, destroyer (A) had three cases and so on. These were bed cases and at the same time there were several cases attending as outpatients from each of those ships. Thus again we see that some factor like contagiousness of virulent strains which acted on susceptible soils, must have been present. And here let me mention that it is difficult to infect healthy gums in a healthy adult, even with virulent material from a case of Vincent's disease. (King (83)).

One might ask if it were not possible that a person developed Vincent's disease whenever his vitamin C level fell below a certain value. I think the answer is "no" because many men with low vitamin C values who have the organisms in their mouths in small numbers, do not develop the malady. Also 3 of my cases have no deficiency of vitamin C. In my experience a traumatizing factor, such as caries or tartar, is necessary as well. I think that the role played by vitamin C deficiency is to

make the gums swollen and tender and more prone to injury, and to undermine the patient's powers of resistance generally.

Then again on another trawler, there were 5 cases attending at the same time as out-patients. Here there was one cup for 19 men. Several other sailors from this ship attended to have fillings of teeth or dentures repaired and were found to be free from disease and to have otherwise clean mouths. Presumably they had been infected under the conditions aboard but they had not taken Vincent's disease proper. i.e. the other factors necessary besides the presence of the organism, were not there. Similar use of permanganate and brushing under supervision, as was done above, prevented new cases.

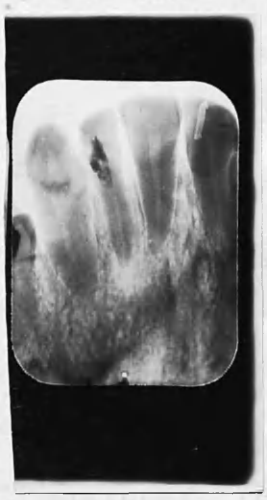
Having given by those two examples, obvious proof that contagiousness is present, let us now consider what are the other factors which operate in a case, since not all on board take the disease.

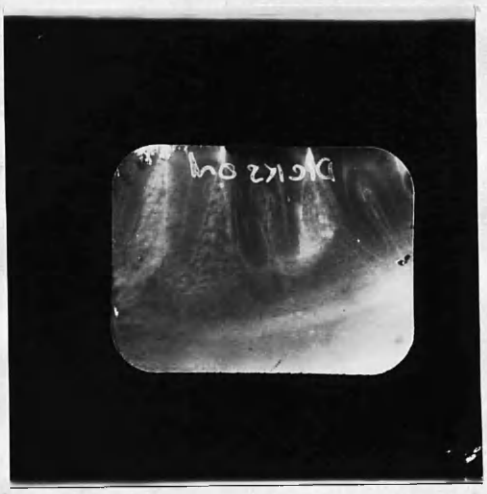
A survey of the 55 cases has shown two constant factors, namely sub-scurvy and caries or tartar.

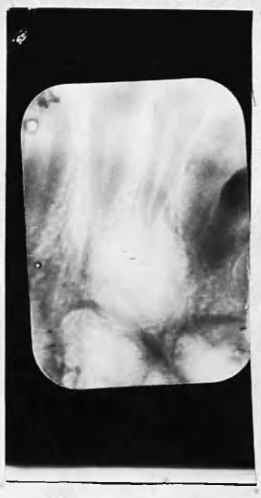
If vitamin C is lacking for 2 to 4 months, completely, scurvy will develop in an adult (McLester (70)). Short of actual, clinical scurvy, the subscorbutic state can exist. (Hess (32), Dalldorf and Russell (47), and others). In this state the gums become tender and swollen and bleed easily on pressure. I personally have seen those cases and where the mouths are clean and the dental toilets are thorough, the condition will respond to ascorbic acid and return to normal in about 2 weeks. This statement is borne out by the findings of Roff and Glazebrook (82) and H. Gordon Campbell (98).

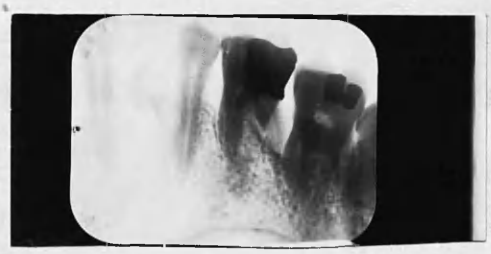
The presence of a ring of hard tartar at the tooth bases or of a carious tooth, affects the situation in two ways in my opinion. Firstly it offers a medium where the Vincent's organisms, often virulent, abound and breed; and secondly it traumatizes the swollen, tender gum and causes a breach of the surface where the organisms are introduced directly into the tissues. In most cases of Vincent's Disease one can see a hard ring of tartar (however small) at the teeth bases, overlapped by swollen, spongy gum, and

this tartar can be picked out from its pocket. In my mind I see as a parallel the case of intestinal diverticulosis where a diverticulum is harmless till it comes to contain a mass of impacted faeces, pregnant with organisms. This by pressure erodes and inflames the wall of the diverticulum, setting up a true diverticulitis.









Alveolar abscesses form sites where the organisms of Vincent's disease lodge, as do overcrowded teeth or the crevices connected with teeth fillings. The accompanying photographs were taken from outpatients with the disease and show in what positions the spirilla and fusiform organisms might lurk. Alveolar abscesses with pus, capable of infecting tender gums, are shown, and fillings with crevices which hide the organisms. The ordinary aerobic organisms in an abscess will use up the available oxygen and allow the anaerobic fuso-spirillary organisms to flourish till they

get a breach of gum surface to invade. (c.v. the onset of tetanus and gas-gangrene in the depths of suppurating, lacerated wounds).

The cases we see where no caries or tartar exists, have the trauma applied in other fashions as for example by the accidental stabbing of the gum with the prong of a dinner fork or by abrasion with a too firm toothbrush;and these can carry virulent organisms too, in addition to being the lacerating agents.

Then again many sailors are in a state of nervous tension in war time and this helps to undermine their vitality and makes them prone to infection.

To give a further instance of how those two factors of sub-scorbutic gums and caries or tartar operate I will describe my experience of shipboard. In a five months voyage on T.S.S. "Clan Ferguson" I observed conditions on board.
The white crew had all been on board, with the exception of the eighth engineer, for one year and were all seagoing men, always at sea. We were in the tropics most of the time and at each port bought cheaply the predominant local fruits e.g. papayas at Beira in Portugese East Africa, pineapples at Mauritius, mangoes and oranges at Dakar, etc. With the exception of this fruit the food was similar to the diet that the sailors on the small craft have to-day. The fruit and refrigerated lettuce was eaten by all on board and with one exception to be mentioned, there were no cases of Vincent's Disease, or of swollen, bleeding gums. The Company allowed 2s.6d. per head, per day for food and the Steward possibly made something on the transactions as is done in most lines. Be that as it may, the abundance of fruit prevented gum disease and the routine examination of the crew showed no evidence of Vincent's gum disease. The exceptional case mentioned above, was that of a Lascar sailor, (Kala Mia H....., 24 years) who contracted syphilis at Antwerp and who appeared at the sick bay with a primary sore. As the next port was 28 days away and as there was no arsenic on board, treatment was pushed with mercury pills. He developed a mercurial gingivitis and later the typical

white membrane of Vincent's Disease appeared. This was confirmed at a hospital in Durban. This Lascar ate fruit daily and had no sub-scurvy. His predisposing lesion was the mercurial gingivitis and the organisms were probably lurking in his mouth though caries was absent and tartar was slight in amount. (Gay (45) states that mercury poisoning can predispose to infection by Vincent's organisms.)

At Durban also I was called by a padre in a Seamen's Mission to see an Englishman who had been given a passage from Western Australia in one of the Hamburg-Amerika Line ships. He was to work his way back to Germany and hoped to get home to Britain from there. It was before the war. Unfortunately he was "green" to the sea life and the Germans would not let him sign any articles till he was 24 hours out. He then found that his wage was a mere pittance and that he lived in miserable quarters, was worked to death and fed literally on scraps. He had very pronounced scurvy, with purpura, tender, swollen muscles, and swollen, bleeding gums. His teeth were healthy and clean and he had no Vincent's disease for which I was on the look out. I mention this to show again that Vincent's Disease does not always develop in scurvy cases as it did in case 38 in my series, just as it does not always develop in pellagra cases (25).

In the winter of 1939-40 I saw for nine months at first hand the conditions on a destroyer and observed the class of men with which this thesis deals. There was overcrowding of necessity and the ever present tension and strain from the menace of mines, U-boats, aircraft and E-boats coupled with long patrols in rough weather. This is bound to tell on the health of the men. Health lectures were repeatedly given and the points stressed were bodily cleanliness, attention to bowels and teeth, and the importance of consulting the M.O. on the first suspicion of "crabs" or scabies or any other suspicious ailment. Then their conduct on shore as regards alcohol and their relationship with the opposite sex was guided and they were told of the importance of prophylaxis against V.D.. The diet was gone into thoroughly and all messes were told of scurvy and its dangers, and all supplemented their diets by eating oranges, tomatoes and other

fruit, raw from the canteen, whose manager was instructed to keep a good supply of fruit.

Whenever scabies or pediculosis pubis appeared, and that was frequently, every member of the mess was examined in the sick bay and when they were thus together an opportunity was taken to stress the importance of fruit in the diet.

Then there were periodic dental parades and those with caries were sent ashore for treatment. No man had foul, tartar-covered teeth. If he had he was reprimanded and the matter dealt with.

Only thus, by chasing up the men and by repeated lectures, can conditions be kept satisfactory. There were no cases of Vincent's disease of the gums on board.

Sailors in from a long patrol, will rush ashore on the first "liberty boat" and usually indulge in an orgy of wine, women and song when they should be resting. If they are not guided in such matters they quickly get into a low state of health and become more susceptible to any illness that is going the rounds. To-day the vast majority of young men in the Forces are called up from decent homes, are not hardened devil-may-care sailors and are only too willing to take advice in those matters.

In concluding those general remarks I will again say that, in my opinion, the vast majority of the cases of Vincent's Disease on the small ships, are due to the factors of traumatising caries or tartar and swollen, tender gums from lack of vitamin C, operating as above described.

Furthermore contagiousness has been demonstrated.

X. TREATMENT.

I will conclude with a few personal observations on the treatment.

It is not sufficient to merely apply local remedies to the gums. I have seen many cases which showed no improvement and even got worse with daily applications of 10% chromic acid, Eusol 1 in 4, 5% crystal violet, Dettol, or Beaumont's paint. (51.). It is true that some cases clear up with local application of medicaments alone, but cases sufficiently severe to warrant their admission to hospital will in nine cases out of ten require more vigorous treatment to get them back to duty quickly.

Then again it was soon evident that cases which attended daily as out-patients took about 3 weeks to cure while the average number of days for hospital cases was 11.07 per case. This is probably due to the fact that hospital cases receive their dental toilet regularly four times a day under supervision, while the out-patient, though he is given a bottle of mouth wash is not so liable to use it regularly; and again the out-patient goes back to his ship between treatments and may get re-infected daily from fomites etc.. Also it is no use to discharge a case before it is properly treated. Cases recur unless adequately treated (Harper (16)).

I have mentioned more vigorous treatment. By this I mean a thorough dental toilet with the removal of debris etc.. From personal experience I firmly believe it is essential if the fewest possible days in hospital per case are to be achieved. Smith (44) mentioned among other things the advisability of thorough dental treatment while Leigh (85) did not scale till all acute symptoms were away. I will now mention in detail the cleaning process used in all my cases as I believe that it is the greatest contributory factor to the cure. Furthermore this method of cleaning the teeth was done as soon as the patient was admitted to hospital, with no ill effects. If an alveolar abscess co-existed however, the teeth at fault were not pulled till the acute symptoms subsided although scaling of the rest of the teeth

was done . If a diseased tooth was pulled straight away it was soon found that the cavity was apt to become infected.

The first photograph shows the instruments used in the routine cleansing method adopted.

(a) A dental mirror.

(b) Scaling tool.

(c) Rotary brushes. (cup and flat shaped)

(d) Spray apparatus.

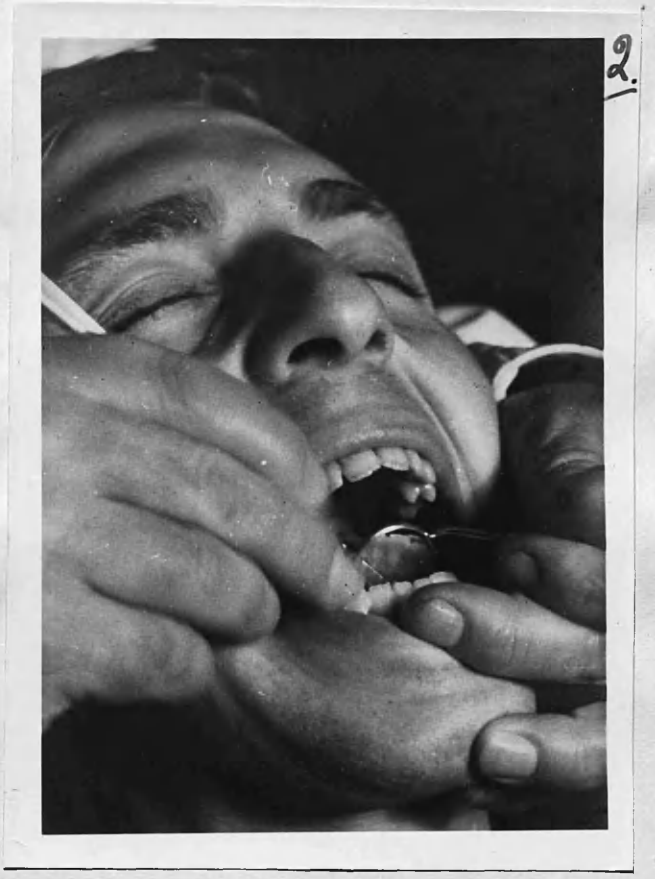
(e) Swab and 10% chromic acid.

Photograph No. 1 shows a typical case of Vincent's disease.

The swollen gums and prominent interdental papillae are evident. The white, filmy membrane on the gums can be made out glistening at places. Moreover the teeth are not as clean as they might be since they appear more white in the ensuing photographs after cleansing. At the upper right gum an ulcerated area at a tooth base is seen. The first thing is to inspect the mouth and teeth, carefully noting the extent of the disease.

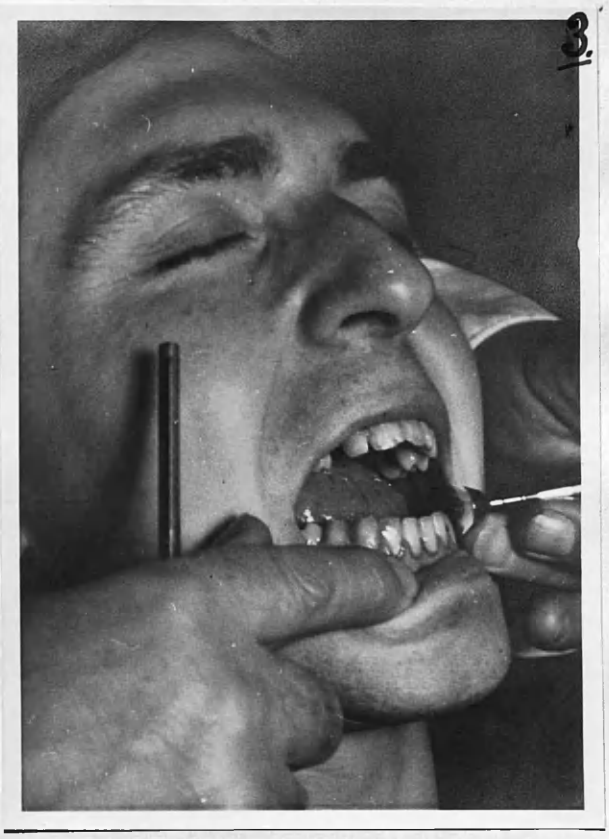
The next step is to scale the tartar from the individual teeth. In many cases it is a hard, crusted mass, forming a collar round the base of the tooth and overlapped by swollen gum. This must all be removed. If it is not eradicated at the very start the time of cure is lengthened, for each piece of foul tartar is teaming with organisms and keeps up the infection.

Photograph No. 2 shows the scaling process, using the dental mirror to see hidden foci of tartar behind the teeth.



Next the rotary brush is used, dipped in powdered pumice stone and glycerine. This polishes the teeth and removes the finer particles of tartar which the scaler misses. Either a flat or a cup-shaped brush is used depending on the region to

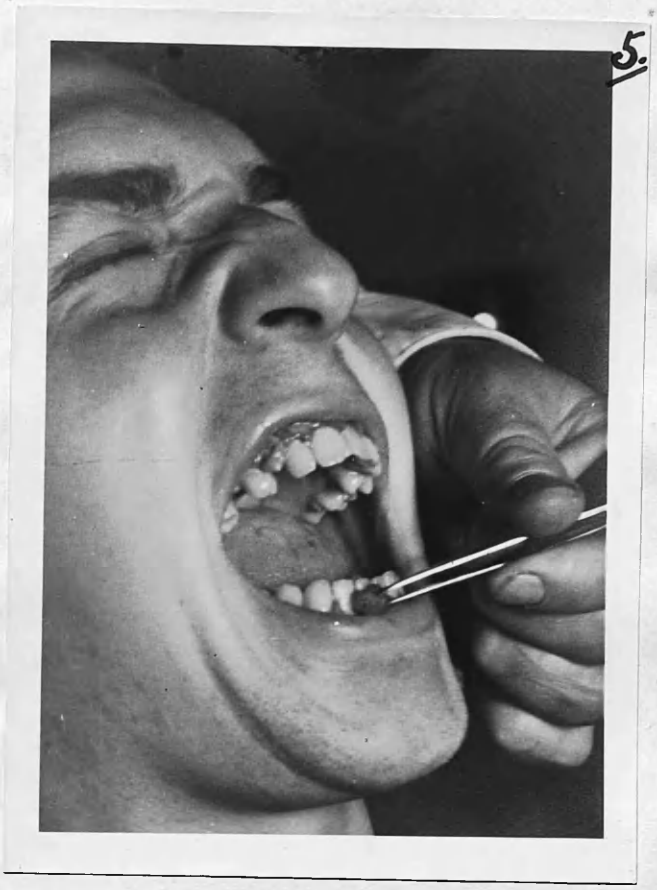
which access is desired. The gums often bleed at this stage but no harm ensues. (Photograph No. 3).



Using the spray the teeth, mouth, labio-gingival and retro-molar sulci are now well sprayed with 1 in 4 Eusol solution as depicted in photograph No. 4.

If there is still bleeding the patient can rinse his mouth with a warm effervescing mouth wash while still on the chair.

The final stage of the initial process is depicted on photograph No 5. below.



The gums get a daily swabbing with a pledget of cotton wool dipped in 10% chromic acid. Adverse criticism has been levelled at this use of chromic acid, on the grounds that it decalcifies the teeth. (Leigh (85) and others.)
This is so if it is used for over 2 weeks but the average number of days in my series was 11.07 and in this period there is certainly no evidence of dental damage from the use of chromic acid. Where the trouble starts, in my opinion, is when the practitioner gives the chromic acid without performing the above mentioned thorough dental toilet. Then the condition drags on for 2 or 3 weeks or more and the chromic acid starts to act on the teeth.

In addition to chromic acid daily there are mouth washes of Eusol (1 in 4) four times a day.

If there is a decayed tooth, it is sprayed with Eusol, mopped dry and "stopped" with a temporary, carbolised dressing.

This shuts off that source of infection while the treatment is carried out.

Then again it is adviseable to order the patient to destroy his old tooth brush, if he has one, when he returns to his ship. If he uses the old brush he is liable to re-infect himself as I have repeatedly seen with out-patients.

I noticed that if hydrogen-peroxide was used as a mouth wash 4 times a day, even when diluted, it tended in 5 or 6 days to make the gums raw and red.

It was better to use potassium permanganate solution or Eusol (1 in 4). The permanganate probably acts in virtue of its oxidising properties. The organisms are anaerobes. However, I favoured Eusol because it dissolves off the membrane in virtue of its chlorine content just as chlorine-containing solutions are used to dissolve fibrin in an empyema cavity. Indeed the typical, filmy, white membrane seems to melt away when the Eusol spray is directed against it.

There is no doubt that simple application of local agents is not sufficient to cure those cases that were admitted to hospital and I soon observed that if the cure had to be effected in under 14 days, early and meticulous dental toilet, as above described, was necessary.

On discovering that sub-scurvy was the predisposing lesion I naturally thought that ascorbic acid should be tried.

Ascorbic acid, even when pushed to the extent of 700 mg. 3 times a day, will not cure alone, in the absence of local treatment. Once the surface of the gum is invaded the tartar or source of infection must be removed.

Cases 10, 18, 24, 33, 41 and 47 were all treated with ascorbic acid before admission and were not deficient in it. Case No.10 had ascorbic acid for eleven weeks with no beneficial results. The results with many out-patients were the same.

It is tempting to think that if a case is first saturated with vitamin C it will clear up quicker than another case not so saturated, when local treatment is once started.

I did not find any acceleration of cure in a case previously saturated with vitamin C. However I noticed that the gums began to lose their fiery red look a day or two sooner than they would have done without previous vitamin C saturation. The actual ulceration took the same time to heal as a case which had no extra vitamin.

I have also noticed, chiefly in large numbers of out-patients, that Beaumont's arsenic solution is not so efficacious as N.A.B. given intravenously (See my previous reasoning: Page 48). Indeed a tonsillar case, with no gum lesions, will usually be clear by the seventh day with 0.45 of a gram of N.A.B. given intravenously, and nothing else. Occasionally haemolytic streptococci were superimposed on a tonsillar Vincent's infection and there M. & B. 693 seemed to get rid of the coccal part of the infection. At any rate the uvular and faucial redness usually went, but M.& B. 693 alone had no effect on the Vincent's Disease proper.

A glance at table I will show the various ways in which the cases were treated in addition to the thorough dental toilet which all had.

TABLE I No. of Cases treated.	GENERAL LINES OF TREATMENT	TOTAL DAYS ILL.	AVERAGE DAYS TO CURE.
12.	N.A.B. + saturn. vit. C + local treatment.	127	10.6
21.	N.A.B.+ local treatment	222	10.6
5.	Local treatment only	57	11.4
8.	N.A.B.+ saturn. nicotinic acid.	90	11.3
6.	Local treatment+ saturn. vitamin C.	67	11.2
2.	Local treatment + saturn. nicotinic acid.	34	17
1.	Saturn. vitamin C + saturn. nicotinic acid.	12	12
55		609	11.07

The 55 cases represented 609 days off duty in all, and averaged 11.07 days per case, off duty. If we consider that the

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The 55 cases represented 609 days off duty in all, and averaged 11.07 days per case, off duty. If we consider that the

average out-patient spends about 3o minutes waiting to see a busy M.O. and is attending daily for about 21 days, and that in 9 cases out of 10 he spends an entire forenoon or afternoon getting from his ship to the dentist and back again, it will be seen that it pays to take those cases into hospital for treatment, in the long run. Most of the small ships of the trawler class have no M.O. or dentist on board.

If we glance at the "average days to cure" column we see that there is really nothing outstanding in the various methods of treatment when one is compared with another. All methods have as a basis the above described, thorough dental toilet and this seems to be the key to the best treatment.

The two cases which took an average of 17 days to cure, comprised a case which was 21 days in hospital and one which was 13 days in hospital. The former had no local treatment till the eleventh day, but nicotinic acid only. The disease actually cleared in ten days after dental toilet etc was begun. If this fact had been considered it would have lowered the figure of 17 to 11.5 days which is in the same category as the other figures in the end column of table I.

As regards saturating with nicotinic acid, I did not get the results claimed by King (83), but my cases did not as a class lack nicotinic acid. There is no point in giving it as a routine measure to the sailor class with which I deal and its expense prohibits this practice in addition.

If we compare the figure of 11.07 days per case off duty, with the figures of earlier workers (David and Hecquet (90) - 12 to 20 days, Pickles (29) - 3 weeks to 2 months, Taylor and McKinstry (19) - 17 days, etc.) we see that the above mentioned routine treatment is sound.

Let me conclude this chapter by stressing that on shipboard the following factors seem to be necessary to prevent the disease:- 1. a varied diet, or the routine diet plus fruits or lightly cooked, fresh vegetables, to prevent a lowering of the vitamin C level of the body with its attendant swelling of the

gums. (c.v. My experiences on T.S.S. "Clan Ferguson" and on a destroyer). 2. Periodical dental parades and repeated health lectures. 3. Proper washing and sufficiency of eating utensils. 4. Segregation and treatment of cases if they arise, with gargling and teeth brushing under supervision, of those remaining on board in the presence of an epidemic.

There is great scope for preventive medicine on shipboard, especially where the menace of Vincent's Disease is constantly present.

XI. SUMMARY OF THESIS.

1. The introduction states my intention to try to make some contribution, however slight, to our knowledge of Vincent's Disease as it affects our sailors on the small ships. Those sailors by virtue of their mode of living are in a class by themselves. It is stressed how opinions regarding the causation of the disease differ widely.

2. Acknowledgements are made to persons who have kindly assisted me in various ways.

3. A full historical outline is given showing how views on the subject and methods of treatment have altered. It is shown how deficiency of different vitamins is the predisposing factor in different communities.

4. The general lines of the investigation are explained and relevant statistics from the hospital are given. It is pointed out that there is a rise in Vincent's Disease with each succeeding quarter and that it is common in small ships operating in the North Sea. The first cases appeared when the war had been

in progress for four months, and two to four months is the time taken for the bodily store of vitamin C to be used up. Also the first case of scurvy appeared in the same quarter of the year as the first case of Vincent's Disease.

The best method of staining the organisms for photography, with simple stains, is described in this section.

5. A histological study of the gums was made and it supported the view that it was a condition of subscurvy that was originally present in the sailor class. Photographs are shown.

6. A full desciption of the urine test for the estimation of vitamin C is given, with a short history of vitamin C and of the test.

Some original tests were carried out with urine in dark and other bottles to show the rate of deterioration of vitamin C.

A controlled test was done, to show how a random sample of urine might appear low in vitamin C but how a test dose will raise the value of vitamin C in the 6 hours specimen if the body is not really deficient.

