The effects of sulphone treatment on the campaign against leprosy, the supply of sulphones, and the need of suitable personnel are discussed.

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TROPICAL EOSINOPHILIA IN EAST AFRICA

BY

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The syndrome now generally known as tropical eosinophilia or eosinophilic bronchitis was first described by Frimodt-Møller and Barton (1940) in Southern India. Under the title "a pseudotuberculosis associated with eosinophilia" they published an account of a syndrome characterized by cough, fever, and loss of weight which ran a prolonged but comparatively benign course. There was a fairly constant similarity in the radiographs of these patients, the characteristic picture showing extensive mottling of both lung fields, resembling somewhat miliary tuberculosis or silicosis. Blood counts revealed a high degree of eosinophilia, and in over half the patients examined 5,000 or more eosinophils per c.mm. were found.

Weingarten (1943) recorded 81 cases of tropical eosinophilia. He found that expiratory dyspnoea was a prominent symptom and that the spleen was often enlarged. After trying various remedies without success he discovered that dramatic improvement resulted from a short course of neoarsphenamine. Further cases were described in India by Simeons (1943), Treu (1943), Menon (1945), and others, while a similar condition was reported from Ceylon by Carter, Wedd, and d'Abrera (1944), Carter and d'Abrera (1946b), and Soysa and Jayawardena (1945). The Ceylon workers discovered mites of various genera in the sputum of more than half their cases, and considered that tropical eosinophilia might "in part at least be explained on the basis of a mite infestation of the respiratory system." Treatment by inorganic arsenicals was found to be effective with neoarsphenamine and "acetylarsan" by injection, and acetarsol or carbarsone by mouth.

While tropical eosinophilia can no longer be regarded as a rare disease in India and Ceylon, its existence in Africa has not yet been generally recognized. Isolated cases have, however, been recorded by Parsons-Smith (1944) and Stephan (1946) in Cairo, while Ritchie (1944) described the condition in a Tanganyika native serving in the Middle East Forces and Hunter (1946) in a European recently returned from West Africa. The present communication describes an investigation carried out in Dar-es-Salaam, Tanganyika. Differential white counts were performed on a consecutive series of 34 patients attending the native civil hospital with symptoms of recurrent bronchitis or asthma. The patients were all males, and consisted of 29 Indians, four Africans, and one Arab. In 28 cases an eosinophilia of less than 20% was found, while in six cases the eosinophilia ranged from 40 to 78%. The latter group, and a seventh case which was referred on account of an unexplained eosinophilia, were submitted to detailed investigation. All were considered to be suffering from tropical eosinophilia;

six were treated successfully with arsenic, while the condition regressed spontaneously in the seventh case.

Clinical Findings

The characteristic picture appeared to be recurrent attacks of cough, wheezing, and low fever separated by intervals in which the only symptom was a spasm of unproductive coughing in the early hours of the morning. In four cases retrosternal pain was present, and three complained of acute irritation of the larynx and upper respiratory tract. After a variable number of attacks the intervals of freedom became shorter, and cough and dyspnoea caused increasing distress. Early cases showed no constitutional symptoms, but those who had been ill for several months complained of lassitude, anorexia, and loss of weight. Case VII conformed to the clinical type described by Frimodt-Møller and Barton as "pseudotuberculosis." That patient complained of chronic cough, pain in the chest, and occasional small haemoptyses without asthmatic attacks.

X-ray Findings.—In three cases no abnormality was seen. In Cases VI and VII there was diffuse mottling of both lung fields, characteristic of the "eosinophil lung pattern," and a suggestion of mottling was seen in the radiographs of Case IV. These cases gave a history of one year's, two years', and ten months' duration respectively.

Pathological Findings.—(a) *Haematological.*—An initial blood count was done on all cases by Dr. Vivarelli. The red cells varied from 4,650,000 to 5,150,000 per c.mm. and the haemoglobin from 94% to 110% (Lovibond)—comparatively high figures in a country where anaemia is prevalent. Leucocytosis was a constant finding, counts ranging from 11,250 to 34,000 cells per c.mm., with an eosinophilia of 52–78%. Except for an occasional eosinophil metamyelocyte no immature cells were seen; the granules varied in size and tended to be larger than normal; they frequently did not fill the cell, and left spaces like vacuoles in films stained by the Leishman method. Whitby and Britton (1942) noted a similar appearance of the cells in eosinophilic leukaemia. Differential counts were repeated two or three times weekly on 200–400 cells, and the percentage of vacuolated cells was seen to diminish in the course of treatment. The blood sedimentation rate was estimated by Westergren's method in three cases; in one case it was raised to 47 mm. after 1 hour, but in the other two cases it was within normal limits. Films from all patients were examined for filariae, but none were found.

(b) *Urine and Stool Examinations.*—No abnormality was found on naked-eye or microscopical examination of the urine, and specimens of stools were negative for parasites, except in Cases V and VI, in which a few ankylostome ova were discovered by concentration methods. In both cases a rise in the total eosinophils was found when the blood was re-examined after deworming.