7. The method used to determine the level of B_2 in the body is fully described and a short history of vitamin B is given.

8. Findings from 55 cases of Vincent's Disease are discussed.
There was no clinical evidence of vitamin B deficiency.
It was considered that the study of the diets showed no evidence of lack of nicotinic acid.
The urine chemical tests showed no deficiency of nicotinic acid in 82.6% of the cases done.
It was described how there was no clinical or dietary evidence of shortage of vitamins A or D.
It is shown how a deficiency of vitamin C is present in many small ships'companies. Reasons for concluding this are given and a discussion is made on the subject on the lines that, (a) 9 true cases of scurvy, from those small ships,were admitted to the hospital between January and December 1940, (b) the dietary study and the manner of the cooking on board shows a low level of vitamin C or a destruction of vitamin C in the food, (c) cases of gingivitis, with swollen bleeding gums, were seen, with no

Vincent's infection, and which responded to ascorbic acid alone, (d) urinary excretion tests showed the almost universal presence of vitamin C deficiency in the diets (reasons are given why the low level of vitamin C is not due merely to the infection) and (e) the Naval limejuice and its ascorbic acid content is commented on.

Blood counts of the cases were done to try to discover the cause or the effects of the disease there.

The incidence of caries or tartar in the teeth of the patients, and of smoking and drinking was considered.

9. The probable mode of onset of the disease is discussed as it affects the small ships' companies, and the mechanism by which the gums are infected.

My observations on random cases, not of Vincent's Disease, show that 63% of sailors have the fuso-spirillary organism in their mouths.

Examples showing that contagiousness exists are given and a successful method of stamping out an epidemic on a small ship is described.

The role that vitamin C deficiency plays in starting the infection is given.

My experience with the disease on shipboard, when methods are taken from the outset to prevent it, is described.

10. Some observations on treatment are appended. Early, routine scaling, brushing and spraying of the teeth is in my opinion a necessity if the fewest possible days in hospital are to be obtained. The use of large doses of vitamins does not quicken the cure though with previous saturation with vitamin C, the gums lose their fiery red appearance sooner than they would do otherwise.

Photographs of the stages of treatment are appended.

11. A bibliography is added.

12. A brief description of each case is given.

I have discussed the problem of Vincent's Disease as I found it in the small ships, and I hope I have contributed, however slightly, to our knowledge of the condition there.

BIBLIOGRAPHY.

1. W.D. Miller "Microorganisms of the Human Mouth" 1890 (Quoted Leigh. 1940)

2. Rauchfuss. Petrograd 1893 (Quoted W. Wingrave 1915.).

3. & 4. Plaut H.C. Deutsche Med. Wchnschr. 1894. 20:920 (Quoted Der Grosse Brockhaus and Varney. 1927)

5. Vincent. Ann. de l'Inst. Past. 12:488. 1896 (Quoted Varney. 1927).

6. Bernheim. Centralbl. f. Bakteriol. 23. 177. 1897 (Quoted Varney. 1927)

7. Vincent. La Semaine Med. 18. 109. 1898.

8. Veillon and Zuber. Archiv. de Med. Exp. pp 10. 517. 1898.

9. Moure. J.Laryngol, Rhinol and Otol. 1898.

10. Vincent H. Bulletins De La Société Medicale des Hopitaux de Paris. xv. 1898.

11. D. Veszpremi. Centralbl. f. Bakt. 38. 136. 1905 (Quoted Rosebury and Foley 1939).

12. Vincent 1905 Lancet.

13. Wyatt Wingrave. Trans. Roy. Soc. Med. 1908.

14. J.D. Rolleston, Brit. Journ. Children's Diseases. lX p.311 July 1912.

15. Wyatt Wingrave. J. Laryngology. Lond. 472-481. 1915.

16. Harper J. Glasgow M.J. 1915 L xxxiv.34-40

17. Bergeron, J.Z. Chicago M.J. 1915 xxvii.145-148.

18. McConnell. N.Y. Med. Journ. 1916. 104; 300. (Quoted Varney 1927)

19. Taylor and McKinstry. Proc Roy. Soc. Med. Vol x.Lond. 1916-17. Sect. Laryng. 43-48.

20. McKinstry 1917. Practitioner 99. 368.

21. D. McKenzie. Med. Press. and Circ. Lond. 1917 ciii. 409.

22. Bouty. B.M.J. Lond. 1917. ii. 685.

23. F.E. Taylor and W.H. McKinstry. B.M.J. Lond. 1918. 1; 101.

24. W.H. McKinstry. J.RAMC. March 1918. pp. 1-22.

25. Journal of R.A.M.C. 33. p. 426. 1918.

26. Eagleton, Mercer and Hudson. B.M.J. Lond. 1918. 1; 15.

27. J.F. Colyer. B.M.J. Lond. 1918. 1; 205.

28. Semple, Price Jones and Digby. J.R.A.M.C. Lon. 1919. 30;217-281.

29. W.N. Pickles. J.R. Nav. Med.Serv. Lond. 1919. V; 87-91.

30. G.E. Hamilton. J.R. Nav. Med. Serv. Lon. 1919. V.

31. F.C.B. Gittings. J.R. Nav. Med. Serv. Lond. 1919. V. 287-290.

32. Hess A.F. Philadelphia. J.B. Lippincott Co. 1920 (Quoted McLester 1939)

33. C.H. Morris U. States Nav. Med. Bull. Wash. 1921.iv. 540-542

34. J.B. Goodall. U. States. Nav. Med. Bull. Wash. 1921. iv. 542-544

35. Apostolides A.G. J. Trop. Med. 25:81. April 1. 1922 (Quoted Rosebury and Foley, 1939)

36. Keilty. Jour. Med. Res. 43:377. 1922.

37. Tunnicliff.Journ. Inf. Dis. 8:316. 1923.

38. Pilot and Davis. Arch. Int. Med. 1924. 34:313.

39. Nichols, H.J. 1924 (Quoted Leigh 1940).

40. Chevalier Jackson. Journ. Amer. Med. Assoc. Lxxxiii. Dec.6.1924.

41. E.W. Goodall, J.Laryngol. xl. 1925. No.2.p.5.

42. Varney, P. J.Bacteriol. 1927. 13:275.

43. D.T. Smith Baltimore. Williams and Wilkins. 1932 (Quoted Rosebury and Foley 1939).

44. D. Smith, Arch. Otolaryng. 18. 760-769. Dec. 1933 (Quoted H. Field, 1940).

45. Gay. Agents of Disease and Host Resistance. Illinois. 1935. (Quoted Brezeale and Greene, 1940).

46. Spies. J.A.M.A. 104. 1377-1380. April 20. 1935.

47. Dalldorf and Russell. J.A.M.A. 104; 1701.1935.

48. Coward and Morgan. B.M.J. 1935. 2; 1043.

49. Clements, F.W. M.J. Australia. 2:615. Nov. 7. 1936.

50. May Mellanby. M.Woman's J. 43. (36-39). Feb.1936 (Quoted Sinclair, 1939).

51. Beaumont. Text Book of Medicine (J. and A. Churchill) 1937.

52. Leigh and Sure. Arch. Path. 24. 434:442. Oct. 1937.

53. Jeghers. J.Amer. Med. Assoc. 1937. clx:756.

54. Harris and Abbasy. Lancet 1937. 2:1429.

55. St. Clair, Thomson and Negus. Diseases of Nose and Throat. (Cassell 1937)

56. K.O's. Elsom. M. Clin. N.A.21. 1230-1236. July 1937 (Quoted Sinclair, 1939)

57. Smith, Persons and Harvey. J. Nutrition 14. 373-381. Oct.10 1937. (Quoted Sinclair 1939).

58. Stitt, Clough and Clough. Practical Bacteriology, Haematology and Animal Parasitology 1938.

59. Ungley. Lancet 1938 1; 875.

60. Leslie Harris. Vitamins and Vitamin Deficiency. vol.1. 1938.

61. Spies, Grant, Stone and McLester. South M.J. 31;1231. 1938 (Quoted McLester 1939).

62. T.W. Ross North West Med. 37; 49. Feb. 1938. (Quoted H. Field, 1940).

63. T. Rosebury and G. Foley. J.Am. Dent. A. 26; 1798-1811. Nov. 1939.

64. R. Tunnicliff. J. Am. Dent. A. 26; 1987-1991. Dec. 1939.

65. J.A. Sinclair. J. Am. Dent. A. 26; 1611-1618. Oct. 1939.

66. Kirkpatrick. Dent. J. Aust. 2; 1; 78. 1939.

67. Harris and Raymond. Biochem. Jour. vol. 33. part 2. 1939.2037.

68. Beaumont and Dodds. Recent Advances in Medicine 9th Edn. 1939.

69. H. Letheby Tidy. Synopsis of Medicine. 1939.

70. McLester. Nutrition and Diet in Health and Disease, 3rd Edn. 1939.

71. Fox, Dangerfield, Gottlich and Joke, B.M.J. 143-147 Aug 3 1940

72. Cecil, Text Book of Medicine (Saunders) 5th Edn.1940.

73. D.G. Stine. J.Missouri M.A. 37; 277-280. July 1940.

74. E.L. Breazeale and R.A. Greene. South Western Med. 24 (20-21) Jan. 1940.)

75. J.L. Bennett. The Psychiatric Quarterly 14; 632-642 July 1940.

76. Bell, Lazarus and Munro, Lancet, Aug. 10th. p. 155. 1940.

77. J. Pemberton. Lancet. Aug. 17th. 1940. p. 217.

78. Dr. L.J. Harris. Lancet. Aug. 31. 1940. p. 259.

79. Spies, Hightower and Hubbard. J.Amer.Med.Assoc. 1940. 115. 292.

80. King. 1940. Spec. Rep. Ser. Med. Res. Counc. Lond. No.241.

81. E.P. Cathcart, Murray and Beveridge. 1940. Spec. Rp. Ser. Med. Res. Counc. London. No. 242.

82. F.S. Roff and A.J. Glazebrook 1940. Brit. D.J. 68; 135.

83. J.D. King. Lancet. July 13th 1940.

84. H. Field (Junr.) The Pharmacopeia and the Physician, J.A.M.A. 114; 1073-1077. March 23. 1940.

85. R.W. Leigh. The Military Surgeon. 86. Jan 1940.

86. Roche Products Limited. London. N. 13. Pamphlet No.L15607(825)

87. Larousse. Du. XXe Siecle. Tome. Sixieme.

88. Meyer's Lexicon. vol. 9.

89. Der. Grosse Brockhaus. Band. 14.

90. M. David and D. Hecquet. Med. Press. and Circ. Lond. 1921. Cxi; 496.

91. Hennessy and Fletcher. Lancet. Lond. 1920. 2;127.

92. C.J. Fuller. Lancet. Jan. 18th. 1941.

93. H. Stuart Barber. Lancet. Jan 18th. 1941.

94. A. Lemierre. Rev. Gén. Clin. 53. 385. 1939. (Quoted Stuart Barber).

95. Wolmar Nordenmark. Skandinav. Arch. Physiol. 70:186.1934. (Quoted Field).

96. Weisberger, Young and Morse. J. Dent. Research. 17; 101-105. April 1938 (Quoted Field).

97. J.L. Dudley Buxton. Dental Pathology (E. and S. Livingstone) 1937.

98. H. Gordon Campbell. B.M.J. March 8th. 1941. 360-361.

99. Vitamins Limited. 23 Upper Mall, London. Nutrition Information Bulletin. C5. March 1941.

CASE NUMBER.	SITE OF DISEASE.	CLINICAL SUB-PELLAGRA.	CLINICAL A. DEFICIENCY	NICOTINIC ACID DEFICIENCY.
1.	Gums	0	0	Absent
2.	Gums	0	0	Absent
3.	Gums and tonsils	0	0	Absent
4.	Gums	0	0	Deficient
5.	Gums	0	0	Absent
6.	Gums and tonsils	0	0	Absent
7.	Gums and tonsils	0	0	Absent
8.	Gums	0	0	Absent
9.	Gums and tonsils	0	0	Absent
10.	Gums	0	0	Absent
11.	Gums	0	0	Absent
12.	Tonsils and gums	0	0	Absent
13.	Gums	0	0	Absent
14.	Gums	0	0	Absent
15.	Gums	0	0	Absent
16.	Gums and tonsils	0	0	Not done
17.	Gums	0	0	Not done
18.	Gums	0	0	Absent
19.	Gums	0	0	Absent
20.	Gums	0	0	Not done
21.	Gums	0	0	Absent
22.	Gums	0	0	Not done
23.	Gums	0	0	Absent
24.	Gums	0	0	Absent
25.	Tonsils and gums	0	0	Not done
26.	Tonsils	0	0	Not done
27.	Gums and tonsils	0	0	Not done
28.	Gums	0	0	Not done
29.	Gums	0	0	Not done
30.	Gums	0	0	Not done

continued.

31.	Gums	0	0	Not done
32.	Tonsils	0	0	Not done
33.	Gums	0	0	Not done
34.	Tonsils	0	0	Not done
35.	Gums	0	0	Not done
36.	Tonsils and gums	0	0	Not done
37.	Gums	0	0	Not done
38.	Gums	0	0	Not done
39.	Gums	0	0	Not done
40.	Tonsils and gums	0	0	Absent
41.	Gums	0	0	Not done
42.	Tonsils	0	0	Not done
43.	Gums	0	0	Not done
44.	Tonsils and gums.	0	0	Not done
45.	Gums	0	0	Not done
46.	Tonsils and gums	0	0	Not deficient
47.	Gums	0	0	Not done
48.	Gums	0	0	Not done
49.	Gums	0	0	Not done
50.	Tonsils and gums	0	0	Not done
51.	Gums	0	0	Absent
52.	Hard palate	0	0	Not done
53.	Gums	0	0	Not done
54.	Tonsils and gums	0	0	Not done
55.	Gums	0	0	Not done

CASE NUMBER	VITAMIN C DEFICIENCY	TOURNIQUET TEST	CLINICAL B_1 DEFICIENCY
1.	Present	Negative	0
2.	Present	Negative	0
3.	Present	Negative	0
4.	Present	Negative	0
5.	Present	Negative	0
6.	Present	Negative	0
7.	Present	Negative	0
8.	Present	Negative	0
9.	Present	Negative	0
10.	Absent when seen	Negative	0
11.	Present	Negative	0
12.	Present	Negative	0
13.	Deficient	Negative	0
14.	Present	Positive	0
15.	Present	Positive	0
16.	Present	Positive	0
17.	Present	Negative	0
18.	None on admission	Negative	0
19.	Present	Negative	0
20.	Present	Negative	0
21.	Present	Negative	0
22.	Present	Negative	0
23.	Present	Negative	0
24.	Not on admission	Negative	0
25.	Present	Positive	0
26.	Absent	Negative	0
27.	Present	Negative	0
28.	Present	Negative	0
29.	Present	Negative	0
30.	Present	Positive	0
31.	Present	Negative	0

continued.

32.	Present	Negative	0
33.	Not on admission	Negative	0
34.	Absent	Negative	0
35.	Present	Negative	0
36.	Present	Negative	0
37.	Present	Negative	0
38.	Scurvy	Positive	0
39.	Present	Negative	0
40.	Present	Negative	0
41.	Not on admission	Negative	0
42.	Present	Negative	0
43.	Present	Negative	0
44.	Present	Negative	0
45.	Absent	Negative	0
46.	Slight deficiency	Negative	0
47.	Not on admission	Negative	0
48.	Present	Negative	0
49.	Present	Negative	0
50.	Present	Negative	0
51.	Present	Negative	0
52.	Present	Negative	0
53.	Present	Positive	0
54.	Present	Negative	0
55.	Present	Negative	0

No. of Case.	R.B.C./Cmm.	H.B. %	C.I.	Total Leucocytes per c. mm.	Polymorphs.	Lymphocytes	Mononuclears.	Eosinophils.	Basophils.
1.	4,820,000	84	o.87	11,000	77	20	3	0	0
2.	5, 100,000	92	0.9	8,500	68	23	5	3	1
3.	3,950,000	86	1.1	9,500	78	20	2	0	0
4.	4,200,000	80	0.91	13,000	67	28	4	1	0
5.	4,750,000	90	0.96	6,500	72	20	5	2	1
6.	3,650,000	64	0.89	12,000	75	20	5	0	0
7.	4,950,000	85	0.86	13,750	62	34	2	2	0
8.	3,280,000	70	1.09	6,900	80	15	3	1	1
9.	5,100,000	94	0.92	10,500	77	21	2	0	0
10.	3,840,000	78	1.01	9,800	57	34	7	1	1
11.	4,300,000	80	0.93	13,600	82	15	3	0	0
12.	4,900,000	94	0.96	10,800	70	22	6	2	0
13.	4,250,000	86	1.02	11,000	70	24	5	0	1
14.	4,000,000	78	0.975	7,800	70	27	3	0	0
15.	4,860,000	98	1.02	9,800	65	28	6	1	0
16.	4,560,000	103	1.14	15,000	68	25	7	0	0
17.	5,200,000	96	0.92	15,500	82	18	0	0	0
18.	4,770,000	92	0.99	11,600	65	29	4	2	0
19.	4,360,000	95	1.1	9,800	78	20	2	0	0
20.	4,200,000	86	1.02	9,500	72	24	4	0	0
21.	3,560,000	64	0.91	5,400	55	38	5	2	0
22.	4,900,000	86	0.88	16,000	80	17	3	0	0
23.	-	-	-	-	-	-	-	-	-
24.	4,280,000	79	0.94	11,000	64	29	5	1½	½
25.	4,480,000	90	1.02	12,600	75	19	6	0	0
26.	5,150,000	97	0.95	8,800	71	25	4	0	0
27.	4,500,000	99	1.1	6,800	75	22	2	1	0
28.	4,700,000	96	1.02	11,000	73	25	2	0	0
29.	4,800,000	100	1.04	10,200	59	34	6	0	1
30.	3,860,000	68	0.89	13,400	78	19	3	0	0
31.	4,920,000	94	0.95	10,200	80	15	4	1	0
32.	4,300,000	95	1.1	9,600	66	32	2	0	0
33.	4,860,000	98	1.02	11,400	75	20	4½	½	0

continued.

34.	4,700,000	92	0.98	5,000	40	53	6	1	0
35.	4,930,000	98	1.0	6,800	71	25	4	0	0
36.	4,370,000	92	1.06	7,100	80	15	3	1	0
37.	4,750,000	95	1.01	11,800	77	20	3	0	0
38.	4,500,000	70	0.78	9,400	69	27	3½	½	0
39.	4,150,000	87	1.06	7,200	80	15	4	½	½
40.	4,360,000	96	1.1	8,400	61	31	5	2	1
41.	4,850,000	98	1.02	10,800	66	29	5	0	0
42.	5,000,000	95	0.95	9,600	74	20	5	1	0
43.	-	-	-	-	-	-	-	-	-
44.	3,740,000	98	1.32	3,200	86	11	3	0	0
45.	4,390,000	88	1.02	15,300	72	24	4	0	0
46.	4,960,000	100	1.02	10,300	71	22	5	1½	½
47.	4,450,000	96	1.09	8,400	66	29	4	1	0
48.	-	-	-	-	-	-	-	-	-
49.	3,870,000	70	0.92	11,800	60	32	6	1	1
50.	4,680,000	98	1.06	10,000	72	25	3	0	0
51.	4,920,000	99	1.01	9,600	78	19	3	0	0
52.	4,100,000	88	1.07	13,600	80	15	4½	½	0
53.	3,860,000	72	0.95	12,500	76	20	4	0	0
54.	4,980,000	98	1.0	9,800	59	34	5	1	1
55.	5,250,000	98	0.94	8,000	66	26	6	1½	½

Number of case.	TYPE OF SHIP AND DURATION OF SERVICE.	DRINKER	SMOKER	TOOTH CONDITION
1.	Minesweeper. 9 months	Beer	Yes	Caries+tartar
2.	Yacht. 13 months	No.	Yes.	Caries.No tartar
3.	Paddle mine-sweeper. (X) 6 mths.	No.	Yes	No caries, tartar
4.	Paddle sweeper (Y) 5 months	Beer ++	Yes	Caries + tartar
5.	Paddle sweeper (Y) 3 months	No	Yes ++	Caries, no tartar
6.	Minesweeper.9mths	Yes	Yes	No caries, tartar
7.	Paddle sweeper (Y) 5 months	Yes	Mode-rate.	Caries + tartar
8.	Drifter 4 mths	No	Mode-rate	Caries, no tartar
9.	Minesweeper. 4 mths	Heavy	No	Slight caries, tartar
10.	Trawler, 1 year.	Yes	Yes	No caries, tartar
11.	Patrol trawler, 4 months	Yes	Yes	Caries + crevices
12.	Paddle sweeper (X) 5 months	No	No	Tartar, no caries
13.	Paddle sweeper (X) 8 months	Heavy	Mode-rate	Caries + tartar
14.	Trawler (Z) 10 mths	Heavy	Mode-rate	Caries, no tartar
15.	Trawler (Z) 11 mths	No	No	Caries, no tartar
16.	Destroyer, 10 mths	Yes	No	Severe caries, tartar
17.	Destroyer (A) 6 mths	Yes	Yes	No caries, tartar
18.	Trawler (A) 8 mths	Yes	No	Caries + tartar
19.	Shore hutments 14 mths	No	Yes	Loose tooth. No tartar
20.	Trawler-sweeper 8 mths	Yes	Yes	Tartar ++ Caries.
21.	Trawler 3 months	Yes	No	Caries + tartar
22.	Trawler (B) 10 mths	Yes	Yes	Tartar
23.	Drifter 9 months	No	Yes	Caries + tartar
24.	Paddle sweeper (Z) 9½ months	No	No	Tartar
25.	French destroyer 4 months	Yes	Yes	Tartar
26.	Destroyer (A) 17 months	Yes	No	No caries No tartar

continued.

27.	Destroyer 4½ mths	Yes	Yes	Tartar + close set teeth
28.	Trawler 8 mths	No	Yes	Caries + tartar
29.	Drifter 6 months	Yes	Yes	Caries + tartar
30.	Drifter 1 year	No	Yes	Tartar
31.	Sloop 4½ months	Yes	No	Caries + tartar
32.	Trawler (B) 8 mths	No	Yes	Caries. no tartar
33.	Drifter 10 months	Yes	Yes	Slight tartar
34.	Shore establishment 5 months	Yes	No	Caries only
35.	Sloop for 4 months	Yes	Yes	Tartar + caries
36.	Minsesweeper (S) 4 months	No	No	Caries + tartar
37.	Trawler 18 months	Yes	Yes	Caries + tartar
38.	Trawler 1 year	Yes	No	Tartar
39.	Trawler (A) 7 mths	Yes	No	Caries + tartar
40.	Battleship 2 weeks Camp 10 weeks	No	Yes	Caries + tartar
41.	Motorboat 1 year	Yes	Yes	Caries + tartar
42.	Paddle sweeper 1 yr.	Yes	Yes	Tartar only
43.	Trawler (A) 15 months	Yes	No	Staining only
44.	Destroyer 8 months	Yes	No	Tartar + caries
45.	Motorboats 1 year	Yes	Yes	Tartar + +
46.	Trawler 5½ months	No	Yes	Tartar + +
47.	Paddle sweeper (Z) 6 months	Yes	Yes	Tartar + caries
48.	Paddle sweeper (Z) 4 months	Yes	No	Caries + tartar
49.	Trawler 18 months	Yes	Yes	Tartar + +
50.	Drifter 5½ months	Yes	Yes	Caries
51.	Destroyer 3½ mths	Yes	Yes	Alveolar abscess + tartar
52.	Destroyer 5 months	No	No	Tartar + caries
53.	Drifter 8½ months	Yes	Yes	Tartar + caries
54.	Tanker 4 months	Yes	Yes	Caries + staining
55.	Destroyer (A) 7 months	No	Yes	Caries + tartar

Number of Case.	SPECIAL FEATURES N.A.B. INJECTIONS, ETC.	COMPLICATION	NUMBER OF DAYS IN HOSPITAL.
1.	N.A.B. 5th day	Adenitis and bronchitis	11.
2.	N.A.B. and Saturn. Vit.C	Adenitis	12.
3.	N.A.B. on 1st day	Adenitis	10.
4.	N.A.B. and Saturn. vit.C	Epistaxis Adenitis	9.
5.	Saturation vit. C	Adenitis	10.
6.	N.A.B. and Saturn. vit.C	Alveolar abscess Adenitis	11.
7.	Saturation vit. C	Alveolar abscess Adenitis	9.
8.	N.A.B.+Saturn. vit.C	Gastritis Adenitis	13.
9.	N.A.B. and Saturn. vit C	Adenitis	7.
10.	Vit. C + Local N.A.B.	Adenitis	15.
11.	N.A.B.+ Saturn vit. C	Bronchitis	16.
12.	N.A.B. + Saturn vit. C	Adenitis	10.
13.	Saturation Vit. C	Bronchitis Adenitis	9.
14.	N.A.B.	None	8.
15.	N.A.B. + Saturn vit. C	Shotty adenitis	10.
16.	N.A.B.	Adenitis	9.
17.	-	Perforation	10 days with Vincent's D.
18.	N.A.B. saturated on admission with C.	Adenitis	10.
19.	Saturated with nicotinic acid	Bronchitis	21.
20.	N.A.B.	Adenitis	12.
21.	N.A.B.	Adenitis Bronchitis	10.
22.	N.A.B.	Adenitis Tonsillitis	10.
23.	N.A.B.	Bronchitis	10.
24.	N.A.B. + Previous Saturation C	Shotty adenitis	10.
25.	N.A.B.	Adenitis	13.
26.	N.A.B.	Shotty adenitis	8.
27.	N.A.B.	Adenitis Gastritis	12.

continued.

28.	N.A.B.	-	5.
29.	-	Bronchitis Adenitis	13.
30.	-	Adenitis	14.
31.	Ascorbic acid	Gastritis Bronchitis Adenitis	12.
32.	M.&B.693 + N.A.B.	Adenitis Bronchitis	13.
33.	Vit. C. First + N.A.B.	Adenitis	10.
34.	M.&B. 693 + N.A.B.	Bronchitis Adenitis	13.
35.	N.A.B.	"Indigestion" Adenitis	11.
36.	N.A.B.	Nyctalopia Adenitis	11.
37.	-	Adenitis	12.
38.	Saturation vit. C.	Anaemia Nyctalopia	12.
39.	N.A.B.	Adenitis	10.
40.	N.A.B. (very ill)	Conjunctivitis Bronchitis Adenitis.	18.
41.	N.A.B. + Saturn. vit. C	Adenitis	9.
42.	N.A.B.	Post nasal discharge Adenitis	7.
43.	N.A.B.	Gastritis	8.
44.	N.A.B.	Adenitis	13.
45.	-	Adenitis	8.
46.	N.A.B. + Saturn. nicotinic acid.	Adenitis	10.
47.	Saturation Vit. C. + Saturation nicotinic acid	Adenitis	12.
48.	Saturation nicotinic acid + N.A.B.	Bronchitis Adenitis	15.
49.	N.A.B. + Saturn. nicotinic acid.	Adenitis	10.
50.	N.A.B. + Saturn. nicotinic acid.	Adenitis	11.
51.	N.A.B. + Saturn. nicotinic acid.	Bronchitis Adenitis	12.
52.	N.A.B. + Saturn. nicotinic acid.	-	8.
53.	N.A.B. + Saturn. Nicotinic acid.	Adenitis Anaemia	12.

continued.

54.	N.A.B. plus saturn. nicotinic acid.	Adenitis	12.
55.	Nicotinic acid saturation first.	Adenitis Bronchitis	13.

SUMMARY OF THE CASES OF VINCENT'S DISEASE.

As previously stated, the great majority of those cases was confined to bed. All cases had a routine scaling of the teeth, brushing with the rotary brush, and spraying with Eusol solution at the outset.

The special methods of investigation have been described in full previously. The tourniquet test was done, by the cuff of a sphygmomanometer being placed round the bare arm and the pressure of mercury being raised for 3 minutes to a hundred mms. The cuff was then removed and ruptured capillaries and petechiae sought for and counted.

The results are summarised in the preceding tables and have been previously discussed. The complications are noted in the tables.

All the cases had a "positive swab" as described earlier and as illustrated by photograph.

Special attention from the outset was paid to the points illustrated by the columns headings in the tables of summary. This was done in an attempt to find some factor common to, or predisposing to the condition in the sailors of our small ships.

The cases are described in brief to conserve paper.

Case 1. P.B. Ordinary Seaman, 55 years.

On a minesweeper for 9 months.

On admission:- Temp. 98.4°. Pulse 100 per min. Bowels open. Tourniquet test - negative. Urine - pale straw colour, S.G. 1028, acid; blood,sugar, bile and albumen - nil. Gum and throat swabs "positive".

Blood count. R.B.C. - 4,820,000 per c. mm. H.B. - 84%. C.I.-0.87. W.B.C. - 11,000/c.mm. Polymorphs 77%, lymphocytes 20%, eosinophils 0%, monocytes 3%, Basophils 0%

Urinary vitamin C estimation. First specimen - 1.1 mg.%, 3 hours spec. - 3.3 mg. %, 6 hours spec. - 1.8 mg.% (therefore vitamin C. deficiency).

Nicotinic acid excreted per day equal to 6.31 mgs. in 42 ozs. urine.

Complaint and history:- This rating has lost 6 lbs of weight in 2 months, has a very bad mouth and complains of toothache and sore, easily bleeding gums. Also has hoarseness, cough and slight pain on breathing. Bad taste in mouth on rising. No diarrhoea and generalised pains are absent. Does not suffer from night blindness. Smokes 20 cigarettes per day. Drinks beer heavily.

O.E. severe caries of teeth with tartar and brown staining. Gums ulcerated and covered with whitish membrane especially behind right lower molar tooth. Breath bad. No glossitis. Tonsils clear. Pulse shows occasional extrasystoles, possibly from the oral sepsis.

Tender shotty glands R. side of neck. Complexion sallow.

Lungs - signs of bronchitis.

Heart - occasional extrasystoles, no murmers. C.N.S. - N.A.D.

No xerophthalmia.

X-ray of chest "Right apex opaque from ? positioning; No infiltration. Congestion and increased shadowing of right base".

Sputa on 28/1/41, 29/1/41 and 30/1/41 - no T.B. detected.

Course:- Day 1. Had the routine scaling, rotary brush treatment and spraying with Eusol solution. Gums painted daily with 10% chromic acid and frequent mouth washes of Eusol 1 in 4. Also bed, full diet and malt and cod liver oil.

Day 4. Complained of earache on R. side. No inflammation of of tympanic membrane. Gums I.S.Q. Chromic acid and Eusol m.w. continued and G.A.C. ear drops given + 15 gr. aspirin in the evening and mist. gent. alk. 1 oz. t.d.s. half an hour before meals.

Day 5. Earache gone and he felt better. The gums were still red and spongy and had the whitish membrane present in the morning. 0.45 gram N.A.B. was given intravenously and vigorous local treatment continued.

Day 7. Slight improvement detected. Gums less red. No pain in neck glands. Chest clear of bronchitis.

Day. 8. Membrane shows less tendency to reform. Less bleeding and no pain in gums now.

Day 11. Discharged from hospital.

This case began to improve after intravenous arsenic on the 5th day.

Diet. Breakfast:- tea,bread, butter ++, jam. Bacon and eggs, fried sausages, porridge. Dinner:- Meat, pork, liver or fish. Potatoes, tinned peas or carrots. Pudding twice a week. No fresh fruit for several months. Tea:- Tea, bread, butter and jam. Supper:- Sausage and mash, or fish and chips.

Case 2. J.G. Able Seaman, 26 years. H.M. Yacht 13 months.

On admission:- temp. 98.2, pulse 85 per minute, bowels regular once a day; tourniquet test negative. Urine - clear, S.G. 1018, acid; bile, blood, sugar and albumen - nil. Gum swab "positive". Blood count. R.B.C. - 5,100,000, Hb. 92%, C.I. 0.9. W.B.C. 8,500/c.mm. Polymorphs 68%, lymphocytes 23%, monocytes 5%, Eosinophils 3%, basophils, 1%.

Urinary vitamin C estimation. 1st specimen - 3.85 mg.%. 3 hours spec. 2.27 mg. %, 6 hours spec. 2.22 mg. % (vitamin C deficiency).

Nicotinic acid excreted per day - 9.3 mg. in 23 ozs. urine.

Complaint and history. For the past three weeks his gums have bled when brushing his teeth. He brushes his teeth regularly. There has been no pain whatever, but occasionally a bad taste in his mouth, worse in the mornings. Appetite good.

No "indegestion". No bronchitis and has had no other illnesses. No rheumatic pains. Bowels regular once a day with no diarrhoea. No other member of his Mess affected (7 in the Mess). Does not drink alcohol. Smokes 20 cigs.a day and 2 ozs. tobacco a week. O.E. Healthy looking, well nourishes patient. Mucosae red and healthy. Gums - Swollen and red with prominent interdental papillae. Bleed easily. Membrane and ulcer on palate behind upper, central incisors. Teeth - no tartar but two decayed molars on left lower jaw. False membrane and bleeding area in front of upper jaw on either side of raphé of upper lip and extending over all the upper gums. Shotty adenitis right and left. Tender left side. Bad breath. No glossitis but furred tongue. Tonsils clear. No xerophthalmia or corneal ulceration. Heart, lungs, abdomen and C.N.S. - N.A.D. Course.- Day 1. Routine scaling, brushing, spraying and painting with chromic acid. Decayed molars stopped with temporary carbolised dressing. M.W. Eusol 1 in 4, t.d.s. Bed and full diet. 0.45 gram N.A.B. intravenously. Saturate with 2,800 mgs. ascorbic acid per day.

Day 4. Now saturated with ascorbic acid. Gums I.S.Q. Treatment continued and ascorbic acid cut down to 300 mgs. per day. Day 6. swab still positive first thing in morning. Gums less red and not bleeding so readily.

Day 8. Gums firmer, still some ulceration. Treatment continued. No tenderness in neck.

Day 10. Ulceration confined to a small area on R. side of upper gum.

Day 12. Ready for discharge.

Diet.- Not canteen messing. Food apt to be overcooked. Breakfast - bacon and tinned tomatoes, (well fried and overcooked), beans, fried bread and bacon; eggs two or three times a week. Bread, butter + + and tea. No grape fruit or any other fruit. Dinner- boiled potatoes, roast meat, cabbage and dried peas soaked in water, stew, cottage pie, boiled pudding or pastry; tinned pears twice a week, no oranges, apples or fresh fruit. Tea-

Scotch rarebit, scrambled eggs on toast, sausage and mash or fresh fish two or three times a week. Supper - bread, butter, cheese or jam, tea or coffee.

A diet, rich in all but vitamin C possibly, which would be killed by the food being overcooked as the patient described.

Case 3. A.G. 36 years. Stoker, Paddle minesweeper X for 6 mths. On Admission. Temp.99.2°. Pulse 86 per min. Bowels - constipated for 2 days. Tourniquet test negative. Urine - straw coloured. S.G. 1022. Albumen, bile, blood and sugar - nil. Throat and gum swabs positive.

Blood count. R.B.C. 3,950,000 / c.mm. Hb. 86%, C.I. 1.1 W.B.C. 9,500/c.mm. Polymorphs. 78%, Lymphocytes 20%, monocytes 2%. Eosinophils 0%, basophils 0%.

Urinary vitamin C estimation. 1st spec. urine - 2.5 mg.%, 3 hours spec. - 4.5 mg.%, 6 hours spec. 3.3 mg.% (vitamin C deficiency)

Nicotinic acid excreted per day - 8.8 mg. in 47 ozs.

Complaint and history.- Sore throat for 3 days and his gums have been bleeding for this time also. Gums however have been tender for 5 days. Sour taste in mouth in mornings lately. Appetite good - wants to eat more than formerly. No bronchitis. No rheumatism but has had stoker's cramps in abdomen and arms in hot weather. No nyctalopia. Constipated lately - usually regular. Smokes 20 cigs.a day and is a non-drinker.

O.E. Thin patient, rather pale. Left cervical adenitis + + . Breath heavy; pale mucosae of eyes; gums red, swollen and bleed easily. Shallow ulcers with greyish yellow sloughs at teeth bases. Tartar and brown staining at teeth bases. Has a plate above with 8 false teeth. Gums sore beneath the plate. No Caries in remaining teeth, but tartar + + at bases. Tonsils - exudate of Vincent's Disease on left tonsil. No xerophthalmia or corneal ulceration. Heart, lungs, abdomen and C.N.S. - N.A.D.

Course.- Day 1. Routine scaling, rotary brushing and spraying of teeth with 1 in 4 Eusol. Gums painted with 10% chromic acid. 0.45 gm. N.A.B. given intravenously. M.W. of potassium permanganate. Bed. Diet low with extras. Two vegetable laxative pills.

Day 4. Membrane on left tonsil has not spread, is yellowish and is peeling off at upper edges. Gums I.S.Q.

Day 6. Practically no membrane on tonsil now. M.W. permanganate t.d.s. and daily chromic acid to gums continued.

Day 7. Tonsils clear. Gums becoming firmer and not so red and swollen. Eusol 1 in 4 applied to gums.

Day 9. Big improvement in gums. Ulceration gone. Eusol and chromic acid continued.

Day 10. Disease is dead. Negative swabs.

Diet.- Canteen messing. Breakfast - Slice of bacon and sausage or egg. Bread, butter + +, tea and jam, no porridge and no fruit. Dinner - Potatoes, meat and vegetables (carrots, turnips, dried peas and cabbage), no pudding. No fruit whatever as he has never had any fruit in this ship. Tea - Bread, butter, jam and tea. Supper - Tea and sandwich with meat or cheese. A monotonous unvaried diet, with little vitamin C. He gets plenty of the articles named.

Case 4. T.K. 19 years. A.B. Paddle minesweeper Y for 5 months. On Admission. Temp. 98.4°. Pulse 92 per min. Bowels constipated slightly. Tourniquet test negative. Urine - pale straw colour, S.G. 1015. Albumen, bile, blood and sugar - nil. Gum swab positive. Blood count. R.B.C. 4,200,000 per c.mm. Hb. 80%. C.I. 0.91. W.B.C. 13,000/c.mm. Polymorphs. 67%. Lymphocytes 28%. Monocytes 4%. Eosinophils 1%, basophils 0%. Urinary vitamin C estimation - 1st spec. urine - 2.5 mg %. 3 hours spec. 1.8 mg.%, 6 hours spec. 2.63 mg.% (vitamin C deficiency).

Nicotinic acid excreted per day - 2.87 mg. in 78 ozs.

Complaint and history. Complains of bleeding and very tender gums for a week. Does not brush his teeth as a rule. Very bad taste in his mouth in the morning. Appetite is good. No vague pains, bronchitis, nor "indegestion". He sleeps well and does not dream. He has felt off colour and easily tired lately and has noticed a slight loss of weight. History of epistaxis on occasion for the past week on blowing his nose. Drinks much beer. Smokes 20 cigs.

a day. No night blindness. There are twenty men in the Mess and for the past 5 weeks they have used 2 cups only. The other cups were broken at sea.

O.E. Tartar at teeth bases. Slight caries of lower molars on right side. Mucosae of gums, swollen, red and easily bleeding. Tonsils clear. Membrane and ulceration on lower gums. Ulcer with whitish slough behind left lower molar. Heavy breath. No glossitis. Shotty, non tender adenitis on left side of neck. Lungs, C.N.S. heart and abdomen - N.A.D. No eye defects.

Course.- Day 1. Routine scaling, rotary brushing, Eusol spraying and painting of gums with 10% chromic acid. 0,45 gm. of N.A.B. given intravenously. 2,100mg. ascorbic acid given daily to saturate him.

Day 3. Gums I.S.Q. and not painful. Random spec. of urine at 11 a.m. showed 8.3 mg. % of ascorbic acid - not quite saturated. Continue 700 mg. ascorbic acid per day.

Day 4. Now saturated with ascorbic acid. Put on 200 mg. per day. Daily 10% chromic acid to gums and 1 in 4 Eusol M.W. t.d.s. Gums less swollen and red but whitish membrane still present.

Day 7. Great improvement in gum swelling. No bleeding. Still some ulceration behind and around lower left molars but this is healing.

Day 9. Fit for discharge.

Diet.- He says the food is good in quality but he complains of its monotony. Breakfast - sausages, or bacon and many fried eggs, bread, butter and tea, tinned milk. Liver and "mash", tinned pork and beans. Porridge available but not eaten by him. Dinner - Roast beef, roast potatoes and tinned peas or cabbage. Jam roll, or jelly and rice. No tinned fruits. The only fruit he has seen for three months was tinned apples on one occasion. Tea - bread, butter, jam and tea. Supper - Egg and chips, or egg and beans. Tea, bread and butter.

This is the invariable diet and is not rich in vitamin C.

Case 5. J.T. 23 years. A.B. Paddle minesweeper Y. for 3 months. and trawler previously for 3 months.

On admission. Temp 97.8°. Pulse 65 per min. Bowels regular 1 i.d. Tourniquet test negative. Urine - S.G. 1022. Acid. Bile, blood, albumen and sugar - nil. Gum swab positive.

Blood Count. R.B.C. 4,750,000 /c.mm. Hb. 90%. C.I. 0.96. W.B.C. 6,500/c.mm. Polymorphs. 72%, Lymphocytes 20%, Monocytes 5% Eosinophils 2%, basophils 1%. Urinary vitamin C estimation - 1st spec. 5 mg. %, 3 hours spec. 4.2 mg. %, 6 hours spec. 4.0 mg.%. (Vitamin C deficiency).

Nicotinic acid excreted per day - 3.83 mg. in 44 ozs. urine.

Complaint and history.- Complains of bleeding, tender gums for 2 weeks. Aching like toothache all over, but was worse on the left side to start with. He brushes his teeth twice a day. His appetite is good, slight loss of weight lately. No diarrhoea, bronchitis or recent indigestion. He had "stomach trouble" 4 years ago. No history of rheaumatic pains and he sleeps well with no dreams. Has not been feeling up to the mark lately and has had a bad breath and foul taste in his mouth. No trouble with his eyesight at night. Non drinker but a heavy smoker.

O.E. Thin patient, with seborrhoeic spots on face and shoulders. He has 6 false teeth in the upper jaw in front. The teeth in the lower jaw have some caries in places but no gross tartar. Some brown staining at the bases. There is some ulceration and whitish membrane on both gums and on the palate behind the upper central incisors. Tonsils are clear of infection. The breath is heavy and the gums swollen, and the interdental papillae prominent. A moderately severe case. No defect in the eyes. Tender adenitis present left side of neck. Heart, lungs, abdomen and C.N.S. - N.A.D.

Course. Day 1. Routine scaling, rotary brushing, spraying with Eusol 1 in 4 and daily painting of gums with 10% chromic acid. Saturate patient with ascorbic acid (700 mg. t.d.s.).

Day 2. - Continue with 10% chromic acid + 700 mg. ascorbic acid

t.i.d. + Eusol (1 in 4) M.W.

Day 3. Random specimen urine shows 7.6 mg.% ascorbic acid - not yet saturated. Continue vitamin C.

Day 4. Saturated with ascorbic acid. Gums I.S.Q. Still pain in left side of upper and lower jaws. Ascorbic acid - 200 mg. per day - given.

Day 7. Less pain in gums. Interdental papillae less swollen and less red. Ulceration vanishing. No tenderness in cervical glands, but glands still enlarged.

Day 9. Gums practically healed.

Day 10. Ready for discharge.

Diet - Same as case 4.

Case 6. F.C. 26 years. Minesweeper for 9 months.

On admission. Temp.100.2°. Pulse 94/min. Bowels regular every day. Tourniquet test negative. Urine - dark, straw colour. S.G. 1025. Albumen, bile, blood and sugar - nil. Gum and throat swabs positive.

Blood count. R.B.C. 3,650,000 per c. mm. Hb. 64%. C.I. 0.89. W.B.C. 12,000/c.mm. Polymorphs. 75%. Lymphocytes 20%, monocytes 5%. Eosinophils 0%. Basophils 0%.

Urinary vitamin C estimation. First spec. -3.4 mg.%, 3 hours spec. 3.8 mg.%, 6 hours spec. 4.5 mg.% (vitamin C deficiency).

Nicotinic acid excreted per day - 13.13 mg. in 35 ozs. urine.

Complaint and history. - Sore throat and difficulty in swallowing for 4 days prior to admission. Gums have bled on cleaning for the past week. He used to clean them every day till they started to bleed. No bronchitis, nor rheumatism. He sleeps soundly and rarely dreams. No loss of weight nor "indigestion". Smokes 14-15 cigs per day. Drinks every week-end and gets "drunk" every Saturday night.

O.E. Thin, pale subject. Teeth - no caries but tartar at the bases. The crowns are clean. Alveolar abscess in left lower jaw. Small ulcer in front of left anterior pillar of the fauces. White spots on both tonsils. Gums red, swollen and bleed easily on pressure. Whitish membrane of Vincent's disease on anterior aspect of upper and lower jaws. Breath very offensive. No glossitis.

Tender R. and L. cervical adenitis. Worse on left side. No xerophthalmia or corneal ulceration.

Heart, lungs, abdomen and C.N.S. - nil.

Course.- Day 1. Two teeth with alveolar abscess at roots removed. Routine scaling, brushing, spraying and painting daily with 10% chromic acid. .45 gm. N.A.B. intravenously. Mist. ferri et ammon. cit. and .45 gm. N.A.B. 700 mgs. vit. C. T.D.S.
Day 3. Now saturated with vitamin C. therefore put on 200 mg. daily.

Daily gums paints with chromic acid and M.W. of Eusol.

Gums I.S.Q. tender adenitis R. side less tender on L.

Day 4. Gums I.S.Q. but marked improvement in throat. No pains in throat or neck, and spots almost gone.

Day 7. Throat clear. Alveolar abscess has stopped draining and is healing in from below. Gums less red and much firmer. Still some membrane in the morning.

Day 9. Only a small ulcerated area on L. side of upper jaw. Neck glands shotty but not paining. Eusol irritating gums therefore used pot. permang. M.W. + daily chromic acid.

Day 11. Discharged.

Diet. - Breakfast - Fish every morning, porridge, bread, toast, butter and tea. No fruit whatever in diet. Last fruit was 3 apples at Christmas, nine weeks previously and he can't remember any before that. Dinner - Meat, potatoes and greens or tinned peas, sago, tinned plums or custard. Tea - Toast, jam, butter and tea. Supper - Meat, potatoes and cabbage. Liver is available but he does not like it.

Case. 7. M.P. 27 years. Paddle minesweeper Y for 5 months.

On admission. Temp. 99°. Pulse 90 per min. Bowels constipated. Tourniquet test negative. Urine - S.G. 1035. Blood, bile, albumen and sugar - nil. Gum and throat swabs positive.

Blood count. R.B.C. 4,950,000/c.mm. Hb. 85%. C.I. 0.86. W.B.C. 13,750/c.mm. Polymorphs. 62%. Lymphocytes 34%. Monocytes 2%. Eosinophils 2%. Basophils 0%.

Urinary vit. C. estimation. First spec. urine 2.5 mg.%. 3 hours spec. urine 3.33 mg.%. 6 hours spec. 3.12 mg.%. (vitamin C deficiency).

Daily nicotinic acid excretion 9.6 mg. in 46 ozs. of urine.
Complaint and history.- Had pains like toothache in his gums for a week. Gums bleeding before this time for 3 days. Bad taste in mouth. Pain in throat and dysphagia for 2 days. Constipated lately. No diarrhoea, muscular pains, bronchitis, nor "stomach" trouble. No undue difficulty in seeing at night lately. Drinks moderately. Smokes 10 cigs. per day. Appetite good.
O.E. gums swollen, red and inclined to bleed. Very slight disease in region of upper L. molars. Alveolar abscess around upper L. molars. Tartar at teeth bases. L. tender cervical adenitis present.
L. tonsil has white granular patch on upper pole. Tonsil not grossly enlarged. Heart, lungs, abdomen and C.N.S. - N.A.D.
No xerophthalmia.
This was a very mild case indeed and would not have been admitted had it not been for the initial pyrexia of 99°.
Course.- Day 1.- Diseased upper L. molars pulled. Routine scaling, rotary brushing, spraying with Eusol (1 in 4) and daily painting with 10% chromic acid. This man refused N.A.B. Eusol M.W. and gargles given 4 i.d.
Saturate with ascorbic acid, 14 tabs. t.d.s., liquid paraffin and cascara 2 dr. of each.
Day 2. - Temp. normal all day. Gargle pot. chlr. 4 i.d.
Day 3. - Still complains of pain in gums.
Day 4. - Still ulcer L. tonsil and tender L. cervical adenitis. Now saturated with ascorbic acid at 5 p.m. Tooth socket cleaning up.
Day 5. - Ulcer and white patch of membrane persist on L. tonsil and pain on swallowing. Continue ascorbic acid 300 mg. /day. Daily chromic acid and Eusol M.W.
Day 6. - Practically no change in gums and tonsils. Area of membrane on tonsil is very slightly smaller than when he was admitted.
It is felt that the case would have cleared by now with N.A.B. but he will not have it.
Day 7. - Slight improvement commencing in gums and spot on top of

L. tonsil smaller. No injection of rest of throat now. Garg. pot. chlor. Daily chromic acid paints on gums and ascorbic acid, 300 mg./ day.

Day 8. - Gums now appear healthy. Spot on top of L. tonsil very small. Shotty non-tender adenitis in neck now. Alveolar abscess healed.

Day 9. - Membrane gone from tonsil, and ready for discharge.

Diet - Same as cases 5 and 4.

Case 8.- J.S. 24 years. Drifter for 4 months.

On admission;temp. 98.2°. Pulse 72/ min. Bowels open. Tourniquet test negative. Urine - S.G. 1028. Acid. Albumen, blood, bile and sugar - nil. Gum swab positive.

Blood count. R.B.C. 3,280,000. /c.mm. Hb. 70%. C.I. 1.09. W.B.C. 6,900 /c.mm. Polymorphs. 80%. Lymphocytes 15%. Monocytes 3%. Eosinophils 1%. Basophils 1%.

Urinary vit. C. estimation. First spec. urine 2.01 mg.%. 3 hours spec. 2.6 mg.%. 6 hours spec. 2.63 mg. %. (vitamin C defic.). Nicotinic acid excreted per day 8.34 mg. in 43 ozs. urine.

Complaint and history.- Three weeks ago was in another hospital with Vincent's disease of the gums for a week, then went on 7 days leave. Now returns with pain, bad taste in mouth, bad breath and bleeding gums. Appetite poor and has had gastric trouble, with pain and flatulence for 5 weeks. Non drinker and only smokes 5-10 cigs.per day. No diarrhoea and no bronchitis or rheumatic pains.

O.E. Bad breath and lower molars on R. side loose and decayed stumps present. Moderately severe case of Vincent's Disease of the gums and R. retro-molar sulcus. Heart, lungs, and C.N.S. - N.A.D. Abdomen - some tenderness in region of R. upper rectus and muscle guarding. No eye defects. No glossitis. No tartar in teeth.

Course. - Day 1. Routine scaling, etc. of teeth. M.W. Eusol and 10 chromic acid. 14 tabs. vit. C. T.d.s. Milk diet and mist. ferri et ammon. cit..

Day 2. - 0.45 grm. N.A.B. intravenously. Continued vit. C. Still complains of pain in upper and lower jaws.

Day 4. - Complains of headache and aching pains all over. Temp. 98.4°. Heart and lungs nil. Random spec. urine had 50 mg.% ascorbic acid (now saturated). Continue 300 mg. vit. C. per day.

Day 5. - Still has severe pain in gums. General aches gone. Temp. 98.4°. Continue daily acid chrom., Eusol M.W., vit. C. 300 mg./day and luminal grains 1. b.i.d.

Day 6. - Temp. 99°. Bowels not open. Gums not so tender and are firming up and losing their bright red appearance. Decayed stumps alone are paining and are extracted today under gas. Throat N.A.D. Chest N.A.D. Shotty, non tender glands R. and L. sides of neck. Mist. alba (2 ozs.) and routine daily gum treatment.

Day 7. - Face swollen after stump extraction. Feels better but upper jaw aching. Constipation persists (enema given). Ascorbic acid, etc. continued. Full diet.

Day 8.- Redness in gums mostly gone though still membrane in places on upper jaw. Swelling of face less and stump sockets fairly clean. Feels better. No gastric complaints. Bowels open.

Day 10. - Gums have slight infection and are sore only where teeth were removed. Continue chromic acid, Eusol and ascorbic acid.

Day 12. - Much better. Sockets almost healed.

Day 13. - Ready for discharge.

Diet.- Breakfast- Tea, bread, butter (ad lib.), jam, bacon and eggs and porridge, tinned sausages and eggs. Dinner - Meat, roast or boiled potatoes, cabbage, sprouts, turnip or tinned carrots. Sweet on Sundays only and then "plum duff". Tea- Cup of tea, and bread and jam. Supper at night - Liver and bacon, with"beans and pork", meat and chips, fresh fish and chips, tea, bread, butter and cheese.

Case 9. - M.M. 29 years. Minesweeper for 4 months.

On Admission. Temp. 97.8°. Pulse 68/min. Bowels regular once per day. Tourniquet test negative. Urine - straw coloured. S.G. 1030. Acid. No blood, bile, sugar or albumen. Throat and

gum swabs positive.

Blood count. R.B.C. 5,100,000 per c.mm. H.b. 94%. C.I. 0.92. W.B.C. 10,500. / c.mm. Polymorphs. 77%. Lymphocytes 21%. Monocytes 2%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. First spec. urine 2.5 mg.%. 3 hours spec. 2.76 mg.%. 6 hours spec. 3.18 mg.%. (Deficiency of vit.C.).

Nicotinic acid excreted per day equal to 11.15 mg. in 40 ozs. urine.

Complaint and history.- Pain in throat and tenderness on L. side of neck for 2 days. No pain in gums and they have not been bleeding. Appetite good and has felt perfectly well till a few days ago. No diarrhoea, insomnia, gastric trouble, vague pains, eye trouble nor bronchitis. Slight dysphagia. Smokes 2 cigs. per day. Heavy drinker.

O.E. Ulcer with white membrane over it on upper pole of L. tonsil. Patient looks pale, breath heavy. R. and L. cervical, tender adenitis present. Tonsils red, but not grossly swollen. L. tonsil sightly larger than R. Gums have whitish membrane on them and are slightly swollen. Some tartar of teeth and early caries of lower L. molars (this is a very mild case of gum infection). Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.

Course.- Day 1. - Routine scaling, brushing, Eusol spraying and application of 10% chromic acid to the gums. 0.45 gm. N.A.B. intravenously. Garg. pot. chlor. 4 i.d.. Saturate with ascorbic acid, 700 mg. t.d.s.

Day 2.- Tonsils still injected. Small white patch persists on L. side. Temp. down. L. adenitis still tender.

Day 3. Throat still sore, but easier than previously. Membrane has vanished from throat but tonsils still swollen. Gums I.S.Q.

Day 4. - No complaints save still very slight pain in L. side of throat. L. tonsil larger and redder than R. L. cervical glands shotty and tender. 300 mg. ascorbic acid t.d.s. as a random spec. urine shows 45 mg.% of ascorbic acid.

Day 6.- Gums appear healthy. Throat better. Shotty non tender glands R. and L. Day 7.- Fit for discharge.

Diet. - Same as case 6.

Case 10. - E.D.N. 35 years. Lt. R.N.V.R. Trawler for 1 year off Faroe Islands.

On admission. Temp. 98.4°. Pulse 70 /min. Bowels open once every day. Tourniquet test negative. Urine clear. S.G. 1015. Acid. bile, blood, sugar and albumen - nil. Gum swab positive. Blood count. R.B.C. 3,840,000 /c.mm. Hb. 78%. CI. 1.01. W.B.C. 9,800/c.mm. Polymorphs. 57%. Lymphocytes 34%. Monocytes 7%. Eosinophils 1%. Basophils 1%. Urinary vit. C. estimation. First spec. urine 20 mg.%. 3 hours. spec. 40 mg.%. 6 hours spec. 53 mg.%. (no sub-scurvy now, but has had ascorbic acid before admission). Nicotinic acid excreted per day equal to 8.75 mg.

Complaint and history.- This officer has spent a year in Faroe Island waters on a trawler. He had no fresh fruit or fresh food during that period except when they called at Kirkwall when they obtained fresh meat. Fish and butter were plentiful in the diet however. The meat was nearly always tinned.

He was in another hospital in Octover 1940 for 5 days with his gum condition but he had to return to duty before cure was complete. He has been taking ascorbic acid regularly since then (11 weeks). He has had no local treatment and his gums are in the same condition as before. He now complains of bleeding gums. There is often blood on his pillow. Pain is present all over. He has an extremely bad taste in his mouth in the mornings. Appetite poor. No history of diarrhoea, chest trouble, night blindness nor rheumatic pains. Drinks heavily, smokes half an ounce pipe tobacco per day.

O.E. Gums swollen, red and bleed easily. Yellowish marginal ulcers. Tartar of teeth but no caries. Some exudate on anterior aspects and in both retro-molar sulci. Breath very foul. Furred tongue but no glossitis. Shotty, non tender adenitis R. and L. Tonsils clear. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.

Course. - Day 1. Gums swabbed with N.A.B. (.3 grm. in 15 cc. water) after spraying with hydrogen peroxide (1 part to water 3 parts) Teeth not to be brushed at first. Mist. ferri ammon. cit. 1 oz. T.D.S. Vit. c. - 250 mgs. / day given.

Day 7. - Gums still slightly swollen,bleeding in places and slightly

sore. They looked rather better but still abnormally red at the margins. He was made to stop smoking which he had resumed after his nicotinic acid test. Teeth scaled and polished for the first time and mouth irrigated with Eusol, as hydrogen peroxide was causing tenderness and redness of the gums. Some bleeding.

Day 10.- No change.

Day 14.- Condition has cleared.

Day 15. - Fit for duty.

Diet. - See history of case.

Case 11. J.P.O. 35 years. Patrol ship for 4 months.

On admission. Temp. 97.8°. Pulse 80/min. Bowels open, no diarrhoea. Tourniquet test negative. Urine. Acid. S.G. 1024. Albumen, sugar, bile and blood - nil. Deposit of urates. Gum swab positive.

Blood count. R.B.C. 4,300,000./c.mm. Hb. 80%. C.I. 0.93. W.B.C. 13,600./c.mm. Polymorphs. 82%. Lymphocytes 15%. Monocytes 3%. Eosinophils. 0%. Basophils 0%.

Urinary vit. C. estimation. First spec. urine 1.3 mg. %. 3 hours spec. 2.4 mg.%. 6 hours spec. 2.2 mg.%. (def. in vit.C.).

Nicotinic acid excreted in 24 hours equal to 8.2 mg.

Complaint and history. - Pain in gums for three weeks and bleeding gums for one week. Heavy breath and taste in mouth in morning. Brushes teeth regularly. Heavy drinker. Smokes 20 cigs. per day. Discharged from U.S. Navy in 1937 owing to chest trouble. No diarrhoea, rheumatic pains, or trouble with eyes. No night sweats. Suffers from breathlessness in confined spaces. No cough. Rheumatic fever 9 years ago. Ankles used to swell but not lately.

O.E. Vincent's Disease of gums - white membrane visible all over. Tongue furred. Breath bad. Prominent interdental papillae. No glossitis. Tonsils had been removed and no disease of tonsil beds. Thin patient, rather pale. Chest - R.M. vesicular in all areas with scattered rales at bases and both apices. Rales very scanty. Heart, C.N.S., abdomen and eyes N.A.D.

swabs positive.

Blood count. R.B.C. 4,900,000/c.mm. Hb. 94%. C.I. .96. W.B.C. 10,800 /c.mm.

Polymorphs. 70%. Lymphocytes 22%. Monocytes 6%. Eosinophils 2%. Basophils 0%.

tooth. X-ray of chest -apices clear. Some evidence of chronic bronchitis

tooth. X-ray of chest -apices clear. Some evidence of chronic bronchitis

and increased shadowing of bases.

No sputum produced.

Course. Day 1. Routine scaling, rotary brushing, Eusol spray and 10% chromic acid paints. Saturate with ascorbic acid - 700 mgs. t.d.s.

Day 2. - M.W. Eusol and usual gum treatment.

Day 3. - Gums I.S.Q. .45 gm. N.A.B. intravenously. Has had 4200 mgs. vit. C. to date and is still not saturated. Continue 14 tabs. vit. c. t.d.s.

Day 4. - Gums I.S.Q. and aching. Throat clear and no cough. Now saturated with vit. c., therefore give 300 mgs./day and routine Eusol m.w. and chromic acid.

Day 5. - Gums still tender. Patch on L. tonsil today. Swab neg. for Vincent's organisms or H.L.B. Tender L. cervical adenitis. Heavy breath. Usual treatment + Garg. Pot. chlor.

Day 6. - Temp. 99.8°. Throat and uvula red. -superimposed streptococcal infection (several cases in ward.) M and B 693 2 t.d.s. + routine treatment.

Day 7. - Gums I.S.Q. though slightly less red. Temp. normal. Throat better. Shotty non-tender cervical adenitis. M and B discontinued. Chest has temporarily cleared with rest and warmth.

Day 11. - Visible improvement in gums but not yet ready for discharge.

Day 14. - Disease confined to small patch on upper jaw's outer aspect at canine teeth. Rest of gums firm and pink.

Day 16 - Discharged.

Diet. - Breakfast. Tea or coffeee, tinned milk, sugar, bread and butter ++. Fresh fish or sausages (tinned) or bacon and eggs. Dinner - Meat, potatoes, tinned carrots or cabbages. Plum duff or rice pudding. Supper - Liver and bacon, sausages and mash, Irish stew or kippers, potatoes, bread, butter, jam and tea.

Case 12. W.F. 22 years. Paddle minesweeper (X) for 5 months. Destroyer previously.

On admission. Temp. 98.4°. Pulse 86/ min. Bowels open. Tourniquet test negative.

Urine. S.G. 1020. Acid. Bile, blood, sugar and albumen - nil. Gum and throat swabs positive.

Blood count. R.B.C. 4,900,000/c.mm. Hb. 94%. C.I. .96. W.B.C. 10,800 /c.mm.

Polymorphs. 70%. Lymphocytes 22%. Monocytes 6%. Eosinophils 2%. Basophils 0%.

Urinary vit. C. estimation. First spec. 2.13 mg.%. 3hrs. spec. 2.45 mg.%. 6 hrs. spec. 2.2 mg.%. (Vit. C. deficiency).

Nicotinic acid excreted per day equal to 6.3 mgs. in 39 ozs.

Complaint and history. - Sore throat for 8 days. Gums sore for 4 months and have been bleeding on and off since then. Now has pains in the gums. Also has pain in R. side of neck. Appetite poor lately and slight loss of weight. No diarrhoea, bronchitis or "rheumatic" pains. No trouble with his eyesight. Non-smoker and non-drinker.

O.E. Breath foul. Tongue moist and furred. Gums swollen, red and tender. Ulceration at teeth margins. Tartar on teeth bases and white membrane on outer aspects of gums and behind upper central incissors. R. tonsil red, swollen and ulcerated, with a whitish membrane in the centre. R. tender, submaxillary adenitis.

Heart, lungs, abdomen and eyes - N.A.D..

Course.- Day 1. Saturate with ascorbic acid, 700 mgs. t.d.s. Routine scaling etc. and 10% chrom. acid daily. .45 gram. N.A.B. intravenously.

Day 3. - Throat free from pain. Membrane getting yellowish and granular. Gums I.S.Q.

Day 5. Throat rapidly improving. Cervical glands shotty and non-tender. Gums less red and firming. Still have whitish membrane of Vincent's disease on them in the mornings. On 300 mgs. ascorbic acid daily.

Day 7. - Throat better. Gums improving. Ulceration clearing round tooth bases.

Day 9. - Only one small, red tender area round base of L. lower bicuspid tooth.

Day 10. - Gums healed. Ready for discharge.

Diet History. Same as case 3.

Case 13. M.N. 43 years. Paddle sweeper (X) for 8 months.

On admission. Temp. 97.2°. Pulse 72 per minute. Bowels open but no diarrhoea. Tourniquet test negative. Urine clear and watery looking. S.G. 1010. Acid. Bile, blood, albumen, sugar - nil. Gum swab "positive".

Blood count. R.B.C. 4,250,000/c.mm. Hb. 86%. C.I. 1.02. W.B.C. 11,000/c.mm. Polymorphs. 70%. Lymphocytes 24%. Monocytes 5%. Eosinophils 0%. Basophils 1%.

Urinary vit. C. estimation. First spec. .8 mgs.%. 3 hrs. spec. urine .87%. 6 hrs. spec. .91%. (def. of vit. c.).

Nicotinic acid excreted per day equal to 5.1 mgs.

Complaint and duration. - Sore gums for one month and bleeding on top and bottom

gums. Consulted a dentist the day before admission to have a tooth extracted which was done (upper L. bicuspid) and the condition was discovered. He smokes 10 cigs. per day and drinks heavily. Has been out of sorts lately. No diarrhoea, pains in limbs or eye trouble. Has had a cough lately and wheeze at nights.

O.E. Both gums infected with Vincent's disease. Swollen, tender. Prominent interdental papillae and membrane in R. retro-molar sulcus. Breath heavy. No glossitis. Tonsils, red, but no ulceration or membrane.

Cervical adenitis R. and L. Caries and tartar of teeth. Heart, abdomen and C.N.S. - N.A.D. No keratomalacia. Lungs - slight bronchitis with vesicular R.M. and scattered rales.

Course. Day 1. Full diet. Saturate with ascorbic acid. 700 mgs. t.d.s. Routine scaling etc., and 10% chromic acid applied daily. Carbolised dressing to carious right lower molar. Mist expectorans t.d.s.

Day 4. - Gums only slightly tender. Still some membrane in places on outer aspects. Red colour getting less intense. Slight cough but chest clear. Ascorbic acid - 300 mg./day. H.W. Eusol. Daily chromic acid paints.

Day 6. - The only tender area is round a filled tooth in the upper left bicuspid area. Only slight bleeding now. Eusol M.W. etc. and ascorbic acid continued.

Day 8. - No pain, and redness gone.

Day. 9. - Discharged.

Diet. Same as case 3.

Case 14. - A.W.P. 21 yrs. Trawler (Z) for 10 months.

On admission. Temp. 97.2°. Pulse 66/min. Bowels open, but no diarrhoea. Tourniquet test positive. Urine - S.G. 1030. Acid. No bile, blood, sugar or albumen. Gum swab "positive."

Blood count. - R.B.C. 4,000,000 /c.mm. Hb. 78%. C.I. .975. W.B.C. 7,800/c.mm. Polymorphs. 70%. Lymphocytes. 27%. Monocytes 3%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. - First spec. 2.5 mgs.%. 3 hrs. spec. 1.6 mgs.%. 6 hrs. spec. 1.6 mgs.%. (Vit. C. deficiency).

Nicotinic acid excreted per day equal to 4.14 mgs.

Complaint and history.- Gums bleeding on cleaning teeth for 3 days. Cleans teeth regularly. No pain. Smokes 12 cigs. per day. Fairly heavy drinker, 4 or 5 pints of beer every night. Appetite good. No loss of weight. No previous rheumatism or

diarrhoea. No chest troubles. He has felt perfectly fit lately.

O.E. Moderate degree of disease of gums. Copious white membrane all over. Breath offensive. Interdental papillae prominent and gums red. No tartar but brown stain at base of teeth and a decayed molar on the left side. No adenitis. No glossitis. Lungs, heart, abdomen, C.N.S. and eyes. - N.A.D.

Course. Day 1. Routine scaling, rotary brushing and spraying with Eusol. Daily painting of gums with 10% chromic acid. .45 gram. N.A.B. intravenously. No. vit. C.

Day 5. - Less formation of membrane. Gums firmer and less red. No bleeding.

Day 8. - Gums firm and healthy. No bleeding and no ulceration. Discharged.

Diet History. - Breakfast - Bacon or sausages and eggs every morning. Bread, butter and tea. Dinner. - Meat and potatoes with one vegetable (cabbage or tinned carrots). No fresh fruit, but tinned fruit most days. Supper. - Liver or bacon and potatoes. Fish and potatoes. Coffee, or tea, bread, butter and cheese.

Patient complained of the monotony of the diet which was always overcooked.

Case 15. B.M. 27 years. Trawler (Z) for 11 months.

On admission. Temp. 97.4°. Pulse 70/min. Constipated. Tourniquet test positive. Urine S.G. 1020. Acid. No bile, blood or sugar. Albumen - V.F.T. Gum swab "positive".

Blood count. R.B.C. 4,860,000. Hb. 98%. C.I. 1.02. W.B.C. 9,800 /c.mm. Polymorphs 65%. Lymphocytes 28%. Monocytes 6%. Eosinophils 1%. Basophils 0%.

Urinary vit. C. estimation. First spec. 2.13 mg.%. 3 hours spec. 2.56 mg.%. 6. hrs. spec. 3.14 mg.%. (vit. C. deficiency).

Nicitonic acid excreted per day equal to 8.0 mg.

Complaint and history. - No special complaints but has a bad taste in his mouth in the mornings. Seen by a dentist 4 days ago for a "filling" and condition was discovered. He brushes his teeth regularly. No bleeding recently but he has had periods of bleeding gums on and off for several weeks. Appetite is good. Slight loss of weight lately. Non-smoker and non-drinker. Never has had diarrhoea. 5 months ago had cramp-like pains in his legs but none since then. No lung trouble.

O.E. Teeth very clean and no tartar. Some filled molars one of which was freshly repaired and had had no filling for some weeks. Another tender, carious

molar was present. Slight membrane, redness and swelling of the gums. Tonsils clear. Shotty, non-tender R. and L. adenitis in neck. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D..

Course. Day 1. - Routine scaling, etc. Daily paint with 10% chromic acid. Saturate with ascorbic acid 700 mgs. t.d.s. Temporary carbolised dressing in one carious molar.

Day 2. - Daily treatment + .45 gm. N.A.B. intravenously.

Day 4. Saturated with ascorbic acid. Gums I.S.Q.

Day 6. Gums firmer and less membrane. No bleeding.

Day 8. Only small areas of disease left.

Day 10. Fit for duty. Disease stamped out.

Diet. - Same as case 14.

Case 16. - E.J.W. 33 years. Destroyer for 10 months.

On admission. Temp. 98.4°. Pulse 100 per min. Bowels open but no diarrhoea. Tourniquet test positive (30 spots in 3 mins.). Urine. S.G. 1022 faintly acid. Albumen, bile, blood, sugar - negative. Gum and throat swabs - positive.

Blood count. - R.B.C. 4,560,000 /c.mm. Hb. 103%, C.I. 1.14. W.B.C. 15,000/c.mm. Polymorphs. 68%. Lymphocytes 25%. Monocytes 7%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. First spec. urine 1.8 mg.%. 3 hrs. spec. 2.1 mg.%. 6 hrs. spec. 2.35 mg.%. (Vit. C. deficiency).

Nicotinic acid excretion test not done.

Complaint and history. Gums spongy and prone to bleed for several months. Bad taste in mouth in mornings. Appetite good and no "indigestion". No attacks of epistaxis and no pains in jts. or limbs. Had been at sea since outbreak of war on the same diet. No chest trouble. Throat has been sore for 3 days. Non-smoker, but drinks when ashore.

O.E. Well nourished patient. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.. Tonsils - filmy white exudate in patches on each side and R. tonsil is swollen, ulcerated and has a slough on it. Teeth and gums - Tartar at bases and between the teeth. Extensive caries. Gums swollen and bleed easily from the teeth sockets. Typical filmy exudate on gums' outer margins and in the sulci behind the lower back molars. Small ulcer and white patch behind upper central incissors and small ulcer with whitish slough on posterior part of soft palate. Adenitis in R. sub-maxillary and R. tonsilar glands.

Course. Day 1. Full diet. Routine treatment with scaling, etc. and 10% chromic acid daily. Slough on tonsil treated with 10% carbolic acid. 4 hourly m.w. of $H_2 O_2$.

Day 3. - .3 gm. N.A.B. intravenously.

Day 8. - Tonsils clear. Gums firmer. No evidence of filmy exudate.

Day 9. - Fit for discharge.

Diet history. - Canteen Messing. Breakfast - Bread, butter ++, tea. Liver, bacon, tinned sausages and eggs. Porridge. Dinner - Meat, potatoes, and one vegetable. Pudding. Tinned milk always. Fresh fruit is available but the patient never takes it. Tea - Bread, butter and jam with tea. Supper - Fish , sausage and mash, liver and potatoes. Bread, butter and tea. This is a liberal diet but never varied. The fault was the patients own; that he had never partaken of the available fruit since he disliked fruit.

Case 17. F.J. 18 years. Destroyer (A) for 6 months.

On admission. Temp. 100.6°. Pulse 96/ min. Bowels - tend to be constipated. Tourniquet test done on 4th day - negative. Urine - S.G. 1030. Acid. No bile, blood, albumen or sugar. Gum swab "positive".

Blood count. - R.B.C. 5,200,000/c.mm. Hb. 96%. C.I. .92. W.B.C. 15,500. Polymorphs. 82%. Lymphocytes 18%. Monocytes 0%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation (Done on 6th day). First spec. 1.8 mg.% 3 hrs. spec. 2.7 mg.%. 6 hrs. spec. 4.55 mg.%. (Vit. C. deficiency).

No nicotinic acid estimation done.

Complaint and history. Patient brought to sick bay c/o severe abdominal pain which came on at 11.15 a.m. (10 hours previously) suddenly. Pain was across middle of abdomen and doubled him up. Bowels open previous day and he had had nothing by mouth since P.M. yesterday.

No history of previous stomach trouble. Smokes and drinks in moderation. Tongue coated. Gums swollen, spongy and with Vincent's membrane all over. No caries but tartar of teeth.

O.E. Abdomen held rigid. Pain on pressure in all areas.

No abdominal respiratory movement and abdomen shows no movement on coughing.

P.R. - N.A.D. No vomiting. Chest - N.A.D. Knee jerks +. Pupils react to light and accommodation. Tenderness in epigastrium and in R. Iliac fossa + + . Blumberg's sign + too. Tentative diagnosis was a perforation of part 1 of the duodenum with the contents trickling down the R. para-colic gutter.

Investigations done:- Half hourly pulse - rate rising; Leucocyte count- 15,500 /c.mm.; X-ray diaphragm - no gas pocket seen.

Urinary report - N.A.D.

Abdomen prepared for operation and 1 c.c. Omnopon and Scopolamine given. Abdomen opened under gas, O_2 and Ether. Upper right paramedian incission. Rectus muscle split. Escape of putrid fluid on opening abdomen. Perforation seen on anterior wall of duodenum about 1 ins. distal to pylorus. Perforn. about ½ ins. in diameter and edges almost cartilaginous in hardness. No suture could be made to stay in duodenum either with Lambert suturing or purse-string. After repeated futile attempts to suture the perforation a piece of adjacent omentum was sutured in position over it. Abdomen was further explored and Morrison's pouch and pelvis seen to be full of greenish fluid which did not smell. As much as possible was mopped out and a supra-pubic stab drain inserted down into the pelvis. A further drain was inserted into Morrison's pouch at the upper end and the abdominal incision was sutured in interrupted fashion.

Condition of patient was satisfactory throughout.

1. 8000 units A.G.G. S. given.
2. Rectal drip instituted.
3. Nil by mouth.
4. Mouth toilet t.d.s.
5. Fowler's position on coming round.
6. Morphia gr. one-sixth on recovery and repeated s.o.s.

Day 2. Temp. 100.6°. Pulse 104 per min. Resps. .20/min.

Pain in abdomen but general appearance quite good. Sweating. Is having continuous rectal drip salines.

Day 3. Temp. 99.8°. Pulse 94/min. Resps. 26 per min.

Slight cough. Slight pain in R. hypochondrium probably due to tube. General appearance is good. Tubes shortened 2 ins. morning and evening.

Day 4. Improving.

Day 5. Abdomen soft and becoming slightly distended in lower half. Tubes removed. Urine shows very faint trace of albumin. Rectal fluids discontinued. On day 2 diet. Slight discharge from stab wound. Enema given. No symptoms. Seen by dental specialist who instituted the routine scaling, brushing, etc., and daily painting of gums with 10% chromic acid.

Day 7. i.e. 3rd day of treatment for Vincent's disease. Paint gums with

acid chrom. and n.w. Lusol 1 in 4.

Day 8. No symptoms of stomach trouble. Slight purulent discharge from centre of epigastric wound. Daily gum treatment. Lusol very frequently. 7th day diet.

Day 10. Satisfactory. Remove alternate sutures. Gums becoming paler and firmer. Less membrane in the mornings.

Day 12. All sutures now removed. Slight sinus in abdominal wound. - apply sulphonamide powder. Gums almost healed. Continue daily chromic acid to gums.

Day 14. Gums healed. Still oozing from abdominal sinus.

Day 16. Sinus is now clean. Transferred to medical ward for medical treatment of peptic ulcer.

Vincent's Disease was cleared with 10 days of vigorous treatment. It was a slight attack.

Diet history.- No fresh fruit on the menu. Breakfast - Bacon or steak and eggs. Bread, butter, tea or cocoa. Dinner - Meat, roasted or stewed, every day. Pork on Sundays. Potatoes and tinned peas usually. Other vegetables three times a week, cabbage and swede. Rice pudding or plum duff every day. Tinned milk. Tea - Bread, butter ++, tea and jam. Supper - Pork and beans, sausage (tinned), fish or liver and potatoes. Cocoa, bread and cheese.

Case 18. H.F. 24 years. Trawler (A) for 8 months.

On admission. Temp. 98.4°. Pulse 68 per. min. Bowels constipated. Tourniquet test negative. Urine - clear. S.G. 1018. Acid. No bile, blood, sugar or albumen. Gum swab "positive".

Blood count. R.B.C. - 4,770,000/c.mm. Hb. 92%. C.I. .99. W.B.C. 11,600 per c. mm. Polymorphs. 65%, Lymphocytes 29%. Monocytes 4%. Eosinophils 2%. Basophils 0%.

Urinary vit. C. estimation. First specimen 3.7 mg%; 3 hrs. specimen 20.0 mg.%; 6 hrs. spec. 25.0 mg.%. No subscurvy now.

Nicotinic acid excreted per day equals 6.3 mgs.

Complaint and history. - His gums have been painful and aching for one month. He consulted a neighbouring "sick quarters" 10 days ago where urine and blood tests showed the presence of sub-scurvy. Since then he has had 300 mgs. ascorbic acid per day. On admission here he is saturated with vit. C. For the past 10 days he has also had antiseptic gargles and local arsenic

paints (N.A.B. 7½ in glycerine). There has been no scaling or dental treatment. He feels no better and his gums have not ceased to bleed for a month. No cough. Slight "indigestion" for 3 to 4 years, worsely lately. Bad taste in mouth in mornings. No myctalopia, diarrhoea nor aches and pains in his limbs. He does not smoke but drinks beer in moderation. Typical ulcer in L. retro molar sulcus.

O.E. Gums red and bleeding and covered with debris. Teeth very foul and covered with tartar. Yellowish sloughs at teeth bases on the gums. A large carious molar is present on L. side. Breath offensive. Toungue furred and moist but no glossitis. Tender adenitis on L. side. Tonsils and fauces clear. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D. Previous local treatment has been of no avail.

Course. Day 1. Thorough scaling, rotary brushing with pumice stone, spraying with Lusol (1 in 4) and daily painting with 10% acid chrom. .45 gm. N.A.B. given intravenously. Carbolised dressing to L. carious molar.

Day 4. Teeth clean. Continue daily gum treatment. Carbolised dressing replaced as it had come out.

Day 6. Gums firmer. Ulcer behind L. molar healing.

Day 8. Less membrane. Gums paler and more firm. No bleeding. Yellow ulcers at teeth bases almost all healed. Interdental papillae shrunken.

Day 10. Fit for discharge.

Diet history. - Breakfast - Bacon and fried tinned tomatoes. Sausage and egg or fried fish or sardines. Kippers, bread, butter and jam. No fruit whatever on the ship.- Dinner. - Roast beef, mutton. Tinned peas, potatoes or greens. Rice or currant puddings occasionally. Tea - Tea, fish or cheese or ham or bacon, etc. Supper - None at night except cheese or corn-beef and tea before retiring.

Case 19. W.H. 32 years. Hutments on shore for 14 months.

On admission. Temp. 98.2°. Pulse 70/min. Bowels open once a day. Tourniquet test negative. Urine - S. G. 1025. Acid. Bile, blood, albumen and sugar - nil. Gum swab positive.

Blood count. R.B.C. 4,360,000/c.mm. Hb. 95%. C.I. 1.1. W.B.C. 9,800/c.mm. Polymorphs. 78%. Lymphocytes 20%. Monocytes 2%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation - First spec. 2.2 mg.%; 3 hours spec. 2.34 mg.%; 6 hrs. spec. 2.65 mg.%. (Vit. C. deficiency).

Nicotinic acid excreted per day equal to 2.87 mgs.

Complaint and history.- Gums tender and prone to bleed for 3 weeks. No diarrhoea. Appetite good and could eat anything. C/o pain in upper and lower jaws on R. side behind central incissors for 5 days. No bad taste in mouth. No "indigestion", rheumatism, eye trouble, nor nyctalopia. Has had slight bronchitis lately. Non-drinker and smokes 15 cigarettes per day. He states that he brushes his teeth regularly every day. No previous illnesses except hernia.

O.E. Thin patient with sunken cheeks. Typical Vincent's Disease of gums. Yellowish white sloughs at base of teeth from R. incissor to R. bicuspid teeth above and below. Gums swollen and easy to make bleed. No tartar at base of teeth, but brown staining at teeth bases. Whitish membrane outer margin of gums and in gingivo-labial furrow. Tongue clean - no glossitis. Shotty glands R. and L. side of neck. Loose R. incissor tooth - tender on pressure. Breath heavy. Chest - evidence of slight bronchitis. Scattered rales in all areas. Heart, abdomen, C.N.S. and eyes - N.A.D.

Course. Day 1. Gums painted once with Eusol. Nicotinic acid 250 gr. per day given i.e. 1 tablet 5 times a day. (At first he took 2 tabs. nicotinic acid at once and he had a severe flushing of his face and cheeks.)

Day 7. No change in condition. Nicotinic acid and Eusol m.w. continued.

Day 11. Teeth and gums show no change, with nicotinic acid and Eusol. Gums still bleeding even on rubbing with cotton wool. Routine scaling, spraying, brushing, etc., of teeth. Typical false membrane stretching from fraenum to R. canine of upper jaw. Hollow tooth stopped with carbolised dressing. Bowmont's arsenical soltn. painted on daily + daily Eusol spray.

Day 14. Less membrane on gums in the mornings. Still tender.

Day 17. Gums firmer. Less pain and bleeding on manipulation. Continue daily arsenical solution application.

Day 20. Mouth appears normal. No membrane visible.

Day. 21. Discharged.

Diet history. - Breakfast - Bacon and eggs or sausages or liver. Herring, bread, margarine and tea. The only fruit he has had is cooked prunes. The last fresh fruit was in Palestine in January 1939. Dinner - Stew, fried steak, cottage pie, potatoes. No vegetables other than potatoes. Prunes or custard, rice, no tinned fruit. Tea - Bread, butter, jam and cheese. Supper - Cocoa, soup, bread, butter and jam.

Case 20. J.A. 26 years. Trawler sweeper 8 months.

On admission. Temp. 101.6°. Pulse 96/min. Bowels not open. Tourniquet test negative. Urine S.G. 1025. Faintly acid. Faint trace of albumen. Bile, blood, sugar - nil. Gum swab positive. Blood count. R.B.C. 4,200,000 /c.mm. Hb. 86%. C.I. 1.02. W.B.C. 9,500/c.mm. Polymorphs. 72%. Lymphocytes. 24%. Monocytes 4%. Eosinophils 0%. Basophils 0%.

Urinary vitamin C. estimation. First spec. 2.78 mg.%. 3 hrs. spec. 1.43 mg. %. 6 hrs. spec. 3.4 mg. %. (Vit.C. Deficiency.)

Nicotinic acid estimation not done.

Complaint and history.- Patient had a head injury and concussion one month ago. Has suffered from headache since then and a swelling appeared in R. side of neck under upper third of the sterno-mastoid muscle. He always feels "giddy". His gums bleed easily. There is a bad taste in his mouth in the mornings. His gum condition has lasted for 3 weeks. He smokes and drinks alcohol. No bronchitis, nor diarrhoea, nor aches and pains. No trouble with his eyes or stomach.

O.E. Gums very foul and breath bad. Whitish film of Vincent's Disease on gums and hard palate. Gums swollen with prominent interdental papillae and ulcers at teeth bases. Tonsils free. Teeth filthy. Many decayed stumps worn down to gum level. Otherwise he is a well nourished patient. Heart, lungs, C.N.S. abdomen and eyes - N.A.D. No external evidence of cranial injury. Soft, cystic, painless swelling which does not appear to be inflammatory at anterior border of R. sterno-mastoid, at level of angle of jaw. There appeared to be no connection between the injury and the cyst. His pyrexia could be accounted for by the dirty condition of his mouth.

No pain, deformity, muscle spasm or limitation of movement of neck. X-ray shows no Pott's disease.

15 c.c. of yellowish green, slightly cloudy fluid were taken from cyst. No cholesterol crystals were found and fluid was sterile. Diagnosis of a bronchial cyst was made.

Course. Day 1. - Routine scaling, etc., and daily painting with chromic acid 10%. Eusol m.w. (1 in 4) three or four times a day.

N.A.B. - 0.45 gm. intravenously. Mist. alba one oz. given.
Day 4. - Gums still bleed on pressure. Less membrane in mouth. Temp. normal. Stumps removed under gas. Sockets kept clean with Eusol m.w.
Day 6. - Gums less red and becoming firmer. Still slight L. tender adenitis.
Day 8. - Interdental papillae shrinking. Teeth sockets keeping clean. Adenitis not tender.
Day 10. - Disease rapidly healing. Only a very few eroded areas remain on the gums. Daily 10% chrom. acid continued.
Day 12. - Fit for discharge.
Diet. - Similar to case 1. He has had no fresh fruit for several months and his green vegetables are overcooked. The milk is tinned.

Case 21. W.C. 27 years. Trawler for 3 months and in a Drifter for 6 months before that.

On Admission. Temp. 98.2°. Pulse 78/min. Bowels not open. Tourniquet test - negative. Gum swab positive. Urine - Pale, straw colour. S.G. 1028. Faintly acid. No bile, blood, sugar or albumen. Blood count. R.B.C. 3,560,000/c.mm. Hb. 64%. C.I. 0.91. W.B.C. 5,400/c.mm. Polymorphs. 55%. Lymphocytes 38%. Monocytes 5%. Eosinophils 2%. Basophils 0%.
Urinary vitamin C. estimation. First spec. 2.7mg.%. 3 hrs. spec. 2.04 mg.%. 6 hrs. spec. 3.22 mg.% (Vitamin C. deficiency). Nicotinic acid excreted per dayequal to 5 mg.
Complaint and history. 5 days ago he noticed that his gums were aching and swollen. Consulted his doctor on day 2. Gums do not bleed. He does not brush his teeth regularly. Bad taste in mouth and heavy breath. Appetite is good. No stomach trouble. Non smoker, but drinks. No "wheezing" in chest, but has had a cough for a week. No diarrhoea nor "rheumatism". He says he feels fit.
O.E. Tall, thin patient with pale mucosae. His colour is poor. Tender adenitis R. side of neck. Shotty glands L.. Healthy scar of former operation for tubercular glands on R. side of neck.

Some caries of upper central incisors and upper back L. molar. The remaining teeth show tartar and brown staining at the roots. Tongue furred - no glossitis. Tonsils free. Vincent's infection of gums - ulcers upper and lower molar regions on R. side. Chest - a few scattered post-tussive rales are audible. Heart, abdomen, C.N.S. and eyes - nil.

Course. Day 1. - Routine scaling etc. Daily treatment with 10% chrom. acid begun. Full diet. M.W. Eusol (1 in 4) t.i.d.. Mist. Ferri et ammon. cit. one oz. t.d.s.

Temporary carbolised dressing to molar.

Day 2. 0.3 gram. N.A.B. intravenously.

Day 5. No tenderness in gums. Still ulceration and some white membrane on outer side gums and in labio-gingival furrows.

Day 7. Gums much firmer and less red. Improving.

Day 10. Discharged.

Diet.- Breakfast - Tinned sausages and beans, or bacon and eggs. Bread, butter and tea. No fruit. Dinner - Stew, potatoes, plus carrots or turnips. No greens. Roast occasionally. Plum pudding or rice which the patient never takes. No tinned fruit. Tea - Sausages or corned meat or ham and eggs. Bread, butter and tea. Supper - Cake and tea. Cheese for the first time a week ago. There are 16 men in the mess and the mate tells one of them at random to go to the N.A.A.F.I. canteen for victuals and to the victualling stores for meat, etc.

This man buys what he can get and often no vegetables are available.

Case 22. W.M. 23 years. Trawler (B) for 10 months.

On admission. Temp 97.6°. Pulse 72/min. Bowels open. Tourniquet test negative. Urine - S.G. 1016. Alkaline. No bile, blood, sugar or albumen. Gum swab positive.

Blood count. R.B.C. 4,900,000/c.mm. Hb. 86%. C.I. 0.88. W.B.C. 16,000/c.mm. Polymorphs. 80%. Lymphocytes 17%. Monocytes 3%. Eosinophils 0%. Basophils 0%. Urinary vitamin C. estimation - First spec. 3.12 mg.%. 3 hrs. spec. 3.3 mg.%. 6 hrs. spec. 3.7 mg.%. (Vitamin C. deficiency).

No nicotinic acid estimation done.

Complaint and history. - Aching started in the gums 12 days ago. Lately he has noticed that the fronts of his gums bleed on brushing the teeth. Bad taste in mornings from gums. No aches in body, and no "stomach" troubles. Is prone to take chest colds. Bowels are regular once a day. Brushes his teeth normally but lately has not done so owing to tenderness and aching. Smokes 20 cigs. a day and drinks beer in moderation. No trouble with his eyes or eyesight lately.

O.E. No gross caries of teeth. Some tartar at teeth bases and deep brown staining of the molar roots. Gums red and swollen and the interdental papillae prominent. Whitish film of Vincent's disease on outer aspect of gums, and an ulcer with a white slough is present in the R. retro-mular sulcus. Erosions of palate behind upper central incisors. Tonsils clear. Tender cervical glands. on right side. Shotty, non-tender glands on left side. Tongue is furred and breath heavy, but no glossitis exists. Pale mucosae. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.

Course.- Day 1. Routine scaling, rotary brushing, spraying with Eusol (1 in 4) and daily painting with 10% chrom. acid. 0.45 gm. N.A.B. intravenously.

Day 4. Complains of sore throat, ear-ache and pain in the neck. Temp. 99°. Slight cough.

O.E. Redness of soft palate and R. side of fauces. Lungs - N.A.D. A superimposed tonsillitis.

Garg. ferri per chlor. t.d.s. + Mist. pectoralis 1 oz. t.d.s.

5% phenol in glycerine drops to R. ear.

Day 7. Fauces less red. Slight ear-ache last night. No pyrexia.

Day 8. No ear-ache. Throat improved. Gums greatly improved and are not tender. More firm and less inclined to bleed. No membrane visible. Ulcer in R. retro-molar sulcus in the last stage of healing.

Day 10. Ready for discharge.

Diet. - Breakfast. Bread, butter, tea, bacon and eggs. Dinner - Roast pork (on Sundays), roast meat. roast or boiled potatoes or meat pie. Cooked tinned carrots or soup and heated tinned tomatoes. Bread or rice pudding. Tea.- Bread, butter, jam and tea. Supper -

Something fried every night. Liver and tinned tomatoes or fish or bacon and beans. Bread, butter and tea. He eats much nut chocolate between meals.

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Case 23. C.G. 28 years. Drifter for 9 months.
On admission. Temp 98.2°. Pulse 78/min. Bowels not open. Tourniquet test negative. Urine - dark straw colour, S.G. 1028. Acid . No bile, blood, sugar or albumen. Blood count not done. Urinary vit. C. estimation. First spec. 2.13 mg.%. 3 hrs. spec. 2.09 mg.%. 6 hrs. spec. 2.37 mg. % (Vitamin C. deficiency). Nicotinic acid excreted per day - 6.38 mg.
Complaint and history. - Troubled with gums for 6 weeks. He had a molar out then and has had pain in the region since. A week ago he had 2 more upper right molars removed for toothache and the pain never cleared up. His gums bleed on brushing the teeth. He has for 3 weeks had a bad taste in his mouth and a bad breath. His appetite is poor and he suffers from bronchitis (2 weeks). There are no general aches and pains and no stomach trouble. Smokes 20 cigs. per day and does not drink. He does not suffer from night blindness nor eye trouble.
O.E. No clinical evidence of anaemia. Tonsils clear. Some tartar and caries of teeth. Severe Vincent's infection of gums with membrane all over gums and in gingivo-labial furrow. Ulcer behind lower R. molars. Breath very heavy. Gums red and swollen and bleed easily. Slight non-tender shotty adenitis on both sides of neck. Heart, abdomen, C.N.S. and eyes N.A.D. Lungs - scattered rales and evidence of slight bronchitis.
Course. - Day 1. Scale, rotary brushing using pumice stone dust and Eusol spray. Daily painting of gums with 10% chrom. acid. M.W. Eusol (1 in 4) 4 i.d. Full diet. One No.9 pill.
Day 2. 0.5 gm. N.A.B. given + daily m.w. and chrom. acid.
Day 5. No pain in gums. Still swelling and bleeding on pressure. Some membrane persists in the mornings. Daily treatment continued.
Day 7. Gums much firmer and less red. Inerdental papillae shrinking. Ulcer in retro-molar sulcus has healed. Chest clear.
Day 9. Great improvement all over.

Day 10. Ready for discharge.

Diet history.- He has had no fresh fruit for 9 months. Breakfast - Egg and bacon, liver or steak, or tinned sausage or fish rarely. Bread, butter ++ and tea. Dinner - Meat and potatoes or stew. The only vegetables given are turnips or cabbages, the latter rarely. Sweet - either plum pudding or custard. Tea - Meat hash (hot or cold), soups, pie with meat and potatoes. Tea, bread, butter and jam.
Before turning in - toast and dripping, or cheese and tea.

Case 24. E.L. 26 years. Paddle minesweeper (Z) for 9½ months.
On admission. Temp. 98.2°. Pulse 84/ min. Bowels open once a day. Tourniquet test negative. Urine - clear. S.G. 1017. Acid. No bile, blood, sugar or albumen. Gum swab positive.
Blood count. R.B.C. 4,280,000/c.mm. Hb. 79%. C.I. 0.94. W.B.C. 11,000/c.mm. Polymorphs. 64%. Lymphocytes 29%. Monocytes 5%. Eosinophils 1½%. Basophils ½%.
Urinary vit. C. estimation. First spec. 5.55 mg.%. 3 hrs. spec. 16.67 mg.%. 6 hrs. spec. 25.0 mg.%. (No vit. C. deficiency now.)
Nicotinic acid excreted per day equal to 4.75 mg.
Complaint and history. - He noticed that his gums were sore when he was on leave 2½ months ago but did not bother about it. Two weeks later he consulted a private dentist who pulled 4 carious teeth and applied chrom. acid only. He had no scaling or other local treatment. For the last three weeks but one he has been treated with ascorbic acid. (3 tabs. 4-hourly) and has had nothing else. Prior to this, blood and urine tests showed a deficiency of ascorbic acid. During the last week he has had ascorbic acid, gargles and arsenic paints only. He still complains of pain and some bleeding of the gums. He has no chest nor stomach trouble. No diarrhoea, and no aches and pains. His appetite is good, though he still has a bad taste in his mouth. He neither smokes nor drinks.
O.E. Gums bleeding, tender and swollen. Whitish film on outer aspects, especially in upper gum. Large ulcer with white membrane on it, in the L. mandibular region. Tongue furred but no

glossitis. Tonsils clear. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D. Shotty adenitis R. and L. Teeth show old standing staining, and tartar at the bases.

Course. Day 1. 0.3 gm. N.A.B. intravenously. Routine scaling, brushing, Eusol spraying and daily painting with 10% chrom. acid. Eusol m.w. (1 in 4) 4 i.d..

Day 3. No pain. Still white membrane and bleeding.

Day 5. Gums less intensely red and somewhat firmer. Retro-mular ulcer healing. Continue daily treatment.

Day 8. Ulceration almost all gone. Gums firm and whiter. No bleeding.

Day 10. Ready for discharge to duty.

Diet.- Breakfast- Eggs and bacon, or liver and tinned sausages, bread, butter, tea. No fruit whatever in the diet. Dinner - Meat and potatoes, tinned peas, greens on rare occasions as these were not always available in the canteen. Rice, and plum duff. Tea - Bread, butter, tea, liver or tinned fish. Supper - Cheese, bread, butter and tea.

This was the only patient from the mess. All cooking utensils were washed in a pail and the sailors did not have their own cups, etc. kept apart. There is the possibility of a carrier here. The treatment he had prior to admission had been of no avail without the thorough removal of tartar and debris.

Case 25. - G.H.F. 27 years. French destroyer for 4 months and R.N.B. for 4 months previously and destroyer 20 months before that.

On admission. Temp. 97.4°. Pulse 82/ min. Bowels not open regularly. Tourniquet test positive. Urine - S.G. 1022. Acid. Faint trace of albumen. No bile, blood or sugar. Throat and gum swabs positive.

Blood count. R.B.C. 4,480,000/c.mm. Hb. 90%. C.I. 1.02. W.B.C. 12,600/c.mm. Polymorphs 75%. Lymphocytes 19%. Monocytes 6%. Eosinophils 0%. Basophils 0%. Urinary vit. C. estimation - First spec. 2.12 mg. %. 3 hrs. spec. 2.08 mg. %. 6 hrs. spec. 2.7 mg. %. (Vitamin C deficiency).

No nicotinic acid estimation done.

Complaint and history.- History of several attacks of Vincent's infection of the throat. 1. Attack in Jan. 1938 lasted 10 days, treated with N.A.B. injections in Haslar. 2. Mild attack in Jan. 1939, lasted 5 days. Treated with throat paints and "tablets to suck" in Haslar. 3. Attack in 4th November, 1940. Lasted 9 days. Treated with N.A.B. injections + throat paint in Haslar. This is his 4th attack and he has pain of a week's duration in the L. side of his throat. His gums are bleeding on pressure too, and are tender. This throat trouble is the only illness from which he suffers. He has had mild sore throats on occasion between the attacks but not severe enough to make him consult the M.O. He has no chest trouble and no nyctalopia. No aches or pains in body or limbs. No nasal trouble and can eat anything. He smokes and drinks in moderation. His teeth bleed easily, and did so also while he was on his first destroyer in home waters.

O.E. Marked retraction of the gums. There is some pyorrhoea as well as marked Vincent's disease of the gums. No actual ulcers but membrane all over. This long standing gum trouble has probably been reinfecting his tonsils and mouth. (Taylor and McKinstry (23)). L. tonsil swollen and has a greyish white membrane on its upper pole and there is a characteristic smell from the case. R. tonsil is small and granular. There is a swollen, tender cervical gland on the L. side and shotty R. adenitis. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.. Teeth show caries and tartar at bases.

Course. Day 1. Routine scaling, brushing etc. Daily paints with 10% chrom. acid. M.w. Eusol (1 in 4) 4. i.d.

Day 2. 0.45 gm. N.A.B. intravenously + usual daily treatment.

Day 5. Fauces feels better and there is no pain in the gums. Now able to take a full diet. Ulcer and membrane rapidly vanishing from tonsil.

Day 7. Throat almost clear. Continue Eusol m.w. 4 i.d. and chrom. acid daily.

Day 8. No spots on L. tonsil. Less pain in neck glands. Gums are firm, less red but still show some membrane in the upper jaw

in the labio-gingival furrow.

Day 10. Gums rapidly healing. No bleeding or pain.

Day 12. Almost better.

Day 13. Discharged to duty.

Diet. - The system is that of canteen messing. Each mess buys its own food from the canteen and each man subscribes. There are 24 men in the mess and each sailor is allowed 1s.7d. per man to feed. They usually save 10s. per head per month.

Breakfast - Tea, bread or rolls, butter and jam, ham and eggs on Sundays. Dinner - Described by him as "good" - potatoes, meat, greens, peas or runner beans. There is never fruit at any time. Each man takes his turn at being the cook and as regards puddings, to use the rating's own words, "If a fellow was good enough to make a duff, there was a duff". All vegetables are tinned and heated up, even the cabbages.

Tea - Bread, butter, jam and tea. Supper - Sausages or liver, bacon, potatoes and tea.

Case 26.

G.T. 17 years. Destroyer (A) for 17 months.

In a different mess from cases 17 and 55.

On admission. Temp.98°. Pulse 84/min. Bowels regular once a day. Tourniquet test negative. Urine, S.G. 1015. Acid. Albumen - very faint trace. Sugar, bile and blood - nil. Throat swab positive, gum swab negative.

Blood count. R.B.C. 5,150,000/c.mm. Hb. 97%. C.I. 0.95. W.B.C. 8,800/c.mm. Polymorphs 71%. Lymphocytes 25%. Monocytes 4%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. First spec. 1.39 mg. %. 3 hrs. spec. 4 mg.%. 6 hrs. spec. 10 mg. %. (No vitamin C. deficiency). No nicotinic acid estimation done.

Complaint and history. - Complains of pain in region of throat for 4 or 5 days. He had a T.A.B. inoculation (.5 c.c.) 2 days ago and suffered from malaise after that but he had felt a sore throat before this. He has a bad headache. His gums are healthy if he brushes his teeth every day but they have bled on occasion for a year if he omits to clean them for 2 or 3 days. This is his first sore throat. No rheumatic pains, chest trouble, nyctalopia,

He is a non-smoker but drinks cider and whisky.

O.E. Good colour and no clinical evidence of anaemia. Gums are very slightly swollen at sides of upper jaw on the outer aspects but they are not ulcerated and have no whitish film on them. No tartar or caries of teeth. Shotty non-tender adenitis on right side only. Fauces inflamed. R. tonsil shows a small ulcer with a white membrane. No glossitis. Heart, lungs, abdomen, eyes and C.N.S. - N.A. D.

Course. Day 1. 1 garg. pot. chlor. 4 i.d. 2 aspirin grain 15 morning and evening. 3. thermogene to neck. Bed and low diet.

Day 2. 0.3 of a gm. intravenously + 1, 2 and 3 above.

Day 3. No soreness in throat.

Day 4. Full diet.

Day 5. No redness of fauces. Ulcer rapidly getting smaller on tonsil.

Day 8. Discharged.

Diet history. Canteen messing but there are three regular cooks. Breakfast - Coffee, bread and butter ++. Bacon, eggs or fish. Dinner - Roast potatoes, meat, and one vegetable (cabbage and tinned carrots, turnips or peas). Puddings rarely. They got fruit (tinned and oranges) regularly till 2 months ago then it became scarce and he has had none since. Tea - Bread, butter, jam or toast. Supper - Same as breakfast, or bacon and eggs, bacon and liver, or bacon and mashed potatoes. He eats much nut chocolate every day between meals. This is a diet which with the addition of a little fruit as he has had up till 2 months ago is very full. Without the fruit and under the cooking conditions of a destroyer the vit. C. is lacking.

Case 27. G.K. 26 years. Destroyer 4½ months.

On admission. Temp 99°. Pulse 84/min. Bowels not open. Tourniquet test negative. Urine. S.G. 1020. Acid. No albumen, bile, blood or sugar. Throat and gum swabs positive.

Blood count. R.B.C. 4,500,000/c.mm. Hb. 99%. C.I. 1.1. W.B.C. 6,800/c.mm. polymorphs 75%. Lymphocytes 22%. Monocytes 2%. Eosinophils 1%. Basophils 0%. Urinary vit. C. estimation.

First spec. 2.43 mg.%. 3 hrs. spec. 2.1 mg.%. 6 hrs. spec. 3.33 mg.%. (Vit. C. deficiency).

No nicotinic acid estimation was done. W.R. - negative.

Complaint and history.- C/o pains in his throat, ears and gums. His gums have pained him for 2 months. He had his two front incisors removed two weeks ago and the gums still ache. He has had a sore throat for 10 days. No history of syphilis. He has no diarrhoea nor chest trouble nor vague pains. No nyctalopia. Lately he has had "indigestion" and consulted the sick berth attendant one month ago. Pain in epigastrium after meals; relieved by alkalis; and ascending flatulence. He is not a heavy drinker. He smokes ½ lb. tobacco per month.

O.E. There is a large slough on the left tonsil. Tender, tonsillar adenitis R. and L. The gums show the appearance of Vincent's infection i.e. swollen, tender, red and bleeding. Yellow ulcers at canine bases and whitish membrane. Breath has the characteristic heavy odour. Teeth have much tartar at bases and are close set and some are loose. The sockets of the 2 lower central incisors are infected. No caries in remainder. Ears - N.A.D. Heart, Lungs, C.N. S. and eyes - N.A.D. Abdomen, - slight guarding on palpation over R. upper rectus. No pain.

Course. Day 1. .3 gm. N.A.B. intravenously. Routine scaling, brushing, etc., and daily gum paints with 10% acid chrom. G.A.C.

Day 2. Throat feels slightly better. Daily treatment + m.w. of pot. permang. and G.A.C. to the left ear.

Day 4. Ulcer on tonsil beginning to clear. Gums I.S.Q.

Day 6. Tonsil almost clear. Gums less red and firmer. Still aching in gums. Continue chromic acid daily and m.w.

Day 8. Tonsils appear normal. No dysphagia.

Day 10. Teeth firm and ulcers at bases clear.

No bleeding or pain in gums. Some membrane in incisor sockets in the morning. Daily treatment + Eusol spray to incisor sockets.

Day 12. Fit for discharge to duty.

Diet. - Canteen Messing. Breakfast - Tea, bread and butter ++. Bacon and eggs. Dinner - Meat, potatoes and vegetable (cabbages, tinned beans or peas). Sweets when the cook can make it and usually then either suet or currant puddings. Tinned fruit and custard on

Sundays. Tea - Bread, butter and jam. Supper - Tinned sausages. Fresh fish or kippers, potatoes and fried tinned tomatoes. Tea, bread and butter.

Case 28. G.L. 29 years. Trawler for 8 months.
On admission. - Temp. 98.2°. Pulse 70/min. Bowels open but no diarrhoea. Tourniquet test negative. Urine. S.G. 1024. Alkaline. No bile, blood, sugar or albumen. Gum swab "positive".
Blood count. - R.B.C. 4,700,000/c.mm. Hb. 96%. C.I. 1.02. W.B.C. 11,000/c.mm. Polymorphs 73%. Lymphocytes 25%. Monocytes 2%. Eosinophils 0%. Basophils 0%.
Urinary vit. C. estimation. First spec. 2.48 mg%. 3 hrs. spec. 3.16 mg. %. 6. hrs. spec. 3.27 mg. % (Vitamin C. deficiency.)
No nicotinic acid estimation done.
Complaint and history. - Has noticed that for the past week his gums have been bleeding on brushing the teeth. No pain. Has had a heavy breath and taste in his mouth lately. No diarrhoea, no stomach trouble, aches and pains nor trouble with his eyes. He smokes 20 cigs/day but is a non-drinker.
O.E. Well nourished patient. Mucosae have good colour. Tonsils and neck glands - normal. Gums show some ulceration above on L. side from median labio-gingival raphé to molar region. Vincent's membrane is present there. Swollen interdental papillae. Large L. upper carious molar. Some tartar at teeth bases overlapped by the swollen gums. Heart, lungs, abdomen and C.N.S. - N.A.D. No keratomalacia, and no glossitis.
Course. Day 1. Routine scaling, brushing and spraying of teeth with Eusol 1 in 4. Daily painting of gums with 10% chromic acid and m.w. Eusol (1 in 4) 4 i.d. .45 gm. N.A.B. intravenously. Carbolised dressing to carious molar.
Day 3. Gums firmer. Less membrane on L. upper jaw in the morning.
Day 5. Swab is "negative" in the morning. Erosions on gums are healed and gums are firmer and are not prone to bleed in any area. Discharged to duty.
Diet history. - Same as case 15.

Case 29. J.W. 31 years. Drifter for 6 months.
On admission. - Temp. 98°. Pulse 82/min. Bowels open once a day. Tourniquet test negative. Urine. S.G. 1028. Faintly acid. Bile, blood, sugar and albumen absent. Gum swab "positive". Blood count. R.B.C. 4,800,000/c.mm. Hb. 100%. C.I. 1.04. W.B.C. 10,200/c.mm. Polymorphs. 59%. Lymphocytes 34%. Monocytes 6%. Eosinophils 0%. Basophils 1%. Urinary vit. C estimation:- First spec. 1.66 mg.%. 3 hrs. spec. 2.3 mg. %. 6 hrs. spec. 3.33 mg.%. (Vit. C. deficiency).
No nicotinic acid estimation done.
Complaint and history. - C/o sore gums for the past 4 weeks but pain much worse in the last 3 or 4 days. The gums are bleeding slightly on pressure. Bad taste in the morning in his mouth. No eye trouble nor nyctalopia. No "rheumatism" nor diarrhoea. His throat does not trouble him. He suffers from slight bronchitis. Smokes ½ lb. tobacco per month and drinks in moderation.
O.E. Well nourished patient with a ruddy complexion. Gums are red, tender and swollen with sloughing margins and ulcers with yellowish sloughs at the teeth bases. Whitish membrane on outer aspects. Tonsils clear. Breath has the heavy, characteristic odour. Tongue furred and no glossitis. Caries of one lower R. molar. Some tartar. Large tender, right cervical adenitis. Lungs - R.M. vesicular with a few scattered, post-tussive rales. Heart, abdomen, C.N.S. and eyes - N.A.D.
Course. Day 1. Routine scaling, brushing and spraying of teeth. Gums bled after this. Daily painting with 10% chromic acid and Eusol 1 in 4 m.w. 4 i.d. Mist. Expect. 3i t.d.s.,2 vegetable laxative pills.
Day 4. Gums still sore and red. Continue daily treatment.
Day 7. Ulcerated areas at bases of teeth clearing. Less membrane in the mornings on outer aspects of gums. Breath still offensive. Chest clear.
Day 9. Gums have lost the fiery red colour they had previously. Interdental papillae back to normal size. Some erosion on outer aspect of R. side of upper jaw. Eusol spray + chromic acid.
Day 11. Condition almost clear. No adenitis. Tongue clean.

Day 13. Discharged to duty.

Diet history.- Breakfast - Porridge twice a week. Ham, bacon and eggs nearly every morning. Bread, butter ++, and tea. Dinner - Usually roast meat. Potatoes. Cabbage, turnips and carrots (all fresh). Custard and occasionally tinned fruit. He cannot remember when he last saw an orange. He averages 2 apples a week. Tea - Bacon and egg, plus tea, bread, butter, jam, marmalade or treacle. Supper - Bread and cheese, onions and cheese.

This is a good diet as regards variety and content of fresh vegetables. The food is all liable to be overcooked however.

Case. 30. S.J. 24 years. Drifter for 3 months and then on survivor's leave for 2 weeks, and drifter all his life previously. On admission. Temp. 99.4°. Pulse 80/min. Bowels open. Tourniquet test positive. Urine S.G. 1018. Acid. No bile, blood, sugar or albumen. Gum swab "positive".

Blood count. R.B.C. 3,860,000/c.mm. Hb. 68%. C.I. .89. W.B.C. 13,400/c.mm. Polymorphs. 78%. Lymphocytes 19%. Monocytes 3%, Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. First spec. 2.0 mg.%. 3 hrs. spec. 2.6 mg.%. 6 hrs. spec. 1.42 mg.%. (Vitamin C. deficiency).

No nicotinic acid estimation done.

Complaint and history. He has had pain in the gums for 4 weeks. Gums bleed a lot. Bad taste in mouth and heavy breath. No diarrhoea, rheumatism, night blindness, nor chest, nor stomach trouble. He does not brush his teeth regularly. He has never had sore throats nor tenderness in the neck. He smokes heavily and does not drink alcohol.

O.E. Tartar at teeth bases, overlapped by swollen gums, and brown staining. Only one small spot of caries on the upper central incisors. Tonsils clear. Gums swollen, bleed easily and ulcerated at bases of teeth, Ulcer and white slough in R. retro-molar sulcus. No glossitis. Heart, lungs, abdomen, eyes and C.N.S. - N.A.D. Shotty, non-tender adenitis R. and L..

Course. Day 1.- Thorough routine scaling, rotary brushing with pumice dust and Eusol spraying. M.W. Eusol 1 in 4 four times a day

and daily gum paints with 10% chromic acid.

Day 3. Gums I.S.Q. Breath still heavy.

Day 6. Less membrane on gums. Interdental papillae less red and shrinking. Ulcer in retro-molar sulcus clean and healing.

Day 9. No bleeding on pressure. Redness gone from gums but some white filmy membrane remains. Retro-molar sulcus now healed.

Day 11. Only one small ulcerated area at base of R. lower bicuspid.

Day 14. Fit for discharge.

Diet history.- Breakfast - Bacon and eggs or tinned sausages. Bread, margarine, jam and tea. Porridge 3 times a week. Dinner - Soup. Stew or roast. Potatoes, cabbages, turnips, carrots. "Duffs" or rice every day. Tinned pears but no fresh fruit. Tea - Tinned salmon or tinned sausages. Bully beef, eggs and bacon. Tea,bread, butter, jam. Fresh fish once a week. Supper. Cheese, bread, pickles and tea.

Case 31. S.R. 21 years. Sloop for 4½ months.

On admission. Temp. 97.2°. Pulse 56/min. Bowels open. Tourniquet test negative. Urine. S.G. 1024. Acid. No albumen, bile, blood or sugar. Gum swab "positive".

Blood count. R.B.C. 4,920,000/c.mm. Hb. 94%. C.I. .95.

W.B.C. 10,200 /c.mm. Polymorphs. 80%. Lymphocytes 15%.

Monocytes 4%. Eosinophils 1%. Basophils 0%.

Urinary vit. c. estimation. First spec. 2.06 mg.%. 3 hrs. spec. 2.35 mg.%. 6 hrs. spec. 2.28 mg. %. (Vit. C. deficiency).

No nicotinic acid estimation done.

Complaint and history.- His chief complaint was that he was troubled with sea-sickness habitually and lately had noticed blood in the vomit. Had epigastric pain 4 days before admission - colic in type. No vomiting when on shore and no nausea then. His bowels are regular and he has neither diarrhoea nor constipation. For the past 3 weeks he has had dirty, red blood in his mouth in the mornings. No fainting and no epistaxis. Appetite good till 4 days ago. No vague pains in his limbs and no eye troubles. Has "wheezed" at night and had an irritating morning cough for 2 weeks. He is a non-smoker, but drinks heavily while on shore.

O.E. He is a rather thin, pale subject. Lungs- scattered rales. Heart, C.N.S. abdomen and eyes - N.A.D. Tonsils clear. Tender R. cervical adenitis. No glossitis. There is much tartar and severe caries of the teeth. The gums are very red, swollen and spongy and bleed easily. White membrane all over and ulcer in R. lower retro-molar sulcus. Breath foul.

Course. Day 1. - Routine scaling, spraying, etc. Daily paints with 10% acid chrom. H_2O_2 m.w. Bed.

Day 3. No pain and no cough. Gums I.S.Q. Ascorbic acid. tabs 2. t.i.d.

Day 5. Slight abdominal pain. Mist. Gent. with rhei. 3ʃ t.d.s. Continue chromic acid daily and H_2O_2 mouth washes.

Day 6. Abdominal pain gone. H_2O_2 discontinued as gums are tender with it. Eusol instead (1 in 4).

Day 7. Still some tenderness in gums. Carious stumps removed under gas.

Day 9. Gums are firmer and interdental papillae more shrunken. No ulcers now.

Day 10. No pain in gums nor neck. Chest clear.

Day 12. Discharged to duty.

Diet history. His meals vary according to the trip he is on. e.g. South bound Convoy (6 days).

Breakfast - Bread, butter ++, marmalade and tea. Dinner - Roast or stewed meat. Roast or steamed potatoes and fresh greens the first day. Tinned vegetables after day 1. Corned beef and potatoes for 3 days owing to being closed up at action stations. Rice pudding once per week. Tea - Bread, butter, jam and tea. Supper - Eggs or sausage and chips, or steak, liver and potatoes, or ham, bread and butter.

Atlantic trip(15 days).

Breakfast - Bread, butter, marmalade and syrup. Cup of tea. Nothing else hot. Dinner - Roast or stewed meat, roast or steamed potatoes. Fresh greens for first 2 days and tinned carrots, peas or beans after that. Tinned fruit and rice pudding once per week. Tea - Bread and butter, jam and syrup occasionally. Supper - Eggs, sausages or chips. Potatoes, steak or liver. Cornbeef pie, ham or fish when it can be obtained from a trawler.

Case 32. B.W. Trawler (B) for 8 months.

On admission. Temp. 102.8°. Pulse 98/min. Bowels not open. Tourniquet test negative. Urine. S.G. 1030. Acid. Trace of albumen. No bile, blood or sugar. Throat swab "positive". Culture negative for K.L.B. but positive for haemolytic streptococcus.

Blood count. R.B.C. 4,300,000/c.mm. Hb. 95%. C.I. 1.1. W.B.C. 9,600/c.mm. Polymorphs. 66%. Lymphocytes 32%. Monocytes 2%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. First spec. 2.95 mg.%. 3 hrs. spec. 3.07 mg.%. 6 hrs. spec. 3.58 mg.%. (Vit.C. deficiency).

No nicotinic acid estimation done.

Complaint and history.- Has had a sore throat for the last 24 hrs. Also some cough. No headache and vomiting. He slept badly last night. No pains elsewhere. He has felt easily tired lately and "run down" but has had no diarrhoea, stomach trouble, nor aches and pains. No trouble with his eyes. He smokes a pipe and is a non-drinker.

O.E. Lungs - scattered rales in all areas. Heart, abdomen, C.N.S. and eyes - N.A.D. Tonsils are both enlarged and spots of white exudate are present on each. Very marked inflammation of fauces (probably from coincident streptococcal infection). Teeth have some caries but no tartar. Gums are free of infection. No glossitis. R. and L. tender cervical adenitis.

Course. Day 1. - He has had M. and B. 693 on the day before admission (amount not stated).

A. M. and B. 693, ℞ four hourly (1) Gargle pot. chlor. t.d.s. (2) Thermogene to neck.

B. Dover's pulv. and aspirin aa gr x nocte.

Day 2. A. 1. 2. and B. continued.

Day 3. Fauces improving. Slept badly. A. - M.andB. 2, 4 hourly. 1. 2 and B..

Day 4. Ol. ric. + A. m 10. ℞ t.d.s. 1, 2 and B.

Day 5. Feeling much better. Throat less red but the exudate still present in same amount. Stop M.andB. Continue 1 and 2. .45 gm. N.A.B. given intravenously.

Day 6. No pain. Chest clear. Definite improvement in general condition.

Day 8. Filmy exudate remains on L. tonsil. Rest of throat clear.

Day 10. L. tonsil almost clear. One minute spot alone remains. Up 2 hrs. No adenitis.

Day 13. No complaints. Throat clear. Discharged to 7 days' sick leave.

Diet history. Same as case 22.

Temp. Chart.	M.	E.	Pulse.	M.	E.
Day 1.	102.8	100		98	90
Day 2.	99.4	99.6		85	80
Day 3.	97.4	98.2		70	76
Day 4 and on.	Normal		Remained steady thereafter.		

Case 33. M.C. Motor drifter for 10 months.

On admission. Temp. 98.4°. Pulse 76/min. Bowels open once a day. Tourniquet test negative. Urine. Clear. S.G. 1015. Very acid in reaction. No bile, blood, sugar or albumen.

Blood count. R.B.C. 4,860,000/c.mm. Hb. 98%. C.I. 1.02. W.B.C. 11,400/c.mm. Polymorphs 75%. Lymphocytes 20%. Monocytes 4½%. Eosinophils ½%. Basophils 0%.

Urinary vit. C. estimation. First spec. 20.0 mg.%. 3 hrs. spec. 56.0 mg.%. 6 hrs. spec. 83.0 mg.% (No deficiency on admission). No nicotinic acid estimation done.

Complaint and history. Developed true scurvy 2½ months ago. (Blood ascorbic acid equal to .26 mg.%. then and urinary ascorbic acid was low too). His gums showed Vincent's disease then. He had nyctalopia, profusely bleeding gums and some anaemia. Also epistaxis. Carious teeth were stopped then and he was put on ascorbic acid daily till the present time. No chest or stomach trouble and no diarrhoea. Slight rheumatism in the shoulders occasionally. He smokes and drinks beer and spirits in moderation.

O.E. Teeth show slight tartar and all caries is "stopped". Shotty, non-tender glands in neck R. and L. Gums swollen and bleeding. Whitish film all over but no frank ulceration. Tonsils clear. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.

Course. Day 1. Routine scaling, brushing and Eusol spraying and

daily paints with 10% chromic acid. Eusol (1 in 4) m.w. 4 i.d. .45 gm. N.A.B. intravenously.

Day 4. Gums I.S.Q.

Day 6. Gums less red and more shrunken. Slight membrane persists in the mornings. Breath less offensive.

Day 8. Gums almost free of membrane and normal in size. No bleeding and pain.

Day 10. Discharged to duty.

Diet. Same as case 23.

Case 34. W.S. 20 years. Shore establishment for 5 months. On admission. Temp. 101°. Pulse 80/min. Bowels open but no diarrhoea. Tourniquet test negative. Urine S.G. 1028. Acid. No bile, blood, sugar or albumen. Throat swab "positive". No K.L.B. on smear or culture.

Blood count. R.B.C. 4,700,000. Hb. 92%. C.I. .98. W.B.C. 5,000/c.mm. Polymorphs 40%. Lymphocytes 53%. Monocytes 6%. Eosinophils 1%. Basophils 0%.

Urinary vit. C. estimation. First spec. 5.31 mg.%. 3. hrs. spec. 8.92 mg.%. 6 hrs. spec. 10.46 mg.%. (No vit. C. deficiency).

No nicotinic acid estimation done.

Complaint and history.- Sick for 7 days with sore throat and sent in with "tonsillitis". Had been on M. and B. 693 before admission. (27 half gram. tabs.). Illness started with epigastric pains and nausea. Pain in throat no better. Appetite poor lately, but no diarrhoea, vague aches and pains or eye trouble. During the last week he has had chest symptoms - Cough, spit and wheeze. Now improving. Great dysphagia persists. He developed a rash the day before admission. He does not smoke but drinks fairly heavily.

O.E. M. and B. rash, articarial, on both forearms and legs. Lungs - No dullness to percussion. Some scattered rales in all areas. Heart, abdomen, eyes and C.N.S. - N.A.D. . Gums free of infection. Some caries of teeth. Congestion of fauces and ulceration of R. tonsil. White, adherent membrane on its upper pole. Adenitis R. and L. sides of neck. Very painful on R. side.

Course. Day 1. Bed. Low, fluid diet. Gargle pot. chlor. 4 i.d. Thermogene to chest and throat. Ol. Ric. 34.

Day 2. Feels better but rash more extensive and on trunk. (No vomiting nor cough.) .3 gm. N.A.B. intravenously.

Day 3. Rash less pronounced. Throat improving.

Day 4. Chest clear. Very little cough.

Day 5. Rash gone.

Day 6. Tonsils both have filmy exudate on them. No sore throat.

Day 8. Throat swab still strongly positive.

Day 9. .45 gm. N.A.B. given.

Day 11. Only a small spot on R. tonsil. No constitutional symptoms.

Day 13. Discharged to duty.

Diet.- Breakfast. Bacon, eggs, Liver, kidney, sausages, bread, butter, tea and marmalade. Dinner - potatoes, cabbage, beef (stew). Peas, beans, mutton, lamb or corned beef. Turnip, carrots. Rice, sago or bread puddings. Apple or jam tarts with custard. Tea - Bread, butter, jam, marmalade. Supper - Egg and chips, sausage rolls, bacon, potatoes, meat pies, beetroot and cheese. Apples 3 or 4 per week. Oranges 2 per week. Bananas 2 per week. Chocolate about 3 bars per day, nut, fruit and nut, and milk. This diet is fuller than the ones the sailors on the ships get and it is reflected in his urinary vitamin C test.

Case 35. J.W. 23 years. Sloop for 4 months.

On admission. Temp. 98.4°. Pulse 78/min. Bowels - tendency to constipation. Tourniquet test negative. Urine. Dark straw colour. S.G. 1026. Acid. No bile, blood, sugar or albumen. Throat and gum swabs "positive".

Blood count. R.B.C. 4,930,000/c.mm. Hb. 98%. C.I. 1.

W.B.C. 6,800/c.mm. Polymorphs 71%. Lymphocytes 25%. Monocytes 4%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. First spec. 3.01 mg.%. 3 hrs. spec. 3.33 mg.%. 6 hrs. spec. 4.0 mg.%. (Vitamin C. deficiency).

No nicotinic acid estimation done.

Complaint and history. - Was 7 weeks in a small emergency hospital with Vincent's disease of gums and left, apparently cured, on 10

days sick leave. He had local applications only and no scaling. Now breaks his leave period c/o a relapse of his symptoms. Consulted dentist 2 days prior to admission with bleeding gums - no pain. Prior to this he has had continuous sea service since the outbreak of war. No diarrhoea, general pains, chest or eye trouble. Appetite fairly good, but some "indigestion" lately. Bad taste in mouth in mornings. Smokes 15 cigs./day. Light drinker.

O.E. Gums red, swollen and bleeding; delicate white film on lower especially. Breath heavy.

Bilateral submaxillary adenitis. Alveolar abscess beside second, bottom, left molar with creamy pus exuding. Has some carious molars and thick, crusted tartar round teeth bases, overlapped by the swollen gums. No scaling done when treated before.

Chest, heart, abdomen, eyes and C.N.S. - N.A.D. No glossitis. Tonsils clear of ulceration and membrane.

Course. Day 1. Routine scaling, brushing and Eusol spraying. Daily paints with 10% chromic acid. .45 gm. N.A.B. intravenously. Much bleeding caused.

Day 4. Still some swelling of gums and white membrane persists.

Day 6. Gums now paler and shrinking. Carious L. lower molar extracted and socket washed with H_2O_2.

Day 8. Gums about normal. Alveolar abscess healing in now. Continue daily treatment.

Day 10. Almost normal. No pain or bleeding. Adenitis has subsided.

Day 11. Discharged.

Diet. Same as Case 31.

Case 36. G.D. Minesweeper (S) for 7 months.

On admission. Temp. 98.0°. Pulse 80/min. Bowels open, but no diarrhoea. Tourniquet test negative. Urine. S.G. 1020. Acid. No albumen, sugar, bile or blood. Tonsil and gum swabs "positive".

Blood count. R.B.C. 4,370,000/c.mm. Hb. 92%. C.I. 1.06.

W.B.C. 7,100. Polymorphs 80%. Lymphocytes 15%. Monocytes 3%. Eosinophils 1%. Basophils 1%.

Urinary vit. C. estimation. First spec. 2.04 mg.%. 3 hrs. spec.

2.98 mg.%. 6 hrs. spec. 3.25 mg.% (Vit.C. deficiency).
No nicotinic acid estimation done.
Complaint and history. c/o sore throat. Has had sore throat on and off for 7 months. Lately has had sore gums. He has not been treated. Sent in for removal of tonsils. No diarrhoea, vague aches and pains, stomach or lung trouble. Says he has difficulty in seeing on the night watch now.
He does not smoke and is practically t.t. Gums bleed on brushing the teeth which he does not do regularly.
O.E. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.
Tonsillar glands enlarged L. ++. Deep ragged ulcer of L. tonsil with dirty grey slough on it. E.N.T. specialist reported that tonsillectomy was not advisable. Gums bleeding, swollen and had white, filmy membrane on outer aspects. Some ulcers with yellow sloughs at teeth bases. Inter-dental papillae swollen and red. Both tartar and caries of teeth. No glossitis.
Course. Day 1. Routine scaling, etc., of teeth. Daily paints with 10% chromic acid. H_2O_2 m.w. 4 i.d. .3 gm. N.A.B. intravenously.
Day 5. Throat clearing. No pain. Allowed up. Stop H_2O_2 and begin Eusol m.w.
Day 7. Throat clear. Depression left where tonsillar tissue has been destroyed. Gums have lost their filmy look and are firmer.
Day 8. Two small patches at back of lower wisdom tooth remain on R. side but they are healing.
Day 10. No membrane or ulceration anywhere.
Day 11. Discharged to duty.
Diet.- Breakfast - Kipper, bread, butter and tea. 2 apples per week eaten. Dinner. Roast mutton, roast potatoes, turnip, Yorkshire pudding. Roast beef. No sweet. Tea - Tea, bread, butter and jam. Supper - Sausages, Bacon and mashed potatoes. Tea.
This is a monotonous diet. All the cases from the ship complained of the monotony of it. There is not enough green food or fruit.

Case 37. W.W. 27 years. Trawler for 18 months.
On Admission. Temp 98.4°. Pulse 70/min. Bowels not open regularly. Tourniquet test negative. Urine - Clear. S.G. 1017. Faintly acid. No bile, blood, sugar or albumen.
Gum swab "positive".
Blood count. R.B.C. 4,750,000/c.mm. Hb. 95%. C.I. 1.01.
W.B.C. 11,800/c.mm. Polymorphs 77%. Lymphocytes 20%.
Monocytes 3%. Eosinophils 0%. Basophils 0%.
Urinary vit. C. estimation. First spec. 1.4 mg.%. 3 hrs. spec. 1.53 mg.%. 6 hrs. spec. 1.61 mg.%. (Vitamin C def.)
No nicotinic acid estimation done.
Complaint and history.- Gums bleed easily on rubbing. Breath is very bad. He is not complaining and was picked up at a routine dental examination. No pains in the limbs, no lung or stomach trouble and no diarrhoea. No eye trouble. He smokes 20 cigs/day and drinks beer and spirits.
O.E. Healthy looking, tanned man. Teeth - some caries, tartar at bases and brown staining. Gums red, tender and swollen. Tartar at teeth bases overlapped by swollen rounded gums. Shotty submaxillary and cervical glands. Tonsils clear. No glossitis. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.
Course. Day 1.- Routine scaling, rotary brushing, and Eusol spraying. Daily paints with 10% chromic acid. Full diet. Eusol m.w. 4 i.d.
Day 4. Gums I.S.Q. Continue daily treatment.
Day 7. Gums less red and breath less offensive. Shrinking of interdental papillae.
Day 9. Practically no membrane in the morning. Gums not bleeding when rubbed.
Day 12. Discharged.
Diet. Breakfast - Egg or kippers. Bacon on Sundays only. Bread, butter ++ and tea. Dinner - Soup, meat, potatoes. Cabbages twice a week. Tinned fruit and custard about twice a week. Tea - Tea, bread, butter and jam. Supper - Cheese or beetroot, or sausage and mash. Tea and bread. This is a poor, monotonous diet.

Case 38. E.E. 56 years. 2nd Hand on a trawler for 1 year. On admission. Temp.98°. Pulse 92/min. Bowels open. Tourniquet test positive. (over 100 spots). Urine - straw coloured. S.G. 1026. Faintly acid. No bile, blood, sugar or albumen. Gum swab positive.

Blood count. R.B.C. 4,500,000/c.mm. Hb. 70%. C.I. 0.78. W.B.C. 9,400/c.mm. Polymorphs 69%. Lymphocytes 27%. Monocytes 3½%. Eosinophils ½%. Basophils 0%.

Urinary vit. C. estimation. First spec. 1.38 mg.%. 3 hrs. spec. 1.43 mg.%. 6 hrs. spec. 2.27 mg.%. (Vit. C. deficiency). Blood ascorbic acid 0.04 mg.%. No nicotinic acid estimation done.

Complaint and history.- Complains of inability to see at night. He had been on the ship for a year with no change of diet, and in trawlers all his life before that. He first noticed the inability to see at night 2 months ago. At this time his gums were spongy and bleeding. Vincent's infection was present and 7 bad teeth were pulled. He still has a few teeth with tartar at the bases. He has used glycerine and thymol m.w. for 2 months but the gums still bleed on pressure and are tender. No sore throats, diarrhoea, epistaxis or pains in the back or limbs. No chest trouble. He drinks spirits but does not smoke.

O.E. Ophthalmologist's report. R + .25 sph. = $\frac{6}{6}$ R $\frac{6}{60}$

L + .25 sph. = $\frac{6}{6}$ L $\frac{3}{36}$

Gums, specially the lower, are very spongy, red and bleed easily. Vincent's membrane present. Tartar encrusted at bases of remaining teeth. Breath very heavy. Tonsils clear. No glossitis. No keratomalacia, no adenitis. Heart, lungs, abdomen, C.N.S. - N.A.D. This is a case of scurvy. Thin, pale subject.

Course. Day 1. Routine scaling, brushing and Eusol (1 in 4) spraying. Ascorbic acid 1,400 mg. t.d.s.. Mist. ferri et ammon. cit. 1 oz. t.d.s. Daily paints of gums with 10% chromic acid. Eusol m.w. 4 i.d.

Day 3. Gums I.S.Q. Sample of urine shows 4.3 mg.% ascorbic acid.

Day 5. Less redness & swelling of the gums. Still bleed on friction. Skin condition on legs suggestive of psoriasis-Lotio

calamin. co.

Day 8. Membrane not so copious. Interdental papillae shrinking.

Day 10. Gums practically normal. Skin condition on legs practically clear.

Day 12. Discharged.

Diet. - Breakfast - Bacon and eggs every morning. Tea, bread, butter ++ and jam. Dinner - Mutton or beef. Pork on Sundays. Potatoes, cabbages, carrots or tinned peas. Tea - Tea, bread, butter and jam. Supper - Tea or coffee, bread, butter, jam. Never any fruit. He has had no apples or oranges for many years now.

Case 39. D.A.G. 32 years. Trawler (A) for 7 months.

On admission. Temp. 98°. Pulse 78/min. Bowels not open. Tourniquet test negative. Urine pale. S.G. 1010. Acid (faint). No bile, blood, sugar or albumen. Gum swab positive.

Blood count. R.B.C. 4,150,000./c.mm. Hb. 87%. C.I. 1.06. W.B.C. 7,200/c.mm. Polymorphs 80%. Lymphocytes 15%. Monocytes 4%. Eosinophils ½%. Basophils ½%.

Urinary vit. C. estimation. First spec. 3.17 mg.%. 3 hrs. spec. 2.86 mg.%. 6 hrs. spec. 2.94 mg.%. (Vit. C. deficiency).

No nicotinic acid estimation done.

Complaint and history. Gums beginning to swell and bleeding on pressure. Aching of lower jaw of two days duration. Bad taste in mouth. No general aches and pains. Slight sore throat 7 days before admission but that is better now. No bronchitis, stomach trouble nor nyctalopia. He drinks heavily but is a non-smoker. Lately he has been very constipated and has had no diarrhoea at any time.

O.E. Front teeth below are loose. Breath offensive. Teeth have thick, hard tartar at the bases. Caries of molars and deep brown staining. Gums swollen and tender. Filmy exudate in labio-gingival furrows. Boggy, eroded areas behind upper central incisors. Tonsils clear, but large ulcer with white membrane in front of R. anterior faucial pillars. Cervical adenitis R. and L. No glossitis. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.

Course. Day 1. - Routine scaling, rotary brushing and spraying with Eusol. Daily paints of gums with 10% chromic acid and pot. permang. m.w. 4 i.d. 3 vegetable laxatives.

Day 2. Daily treatment + 0.45 gm. N.A.B. intravenously.

Day 4. Gums I.S.Q. Less aching.

Day 6. Ulcer on anterior pillar of fauces clearing up. Smaller in size. Gums less red and fiery.

Day 8. Noticeable improvement in gums. Not bleeding on pressure. More firm. Faucial ulcer healed.

Day 9. Practically no visible membrane. Neck not tender.

Day 10. Discharged.

Diet. Same as case 18.

Case 40. F.S. 18 years. Battleship 2 weeks, and camp for 10 weeks previously. Steel worker in civil life.

On admission. Temp. 102.6°. Pulse 84/ min. Bowels not open. Tourniquet test negative. Urine deep straw colour. S.G. 1025. Acid. No bile, blood, sugar or albumen. Throat swab strongly positive. (Hundreds of fusiform organisms only, seen).

Blood count. R.B.C. 4,360,000/c.mm. Hb. 96%. C.I. 1.1. W.B.C. 8,400 per c.mm. Polymorphs. 61%. Lymphocytes 31%. Monocytes 5 %. Eosinophils 2%. Basophils 1%. A Türks cell was seen.

Urinary vit. C. estimation. First spec. 2.7 mg.%. 3 hrs. spec. 2.65 mg. %. 6 hrs. spec. 2.8 mg.% (Vitamin C deficiency).

Nicotinic acid excreted per day equal to 5.14 mg.

Complaint and history. Sore throat and sore eyes came on 24 hrs. ago. Sudden onset with slight headache too. Lips dry and sore. Cough developed. He became ill in the night. He smokes 10 cigs. per day and does not drink alcohol. No previous "rheumatism", lung trouble or diarrhoea.

O.E. Ill and toxic patient. Lips ashy grey, dry and sore. Offensive breath, not like a diphtheria smell. Blepharitis in both eyes. Signs of bronchitis in the chest. Throat - injected. Tonsils not large but covered with a white, filmy, exudate. Swab and culture - negative for K.L.B. Has been on M and B 693 outside so agranulocytosis was suspected. Blood count ruled this out. Teeth unclean and some caries. Heart, C.N.S. and abdomen - N.A.D. Tender, swollen adenitis R. and L. in neck.

Course. -Day 1. P.M. of day of admission. Extension of white film

on inside of mouth and throat. Small ulcers appeared on buccal mucosa. Very ill. M and B 693 - 2 t.d.s. Thermogene to neck and chest. Garg. pot. chlor. 2 hourly. Lotio A.B. + ungt. hydrarg. ox. flav. to eyes. Fluids.

Day 2. Feels a little better. Extension of white film in the mouth. Still ill. Mandl's paint and brandy ½ oz. 4 hourly in addition. N.A.B. 0.45 gm. intravenously.

Day 3. Feels better. appearances of mouth slightly improved. Vomited during the night.

Day 4. Mouth no worse. Enema given. M. and B. 693 - 1 t.d.s. and Beaumnnt's arsenic paint given.

Day 5. Membrane less in amount. Eyes better.

Day 7. Membrane vanishing. Many small ulcers persist in buccal mucosa.

Day 8. Mouth improving. No generalised membrane.

Day 10. Much improved generally. Few ulcers still present on inside of cheeks. Gums filmy looking but tonsils better. Shotty glands R. and L. in neck now. Wants up.

Day 11. Up one hour. full diet. Ulcers have cleared up.

Day 17. Steady improvement. Has been on Beaumont's paint only for the last six days.

Day 18. Discharged to 14 days sick leave.

Diet. In the Camp. Breakfast - Sausage, egg, bread, butter and tea. One apple per week was the only fruit. Dinner - Meat, cabbage and potatoes every day. Milk pudding or boiled apples. Tea. - Jam, bread, tea or cake. Supper - Corned beef + beetroot, or liver and gravy, or meat and potato. The only fresh fruit was the one apple per week. Cabbage was the only vegetable.

CASE 40.

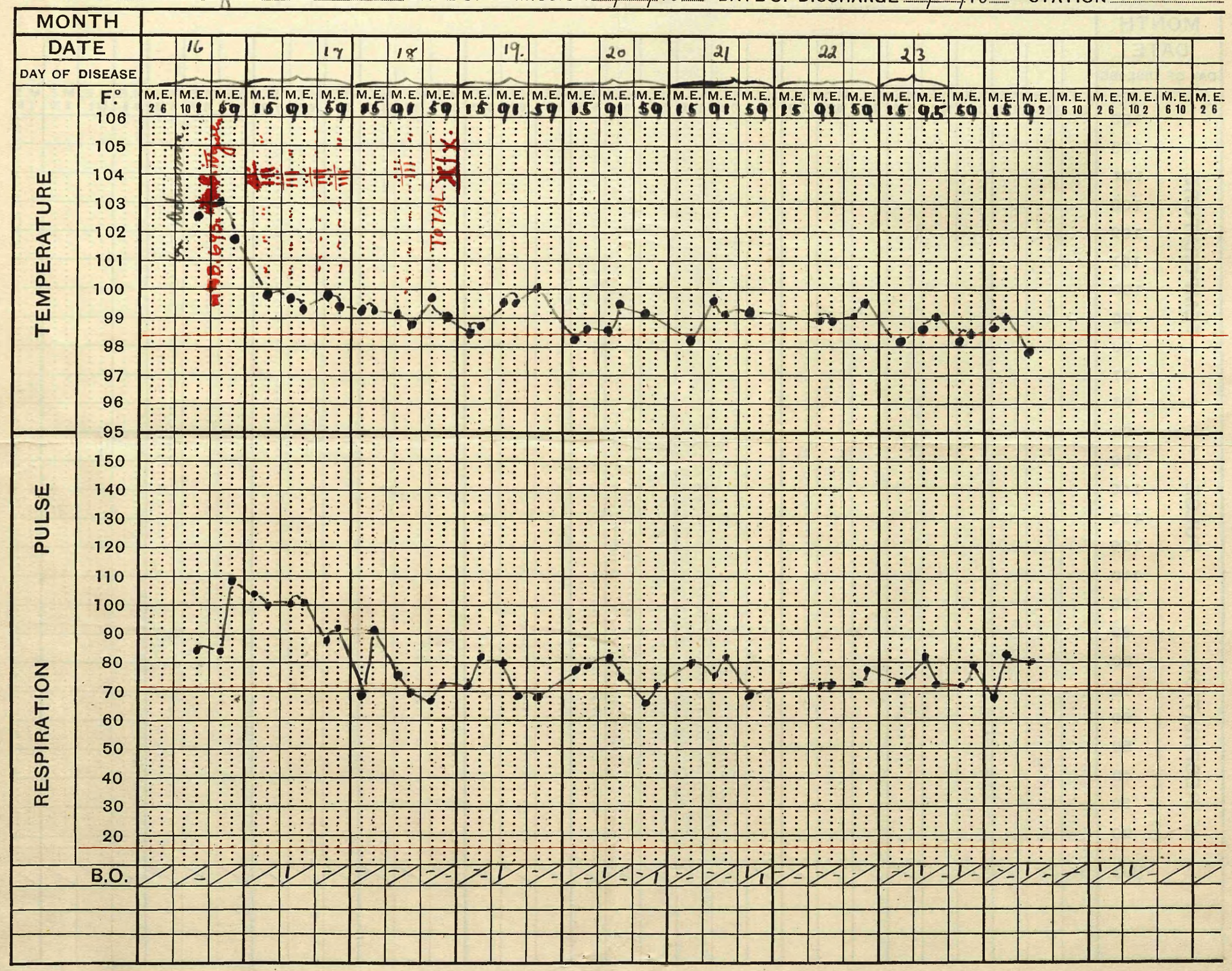
UNIT OR SHIP Battleship.

CLINICAL CHART
(TO BE ATTACHED TO CASE SHEET)

ARMY FORM. B. 181
NAVAL FORM. M. 41
R.A.F. FORM. 549

NO. ____ RANK AND NAME F. W. Stacey. Gnr. Sea AGE 18 yrs. HOSPITAL ____

DIAGNOSIS Vincents Angina. DATE OF ADMISSION 16/12/1940. DATE OF DISCHARGE __/__/19__ STATION ____



FORMS/181/17.

SIGNATURE OF M.O./c ____

Case 41. S.E. 26 years. Has lived in motor boats for 1 yr.
On admission. Temp. 98.6°. Pulse 78/min. Bowels regular once a day. Tourniquet test negative. Urine - clear, S.G. 1020. Faintly acid. No bile, blood, sugar or albumen. Gum swab positive.
Blood count. R.B.C. 4,850,000/c.mm. Hb. 98%. C.I. 1.02. W.B.C. 10,800/c.mm. Polymorphs 66%. Lymphocytes 29%. Monocytes 5%. Eosinophils 0%. Basophils 0%.
Urinary vitamin C estimation. First spec. 20.0 mg.%. 3 hrs. spec. 29.5 mg.%. 6 hrs. spec. 36.36 mg.%. (No deficiency of vit.C).
No nicotinic acid estimation done.
Complaint and history.- Has had "Trench Mouth" for 4 weeks. He was seen by a dentist 16 days ago. Urine tests then showed a deficiency of vitamin C and the blood value was 0.21 mg.%. The only treatment ~~was~~ given was daily paints with 10% chromic acid + 300 mg. vit. C daily. There has been no improvement in his gums which are tender and raw. He has had no diarrhoea, stomach upsets, lung or eye trouble and no vague aches and pains. He smokes and drinks in moderation.
O.E. Typical "Trench Mouth". Bright red, tender, swollen gums. Prominent interdental papillae. White membrane present in gingivo-labial furrows. Pain on everting the cheeks. Teeth have no tartar visible but some is embedded and overlapped by the swollen gum. Slight caries. Tonsils clear. No glossitis. Shotty non-tender adenitis R. and L. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.
Course.- Day 1. Embedded tartar scaled. Rotary brushing and Eusol spraying of teeth. M.W. pot. permang. 4 i.d. Daily gum paints with 10% chromic acid. 0.45 gm. N.A.B. given intravenously.
Day 4. Less pain in gums. Redness subsiding.
Day 6. Still some membrane in gingivo-labial sulcus. Interdental papillae less prominent. Continue daily treatment.
Day 8. Gums very much better. No red, angry colour now. Colour is a uniform healthy pink. Practically no membrane.
Day 9. Fit for discharge.
Diet. Same as case 2 but no eggs for tea.

Case 42. C.H. 40 years. Paddle minesweeper for 12 months.
On Admission. Temp. 99.0°. Pulse 97/min. Bowels open. Tourniquet test negative. Urine - N.A.D. S.G. 1018. Gum swab negative. Throat swab positive.
Blood count. R.B.C. 5,000,000/c.mm. Hb. 95%. C.I. 0.95. W.B.C. 9.600/ c.mm. Polymorphs. 74%. Lymphocytes 20%. Monocytes 5%. Eosinophils 1%. Basophils 0%.
Urinary vit. C estimation. First spec. 3.95 mg.%. 3 hrs. spec. 4.02 mg.%. 6 hrs. spec. 4.61 mg.%. (Vitamin C deficiency)
No nicotinic acid estimation done.
Complaint and history. Has had several attacks of shivering followed by a hot stage then sweating. No illness before yesterday. Had several severe attacks yesterday. No headache nor vomiting. He has had no cough, general pains, nor nyctalopia. Sore throat - R. side only. No pain in neck. No diarrhoea. He was thought to have had malaria in 1934 but this was never confirmed by blood film. He smokes 10 cigs. per day and does not drink alcohol.
O.E. Vincent's infection of R. tonsil. Post-nasal discharge. Chest - N.A.D. Heart, C.N.S. and eyes - N.A.D. Abdomen - No splenic enlargement. Blood film - no evidence of malaria. Gums swollen but not infected. Some tartar but no caries of teeth. No glossitis. Slight R. cervical, non-tender adenitis.
Course. Day 1. Teeth brushed and sprayed at outset. 0.45 gm. N.A.B. intravenously. Gargle pot. chlor. 4 i.d. and Eusol m.w.
Day 2. No pain in throat. Still a white patch on R. tonsil. Not spreading.
Day 4. Slough is dirty yellow in colour. Appears to be shrinking.
Day 5. Ulcer on tonsil rapidly healing at edges. Slough coming away "en morceaux". Continue m.w. and gargles.
Day 6. Only a very small white spot on upper pole of tonsil. Patient has no pain and feels fit.
Day 7. Ready for discharge.
Diet. Breakfast - Sausages, eggs or bacon. Tea, bread and butter ++. Dinner - Roast beef and potatoes. Dried peas and cabbage are the only vegetables. Rice puddings about once a week. They have no

fruit in the diet. The last orange was 6 months ago.
Tea - bread, tea, butter and jam. Supper - Eggs and bacon or sausages. He complains of the monotony of the diet.

Case 43. H.A. 33 years. Trawler (A) for 15 months.
On admission. Temp. 98.2°. Pulse 64/min. Bowels constipated.
Tourniquet test negative. Urine - dark colour. S.G. 1026.
Alkaline. No bile, blood, sugar or albumen. Gum swab positive.
Blood count. - Not done.
Urinary vitamin C estimation. First spec. urine 2.06 mg.%.
3 hrs. spec. 2.03 mg. %. 6 hrs. spec. 2.27 mg.%. (def.of vit.C).
No nicotinic acid estimation done.
Complaint and history.- Complains of a gumboil 7 days ago on the R. lower jaw posteriorly. Then toothache in the R. lower pre-molar. No bleeding of gums. Bad taste in mouth in mornings. Breath heavy. No cough. Troubled with constipation and for a week has had a "bad stomach" i.e. pains before meals relieved by food, and flatulence. He has not been chewing his food owing to tender gums recently. He is a non-smoker but drinks beer. No "rheumatism" or eye trouble.
O.E. Well built patient with no clinical evidence of anaemia. False membrane on cheek behind lower R. molar. Gums spongy but not bleeding readily. Breath heavy. Teeth have recently been brushed before entering hospital but there is evidence of staining at the bases and slight caries recently stopped down at the gum margins. Tonsils clear. No adenitis. Heart, lungs, C.N.S. and eyes - N.A.D. Abdomen - Non-tender but very slight guarding in epigastrium. No glossitis.
Course. Day 1. Routine scaling, rotary brushing and Eusol spraying. Daily painting with 10% chromic acid. 0.45 gm. N.A.B. intravenously. M.W. Eusol (1 in 4) 4 i.d. 1 No. 9 pill.
Day 4. Less tenderness in gums. Raw, red area uncovered by membrane behind lower R. molar. Some filmy membrane on outer aspects of upper gums.
Day 6. Improvement in R. retro-molar ulcer. Very little membrane on gums first thing in the morning. Breath less offensive. Continue

daily treatment.

Day 7. Gums of normal colour and size. Sponginess gone.

Day 8. Fit for discharge.

Diet history.- Same as case 18.

Case 44. G.B. 23 years. Destroyer for 8 months.

On Admission. Temp. 100°. Pulse 84/min. Bowels not open. Tourniquet test negative. Urine - S.G. 1017. Faintly acid. No bile, blood, sugar or albumen. Gum and throat swabs both positive.

Blood count. R.B.C. 3,740,000/c.mm. Hb. 98%. C.I. 1.32. W.B.C. 3,200/c.mm. Polymorphs 86%. Lymphocytes 11%. Monocytes 3%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. First spec. 2.93 mg.%. 3 hrs. spec. 3.27 mg. % . 6 hrs. spec. 3.85 mg.%. (Vitamin C deficiency)

No nicotinic acid estimation done.

Complaint and history. - Sore throat for 2 to 3 days. Pain in gums 2 weeks ago, starting in R. wisdom tooth and after extraction of the tooth the pain went round the mouth to a similar position on the L. side. No bleeding of gums. Has felt off colour lately. Bowels are constipated. Not troubled with general aches and pains, stomach, lung or eye trouble. He does not smoke but drinks alcohol in moderation.

O.E. No constitutional symptoms. R. tonsil enlarged and covered with a copious exudate, filmy and white. L. tonsil injected and red but has no membrane on it. Both tonsillar glands large and tender. Tonsillar-smear - no B. Diphtheriae in direct smear. Culture and subsequent result - Negative for K.L.B. and haemolytic streptococci. Gums swollen and redder than normal. Covered with filmy exudate. No ulceration. Teeth show much tartar and caries of molars. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D.

Course. - Day 1. Routine scaling, brushing and Eusol spraying. Carbolised dressing to carious molars. 10% chromic acid daily to the gums. 0.3 gm. N.A.B. intravenously. Bed and low diet. Garg. pot. chlor. t.d.s. Thermogene to the throat. Liquid paraffin and cascara 2 drachms.

Day 2. Throat feels easier. Some slough separating. Continue gargles and thermogene.

Day 6. Throat appears clear of infection. Up for 1 hour. Gums still infected. Membrane in upper labio-gingival sulcuc and red tender area on R side there. Continue chromic acid daily, also H_2O_2 m.w. 4 i.d.

Day 8. Less membrane and visible improvement of gums, but H_2O_2 makes them raw - use the latter twice a day.

Day 10. Gums appear healed but he still complains of pain in the L molar region. M.W. H_2O_2 continued.

Day 12. No complaints and mouth appears healthy.

Day 13. Discharged.

Diet. Breakfast - Ham, sausage, or steak and eggs. Porridge 3 times a week. Bread, butter or margarine and tea. Dinner - Roast or stewed meat with roast or boiled, mashed potatoes and gravy. One vegetable - cabbage, tinned carrots or dried peas. (soaked in water). Plum duff on Sundays. Tea - Bread, butter, jam, tea, and biscuits extra. Supper - Sausage and "mash" with pork and beans. Liver and bacon. Fish and chips. Meat pies. Bread, butter and tea.

Case 45. W.H. 27 years. Shore establishment for meals, but working in motor boats by day for I year.

On admission. Temp. 97°. Pulse 64/min. Bowels open but no diarrhoea. Tourniquet test negative. Urine clear. S.G. 1015. Strongly acid. No bile, blood, albumen or sugar. Gum swab positive.

Blood count. R.B.C. 4,390,000/c.mm. Hb. 88%. CI. 1.02. W.B.C. 15,300 /c.mm. Polymorphs 72%. Lymphocytes 24%. Monocytes 4%. Eosinophils 0%. Basophils 0%.

Urinary vit. C estimation. First spec. 1.2 mg.%. 3 hrs. spec. 2.04 mg.%. 6 hrs. spec. 12.5 mg.%. (No vitamin C deficiency). No nicotinic acid estimation done.

Complaint and history.- Complains of sore gums for one week. Had "tonsillitis" a week ago for 4 days. Also had it a year ago. Gums bleed easily. Always has bad taste in his mouth in the mornings. He had a bad attack of "indigestion" a week ago,

but this has cleared up.- Discomfort and acid eructations after meals. Bowels regular. No pains or aches in his limbs. No eye troubles. He smokes 15 to 20 cigs/day, and drinks in moderation.
O.E. Gums swollen and red. White film between the teeth and embedded at the roots. Teeth dirty and unclean on admission. No caries. Roof of mouth behind central incisors boggy and eroded. Tonsils large but clear of infection. Shotty, non-tender adenitis R. and L. Mucosae pink and not anaemic. Heart, lungs, abdomen, eyes and C.N.S. - N.A.D. A mild case of Vincent's infection of the gums.
Course. Day 1. Thorough scaling, rotary brushing with pumice stone powder and then spraying with Eusol solution. Daily gum paints with 10% chromic acid and m.ws. of Eusol (1 in 4) 4 i.d.
Day 4. Membrane less copious and interdental papillae less swollen and healthier. Still membrane behind upper central incisors.
Day 6. Great improvement. Practically no pain in the gums unless on manipulation.
Day 7. Almost fit for discharge. Gums shrunken and appear normal. Have lost their glazed appearance.
Day 8. Discharged to duty.
Diet. Breakfast - Bread, butter, marmalade and tea, except one day in four when he gets fish or bacon and eggs. He has an orange every day for breakfast. Dinner - Meat, potatoes and cooked fresh vegetables. Sprouts ++. Milk pudding. Tea - Bread, tea, butter, jam and scone. Supper - Cooked supper, fish etc., + tea, bread and butter.
This man gets apples and other fruit regularly from the shore canteen.

Case 46. M.G. 19 years. Trawler for 5½ months.
On admission. Temp. 98.8°. Pulse 82/min. Bowels not open. Tourniquet test negative. Urine. Dark straw colour. S.G. 1025. No bile, blood, sugar or albumen. Gum and throat swabs "positive". Blood count. R.B.C. 4,960,000/c.mm. Hb. 100%. C.I. 1.02. W.B.C. 10,200/c.mm. Polymorphs. 71%. Lymphocytes 22%. Monocytes

5%. Eosinophils 1½%. Basophils ½%.

Urinary vitamin C estimation. 1st. spec. 4.65 mg.%. 3 hrs. spec. 5.82 mg.% 6 hrs. spec. 6.17 mg.%. (Slight vit. C. deficiency).

Nicotinic acid excreted per day equal to 4.35 mgs.

Complaint and history. Pain in throat on swallowing for 3 days. Has been off colour for a week. Gums bleeding and tender on chewing hard food for about two weeks. Bad taste in mouth in the mornings and poor appetite lately. No stomach or lung trouble and no trouble with his eyes at night. Has never had vague aches and pains in his limbs and no diarrhoea. He smokes cigarettes and a pipe in moderation and does not drink alcohol.

O.E. Teeth foul and covered with tartar which was encrusted round teeth bases. Gums swollen, red and tender. Gum margins ulcerated and have yellowish sloughs on them. Left tonsil ulcerated and has white patches on it. Tender left cervical adenitis. Heart, lungs, abdomen, eyes and C.N.S. - N.A.D.

Course. Day 1. Thorough routine scaling, brushing and Eusol spraying. Daily gum paints with 10% chromic acid. Nicotinic acid 50 gr. five times a day given. (250 gr./day).

Day 3. Throat still painful. Daily nicotinic acid and chromic acid continued. .3 gm. N.A.B. intravenously.

Day 4. Pain in throat gone. Gums I.S.Q.

Day 6. Gums less red and beginning to alter in appearance. Still much membrane in labio-gingival sulcus. Ulcers at teeth bases contracting and looking more healthy.

Day 8. All the fiery appearance has gone from gums. Practically no membrane. Throat healed.

Day 10. Gums shrunken and no membrane seen. Patient ready for discharge.

Diet history.- Breakfast - Fish or bacon or tinned sausages and eggs. Bread, butter, tea. Porridge occasionally. Dinner - Roast or stewed meat, Yorkshire pudding, pork. Cabbage, peas, carrots or sprouts, "bubble and squeak". Sweet on Sundays and Thursdays - plum duff or milk pudding. Tea - Bread, butter, jam and tea. Supper - Beans, liver and bacon, fish and chips, or sausage pie. Bread, butter and cheese. No fruit at all in the diet and as they do much sea time they get no chance to buy it ashore.

Case 47. J.M. 29 years. Paddle sweeper (Z) for 6 months.
On admission. Temp. 98.2°. Pulse 65/min. Bowels regular once a day. Tourniquet test negative. Urine. S.G. 1022. Acid. No bile, blood sugar or albumen. Gum swab positive. Blood Count - R.B.C. 4,450,000/c.mm. Hb. 96%. C.I. 1.09. W.B.C. 8,400/c.mm. Polymorphs 66%. Lymphocytes 29%. Monocytes 4%. Eosinophils 1%. Basophils 0%.
Urinary vit. C. estimation. 1st spec. 15.6mg.%. 3 hrs. spec. 31.45 mg.%. 6 hrs spec. 42.36 mg.%. (No deficiency of vit.C.)
No nicotinic acid estimation done.
Complaint and histroy. Has had sore and bleeding gums for 5 to 6 weeks. No sore throats. Appetite poor lately and has felt easily tired and off colour. Bad taste in mouth in the mornings. No "Rheumastism", diarrhoea or abdominal or lung upsets. He has no difficulty in seeing at night. He both smokes and drinks in moderation. He has been treated for 14 days with ascorbic acid (300 mg. daily) and glycerine and thymol m.w. with no improvement. He had no scaling, etc.
O.E. Some caries of molars. Scales and tartar at bases of the teeth. Overcrowding of the teeth and very little room between them. Tonsils clear but large. No glossitis. Gums tender, red and swollen. Lower gums bleed on instrumentation. Ulceration in R. retro-molar sulcus. Heart, lungs, abdomen, C.N.S. and eyes - N.A.D. Shotty, non-tender adenitis on R. side only.
Course. Day 1. Routine scaling, brushing and Eusol (1 in 4) spray. Daily 10% chromic acid to gums. Full diet. Nicotinic acid, 250 mg. daily, and H_2O_2 m.w. regularly.
Day 4. Gums I.S.Q.
Day 6. Gums less tender and less red.
Day 7. Carious molars now extracted under lingual nerve block. Sockets kept clean with H_2O_2 m.w.
Day 8. Eusol substitued for H_2O_2 as gums are tender. Still some membrane on outer aspect of lower gum. Ulcer in R. retro-molar sulcus healed. Some redness and glassy membrane mark the site.
Day 10. Great improvement. Teeth clean and gums almost clear of infection. Teeth sockets healthy and healing.

Day 12. Ready for discharge.

Diet. Same as Case24.

Case 48. J.E. 18 years. Paddle sweeper (Z) for 4 months.

On admission. Temp. 98.6°. Pulse 76/min. Bowels constipated. Tourniquet test negative. Urine - pale straw colour. S.G. 1022. Acid. No bile, blood, sugar or albumen. Gum swab positive.

Blood count. - Not done.

Urinary vitamin C. estimation. 1st spec. 2.45 mg.%. 3 hrs. spec. 2.62 mg.%. 6 hrs. spec. 2.53 mg. %. (Vit. C. deficiency).

No nicotinic acid estimation done.

Complaint and history. Bad taste in mouth and aching pains in his gums for 3 weeks. No pain in throat and no dysphagia. Has had a slight cough and "wheeze" at nights for a week. No "rheumatic pains", stomach or eye trouble. No diarrhoea or sore tongue. He does not smoke but drinks beer occasionally.

O.E. Gums soft, swollen and ulcerated at teeth bases. Breath heavy. Tonsils free of infection. Shotty adenitis R. and L. No glossitis. Slight caries of teeth (some fillings) and encrusted tartar on teeth. Heart, abdomen, C.N.S. and eyes -N.A.D. Scattered rales in chest on deep breathing. (mild bronchitis).

Course. He was given nicotinic acid (gr.50 t.d.s.) every day for 8 days before admission, in a neighbouring sick quarters. There was no local treatment. No improvement resulted. His pain has got worse.

Day 1. In hospital. Routine scaling, brushing and Eusol spraying. 0.45 gm. N.A.B. intravenously. Daily gum paints with 10% chromic acid. 2 vegetable laxative pills. Bed and a full diet.

Day 3. Gums I.S.Q.

Day 5. Pain in gums is easier. Less redness evident. Ulcers are beginning to contract and heal. Still much membrane in upper labio-gingival sulcus and on outer aspects of upper gums. Continue daily chromic acid paints and Eusol (1 in 4) mw.s.

Day 8. No pain in gums now but upper gum is still ulcerated and is covered with white membrane. Substitute pot. permang. for

Eusol as a m.w.

Day 11. Membrane is getting less in amount. Gums shrunken and more normal in colour.

Day 14. Gums appear clear of infection. Swab taken in the morning is "negative".

Day 15. Discharged.

Diet. Same as case 24.

Case 49. P.L. 21 years. Trawler hand for 18 months.

On admission. Temp. 98.6°. Pulse 68/min. Bowels open once a day. Tourniquet test negative. Urine - S.G. 1028. Faintly acid. No bile, blood, sugar or albumen. Gum swab positive.

Blood count. R.B.C. 3,870,000/c.mm. Hb. 70%. C.I. 0.92. W.B.C. 11,800/c.mm. Polymorphs 60%. Lymphocytes 32%. Monocytes 6%. Eosinophils 1%. Basophils 1%.

Urinary vit. C. estimation. 1st spec. 3.72 mg.%. 3 hrs. spec. 3.84 mg.%. 6 hrs. spec. 4.16 mg.% (Vitamin C. deficiency).

No nicotinic acid estimation done.

Complaint and history. Has had bleeding and tender gums for 3 weeks. Now he cannot eat solid food without pain. Blood on his pillow in the morning when he awakes and bad taste in his mouth then. No diarrhoea, "rheumatism", chest or stomach trouble. He has not noticed increased difficulty in seeing at night lately. He smokes about 20 cigarettes per day and only drinks spirits in moderation.

O.E. Eyes, heart, lungs, abdomen and C.N.S. - N.A.D. Tonsils free from infection but large and unhealthy looking. Teeth are yellow and unbrushed and have thick, hard tartar at their bases, overlapped by the swollen gums. No caries. Lower gum swollen, red and with prominent interdental papillae. No frank ulceration but foul breath and film of white membrane over gum and in labio-gingival sulcus. Non- tender shotty adenitis R. and L. Upper gum swollen and red but has no membrane. A moderate case.

Course. Day 1. Routine scaling, brushing, spraying and daily painting with 10% chromic acid. Mist. ferri et ammon. cit. 1 oz. t.d.s. Nicotinic acid - 50mg. 5 times a day. 0.45 gm. N.A.B.

intravenously.

Day 3. Gums I.S.Q. Still tender and paining.

Day 5. Gums less tender and have lost their bright red colour at the margins.

Day 7. No pain in gums. Interdental papillae less prominent. Membrane not so copious first thing in a morning.

Day 8. Great improvement. Gums less swollen and normal in colour.

Day 10. No membrane visible. No bleeding on rubbing gums. Discharged to duty.

Diet.- Breakfast - Steeped salted ham and preserved eggs. Porridge and tinned milk. Tea, bread and butter ++. Dinner - Nearly always "bully beef" in various guises. Potatoes and tinned vegetables heated up. Tinned pears on Sundays. Plum duff twice a week. Tea - Bread, butter, jam, biscuits and tea. Supper - Pork and beans and egg. Sausage and mash. Bully beef, cheese, and butter and bread. Cocoa.

Case 50. A.H. 26 years. Drifter for 5½ months.

On admission. Temp 98.0°. Pulse 72 per min. Bowels not open. Tourniquet test negative. Urine - pale straw colour. S.G. 1018. Acid. No bile, blood, sugar or albumen. Gum and throat swabs strongly positive. Many B. fusiformes evident.

Blood Count. R.B.C. 4680,000 /c.mm. Hb. 98%. C.I. 1.06. W.B.C. 10,000/c.mm. Polymorphs 72%. Lymphocytes 25%. Monocytes 3%. Eosinophils 0%. Basophils 0%.

Urinary vit. C. estimation. 1st spec. 2.31 mg.%. 3 hrs. spec. 2.85 mg.% 6 hrs. spec. 3.16 mg.% (Vit. C. deficiency).

No nicotinic acid estimation done. W.R.+

Complaint and history. Had three injections of N.A.B. for primary syphilis, the last one month ago. Treatment lapsed as he changed his base. Gums were sore at that time but lately the pain has left them. Now complains of pain in the throat of 3 days duration. Gums bleed on brushing them. No diarrhoea, but constipated for the past week. He has never had vague aches and pains in his limbs. No recent lung, eye or stomach ailments. He both smokes and

drinks heavily.

O.E. Heart, lumgs, abdomen, C.N.S. and eyes - N.A.D. Slight scar at fraenum of penis and shotty glands both groins. R. tonsil ulcerated and white membrane adherent to its upper pole. R. cervical tender adenitis. L. tonsil injected but no ulceration. Gums red and swollen and covered with white membrane on outer aspects. Breath very offensive. Carious molar and no gross tartar. This is a very mild gum infection.

Course. Day 1. Teeth cleaned with rotary brush and Eusol spray. Daily 10% chromic acid applications to the gums. 0.45 gm. N.A.B. intravenously. Garg. pot. chlor. 4 i.d. 2 vegetable laxative pills. Nicotinic acid - one 50mg. tablet 5 i.d. Carbolised dressing to molar.

Day 2. Pain in throat much easier.

Day 3. Membrane is not spreading and is yellowish in colour. No pain. gums I.S.Q.

Day 5. Membrane rapidly vanishing from R. tonsil. Tenderness going from neck glands.

Day 7. Tonsil now clear of membrane. No pain in throat. Labio-gingival furrows have shrunk and very little membrane remains on outer side of upper gum at level of R. bicuspid tooth.

Day 9. Remaining raw area on upper gum almost free of infection. No membrane visible. Neck glands shotty but non-tender.

Day 11. Discharged to duty. No inflammation or membrane in any area.

Diet.- Same as Case 30.

Case 51. W.O. 26 years. Destroyer for 3½ months and trawler previously.

On admission. Temp 99.4°. Pulse 80/min. Bowels open. Tourniquet test negative. Urine clear. S.G. 1012. Acid. No bile, blood sugar or albumen. Gum swab positive.

Blood count. R.B.C. 4,920,000/c.mm. Hb. 99%. C.I. 1.04. W.B.C. 9,600/c.mm. Polymorphs 78%. Lymphocytes 19%. Monocytes 3% Eosinophils 0%. Basophils 0%.

Urinary vit. C estimation. 1st spec. 2.16 mg.%. 3 hrs. spec. 2.23 mg.% 6 hrs. spec. 2.84 mg.% (vit. C. deficiency).

Nicotinic acid excreted per day equal to 3.42 mg. in 46 ozs. urine.
Complaint and history. Pain in the gums for 2 weeks and tenderness of left side of neck. Gums sore on chewing food and bleed on biting crusts and other hard food. Bad breath and bad taste in the mouth in the mornings. Has had a cough and spit for ten days. No diarrhoea, no stomach trouble, no nyctalopia and no general aches and pains. He smokes and drinks in moderation.
O.E. Bad breath evident. Gums filmy and swollen. Easily bleed on rubbing. Red and eroded behind lower L. molars, and much white membrane there. Some erosion and bogginess up behind central incisor teeth. L. lower wisdom tooth septic at root and pus exuding on outer aspect of gum. Tonsils free and no glossitis. Tender, non-supurating, L. cervical adenitis present. Tartar at teeth bases. Heart, abdomen, C.N.S. and eyes - N.A.D. Lungs - R.M. vesicular in all areas with scattered post-tussive rales audible. Mild bronchitis.
Course. Day 1. Routine scaling, brushing and Eusol spraying. Daily paints with 10% chromic acid and m.w. Eusol (1 in 4) 4 i.d. Nicotinic acid 50 mg. 5 i.d.
Day 2. Still tenderness in gums. 0.3 gm. N.A.B. given intravenously. Nicotinic acid and chromic acid continued and pot. chlor. m.w. instead of Eusol.
Day 5. Gums firmer and less tender. Septic wisdom tooth pulled under gas. Socket sprayed with H_2O_2. Chest appears clear.
Day 8. Socket healing. Less membrane in retro-molar sulcus and on outer aspects of gums. No pain in neck glands. H_2O_2 m.w.s
Day 10. No tenderness or bleeding. Still a small healing ulcer behind L. lower molars. Tooth socket clean and rapidly granulating in.
Day 12. All areas free from membrane and ulcers healed. Discharged.
Diet.- Breakfast - Liver or sausages or bacon with eggs. Fresh fish about twiece a week. Tea, bread, butter ++. Porridge about 3 times a week. Dinner - Stewed or roast meat, pork once a week, roasted or boiled potatoes. Cabbages, turnips or carrots. Rice pudding and jam twice a week. Tea - Tea, bread, butter and jam. Supper - Bread and cheese, butter and cocoa. Cold meat or sausages

with tinned beans. No fresh fruit provided in the diet.
All vegetables are well heated.

Case 52. - J.R. 21 years. On a destroyer for 5 months.
On admission. Temp 98.6°. Pulse 80/min. Bowels open regularly every day. Tourniquet test negative. Urine - N.A.D. Swab from palatal ulcers "positive".
Blood Count. R.B.C. 4,100,000/c.mm. Hb. 88%. C.I. 1.07. W.B.C. 13,600./c.mm. Polymorphs. 80%. Lymphocytes 15%. Monocytes 4½%. Eosinophils ½%. Basophils 0%.
Urinary vit. C. estimation. 1st spec. 3.56 mg. %. 3 hrs. spec. 4.21 mg. %. 6 hrs. spec. 4.87 mg.%. (Vit. C. deficiency).
No nicotinic acid estimation done.
Complaint and history.- First felt a rough area with his tongue on the roof os his mouth a week ago. Area became tender and painful and it hurt him to chew. His gums have been bleeding lately on brushing his teeth. For the last five days he has been on nicotinic acid tablets (1,4 i.d.), but the pain is no better. He has had no other symptoms - no diarrhoea, lung or gastric trouble and no trouble with his eyes or eyesight. He neither smokes nor drinks alcohol.
O.E. Heart, lungs, abdomen, eyes and C.N.S. - N.A.D. Several small ulcers are present at the back of the hard palate and behind the upper central incisors. Filmy white membrane on palate and over ulcers. Edges of ulcers sharply defined and bright red. Tonsils clear and no glossitis. Gums swollen and red and bleed on pressure, but no evidence of ulceration or membrane on them. Caries of R. upper molars and tartar between teeth bases.
No adenitis palpable.
Course. Day 1. Full diet and bed. Routine scaling, etc. of teeth. Temporary carbolised dressing to carious molar. 0.3 gm. N.A.B. intravenously. Glycerine and arsenic paint to ulcers and gums. Nicotinic acid - 50 mg. 4 i.d.
Day 3. Less pain in the palate. Still some membrane on the ulcers.
Day 5. Ulcers visibly healing. Gums show no sign of infection but are still rather red.

Day 7. Ulcers healed and membrane gone from palate. "Negative" swabs in the morning before treatment. Gums firmer.
Day 8. Discharged to duty.
Diet. Same as case 27. No fresh fruit available for several months and all vegetables about a week old and well cooked.

Case 53. R.J. 27 years. Drifter for 8½ months.
On admission. Temp. 98.2°. Pulse 70/min. Bowels open once a day. Tourniquet test positive. Urine - dark straw colour. S.G. 1026. Acid. No bile, blood, sugar or albumen. Gum swab positive. Blood count. R.B.C. 3,860,000/c.mm. Hb. 72%. C.I. 0.95. W.B.C. 12,500. /c.mm. Polymorphs. 76%. Lymphocytes 20%. Monocytes 4%. Eosinophils 0%. Basophils 0%.
Urinary vit. C. estimation. 1st spec. 3.81 mg.%. 3 hrs. spec. 3.96 mg.%. 6 hrs. spec. 4.75 mg.%. (Vit. c. deficiency).
No nicotinic acid estimation done.
Complaint and history. Bleeding and tender gums for 3 weeks. Consulted Naval Dentist 2 weeks ago and was given glycerine and thymol m.w.s 3 i.d. and nicotinic acid (50 mg. 4 i.d.);bleeding in gums has continued and pain is no better. He is now afraid to eat solid food and he has a bad breath and foul taste in his mouth in the morning. No diarrhoea, lung or stomach trouble. No trouble with his eyes and has not noticed undue difficulty in seeing at night lately. No history of vague aches and pains in the limbs. He both smokes and drinks heavily.
O.E. Thin, pale patient. Breath offensive. Gums red, swollen and tender. Bleed easily on rubbing. Red, tender, spreading area in R. upper gingivo-labial sulcus. Ulceration up behind central incisors and in R. retro-mular sulcus. Tonsils free and no glossitis. Tender R. cervical adenitis. No xerophthalmia. Heart, lungs, abdomen and C.N.S. - N.A.D. Teeth - Some caries at gum margins and thick encrusted tartar, overlapped by swollen gums.
Course. Day 1. Thorough scaling, rotary brushing and spraying with Eusol. Daily gum paints with 10% chromic acid. 0.45 gm. N.A.B.

intravenously. Mist. ferri et. ammon. cit. 1 oz. t.d.s.

Day 4. Less pain in the gums. Still swelling and redness of gums, however.

Day 6. Gums less fiery. Still some membrane in R. retro-molar sulcus and in R. upper labio-gingival furrow. Continue daily chromic acid paints and m.w. Eusol 4 i.d.

Day 8. Gums have shrunk markedly. No pain on rubbing now. Still an area with membrane on R. upper jaw. Area behind upper central incisors is clear. Interdental papillae normal in size.

Day 10. Gums normal, apart from a small area covered with membrane on upper jaw on R. side. Continue chromic acid and Eusol.

Day 12. Discharged with all areas clear of membrane and gums firm and healthy.

Diet.- Breakfast - fresh fish twice a week. Liver or bacon and eggs every other day. Porridge twice a week. Bread, butter, tea, and tinned milk. Dinner - Roast or stewed meat, cottage pie or bully beef, roast or boiled potatoes, one other vegetable - cabbage, tinned carrots or dried, steeped peas. Rice pudding or plum duff occasionally. Tea - Bread, butter, jam, biscuits and tea. Supper - Cheese and eggs, bully beef and tinned beans, or sausage and mash.

Case 54. S.W. 33 years. Tanker for 4 months.

On admission. Temp. 99.2°. Pulse 86 /min. Bowels constipated. Tourniquet test negative. Urine - dark in colour. Specific Gravity 1020. Alkaline. No bile, blood, sugar or albumen. Throat swab positive. Gum swab positive.

Blood count. R.B.C. 4,980,000/c.mm. Hb. 98%. C.I. 1.0. W.B.C. 9,800/c.mm. Polymorphs. 59%. Lymphocytes 34%. Monocytes 5%. Eosinophils 1%. Basophils 1%.

Urinary vit. C. estimation. 1st spec. 1.85 mg. %. 3 hrs. spec. 2.07 mg. %. 6 hrs. spec. 2.36 mg.%. (Vit. C. deficiency).

No nicotinic acid estimation done.

Complaint and history. He developed aching gums and bleeding

gums 10 days ago and consulted 8 days ago as an outpatient. He was given no local treatment but Eusol m.w.s 3 i.d. and nicotinic acid - 50 mg. 4 i.d. No improvement resulted. Two days ago his throat became sore and he experienced difficulty in swallowing. He is constipated and has had no diarrhoea. No "rheumatism", nyctalopia or lung or stomach troubles. He smokes 20 cigs. per day and drinks in moderation.

O.E. L. tonsil swollen and ulcerated on its upper pole. White membrane with red areola present there. R. and L. shotty, tender, cervical adenitis. Ulcer with white membrane on left, anterior pillar of the fauces. Gums swollen and tender. They bleed copiously on instrumentation. Ulcerated gum margins at the bases of the lower pre-molar teeth. No tartar but deep brown staining at teeth bases and a large hollow, carious, L., lower molar exists. Heart, lungs, abdomen, C.N.S. and conjunctivae - N.A.D.

Course. - Day 1. Thorough scaling, rotary brushing with pumice stone and Eusol spray. Daily gum paints with 10% chromic acid. 0.45 gm. N.A.B. intravenously. Garg. pot. chlor. 4 hourly. Carbolised dressing to carious molar.

Day 2. Pain in throat gone. Tenderness in gums persists. Temp. normal.

Day 4. Ulcer getting smaller on L. tonsil. Gums I.S.Q.

Day 6. Practically no membrane on tonsil. Gums less red and firmer. Membrane on left,anterior, faucial pillar persists.

Day 7. Tonsils clear of membrane. No pain in throat or gums.

Day 9. Very little membrane remains on gums and ulcers at base of teeth have healed. Breath fresh.

Day 11. Gums appear healthy. Glassy appearance on outer margins has gone.

Day 12. Discharged.

Diet.- His diet was monotonous and he complained of the lack of greens and absence of fruit. Breakfast - Steak or ham and eggs. Tinned herring once a week. Bread, butter, jam and tea. Dinner - Soup every day. Meat, potatoes and tinned vegetables

(peas, carrots or parsnips). Pudding twice a week. Tea - Cake, biscuit, bread, butter, jam and tea. Supper - Soup, meat or fish and potatoes. Savoury and coffee.

Case 55. L.S. 23 years. Destroyer A for 7 months.
On admission. Temp 98.4°. Pulse 66/min. Bowels open once a day. Tourniquet test negative. Urine - clear. S.G. 1010. Faintly acid. No bile, blood or sugar. Very faint trace of albumen. Gum swab positive. (Mostly fusiform bacilli).
Blood count. R.B.C. 5,250,000/c.mm. Hb. 98%. C.I. 0.94. W.B.C. 8,000/c.mm. Polymorphs 66%. Lymphocytes 26%. Monocytes 6%. Eosinophils 1½%. Basophils ½%.
Urinary vitamin C estimation: 1st specimen 2.74 mg.%. 3 hrs. spec. 3.85 mg.%. 6 hrs. spec. 4.61 mg.%. (Vitamin C deficiency). No nicotinic acid estimation done.
Complaint and history. Consulted dentist as an out-patient with tooth-ache two weeks ago. Was tried on nicotinic acid as he was seen to have a moderate degree of Vincent's infection. Nicotinic acid (mg.50, t.i.d.) was given and glycerine and thymol m.w.s. His gums have not improved and still bleed on rubbing or on chewing hard food. General gum aching persists. He has never had epistaxis, diarrhoea, "rheumatism" or stomach trouble. He has lately had bronchitis. He smokes 15 cigs. per day and is t.t.
O.E. Gums are covered with a filmy white membrane and the breath is offensive. Interdental papillae are prominent and an ulcer is present in the R. retro-molar sulcus. R. tender cervical adenitis is present. No glossitis. Teeth have hard, encrusted tartar overlapped by the swollen gums. Some slight caries is present too. Heart, abdomen, C.N.S. and eyes - N.A.D. Lungs - R.M. vesicular but scattered rales are heard on deep breathing in all areas.
Course. Day 1.- Thorough scaling of encrusted tartar, rotary brushing, and Eusol (1 in 4) spraying. Daily gum paints with 10% chromic acid. M.W. pot. permang. 4 i.d. Bed. Mist. pectoralis 1 oz. t.d.s.

Day 3. Gums still swollen and tender.

Day 5. Less pain in the gums. Ulcer in retro-molar sulcus is shrinking and healing. White membrane persists in labio-gingival sulci above and below.

Day 8. Ulcer in R. retro-molar sulcus almost entirely healed. Disease is confined mostly to a strip along the outer side of the upper gum on the L. side. Thorough daily paints with 10% chromic acid. Start m.w. Eusol (1 in 4) 4 i.d.

Day 10. Chest clear. Memrbane and redness disappearing from all areas of gums. Gums of a normal pink colour, and have lost their fiery, red look.

Day 12. Mouth appears healthy. No membrane in the morning.

Day 13. Discharged.

Diet. - See case 17.

FINIS.