(c) *Sputum.*—Specimens were repeatedly examined for tubercle bacilli, but none were found. Sputum from Cases II–VI was examined by Dr. Vivarelli for mites by the method described by Soysa and Jayawardena. Twenty-four-hour specimens were collected into sterile stoppered bottles, and the mucopurulent material was disintegrated with 10% potassium hydroxide. After it had cleared, enough formalin to give a 10% concentration was added and the liquid left for 24 hours. It was then centrifuged and the deposit systematically examined under the microscope. Four mites of *Tyroglyphus* and a few ova were found in specimens from Case II, one mite of *Tyroglyphus* in Case VI, and an unidentified mite in sputum from Case IV. No mites were found in sputum from Cases III and V.

Treatment

Case II was treated with weekly injections of neoarsphenamine; in the other cases it was intended to follow the method advocated by Soysa and Jayawardena. They gave carbarsone or acetarsol 0.25 g. twice daily until clinical cure had been obtained and the eosinophils had fallen to a satisfactory level. Using this method, it was found that a Jarisch-Herxheimer type of reaction occurred between the second and fifth days. This consisted in a brisk exacerbation of symptoms, usually accompanied by some rise in temperature. After two or three days rapid clinical improvement began, with complete and apparently permanent disappearance of symptoms during the next 5 to 10 days. The haematological response was much slower, and the original intention of treatment by a prolonged course of acetarsol was abandoned in favour of a 10- or 12-day course of acetarsol or carbarsone followed, after an interval, by weekly injections of neoarsphenamine. It was found that there was a fairly rapid fall in the eosinophils during the first four or five weeks of treatment, but after this no significant fall occurred until after treatment had been discontinued. Case V was given a 10-day course of carbarsone and four injections of neoarsphenamine, treatment being abandoned when the eosinophil count was still 29%. Two months later the eosinophils had fallen to 4% (a total of 248 per c.mm.). Case I was of equal severity and Case III was practically asymptomatic. Both were given considerably more arsenic without a more rapid clinical or haematological response being obtained.

In Case II, which was treated with neoarsphenamine alone, the clinical response was slower, but the haematological response was approximately the same as in the other cases.

Case I

An Indian aged 24 had had a cough, retrosternal pain, and a low fever for two to three weeks which were not improved by sulphapyridine and expectorants. On June 2, 1945, the temperature was 99° F. (37.2° C.), the blood pressure 135/95. Sonorous rhonchi were heard in all areas of the chest and moist rales at both bases. A blood count showed: red cells, 5,350,000; haemoglobin, 110%; colour index, 1.02; white cells, 34,000 (polymorphs, 7.5%; lymphocytes, 24.5%; eosinophils, 66%; eosinophil metamyelocytes, 2%). Most of the eosinophils appeared vacuolated. In three specimens of sputum tubercle bacilli were not found; nothing abnormal was observed in the urine, and no ova or parasites were present in three samples of stools. A skiagram of the chest revealed no abnormality.

On June 4 treatment was begun with acetarsol 0.26 g. twice daily by mouth. On the third day there was a sharp rise in temperature which lasted three days and was followed by rapid clinical improvement. By June 17 he was symptom-free, and ten days later his chest was entirely clear. On June 28 acetarsol was discontinued after 50 tablets (13 g.) had been taken. His white blood cells numbered 11,000 (37% eosinophils). In the course of the next six weeks he was given carbarsone, 0.5 g. daily for five days, followed by four weekly injections of neoarsphenamine (one of 0.6 g. and three of 0.75 g.). On Aug. 18 his white count was 9,600 (19% eosinophils). On Oct. 23 it was 7,176, with no eosinophils. He remained in good health.

Case II

An Indian aged 18 had had recurrent attacks of cough for 12 months. Originally dry, the cough had become productive in the last six months, and later he had coughed up large quantities of mucopurulent sputum. Retrosternal pain and increasing dyspnoea had caused him considerable distress, and he had been unable to work for several months. He had had in-patient and out-patient treatment without improvement.

Examination on June 4 showed him to be a thin and dejected individual, very distressed by continuous dyspnoea and constant spasms of coughing. The temperature was 99° F. (37.2° C.). Chest expiration was prolonged and loud sonorous rhonchi were

heard all over both lungs. The spleen was enlarged 3½ in. (8.9 cm.) below the costal margin. The Wassermann reaction and Kahn test were negative. A skiagram of the chest revealed no abnormality. Examination of the urine and stool showed nothing abnormal. Tubercle bacilli were not found in five specimens of sputum. A blood count showed: red cells, 4,660,000; white cells, 13,750 (P., 25%; L., 19%; E., 56%). A sternal puncture gave the following readings: neutrophil polymorphs, 15%; eosinophil polymorphs, 35.2%; basophil polymorphs, 1%; neutrophil metamyelocytes, 5%; eosinophil metamyelocytes, 18%; neutrophil myelocytes, 1.2%; eosinophil myelocytes, 8%; neutrophil premyelocytes, 0.4%; eosinophil premyelocytes, 1%; eosinophil myeloblasts, 1%; normoblasts, 7.2%; erythroblasts, 1.2%; macropolycytes and megakaryocytes, 0.6%; mitotic cells, 0.4%. The blood sedimentation rate (Westergren) was 47 mm. after one hour, 56 mm. after two hours.

The patient was given eight weekly injections of neoarsphenamine (0.15 g., 0.3 g., 0.6 g., and five of 0.75 g.) beginning on June 7. Four mites of *Tyroglyphus* were found in a 24-hour specimen of sputum collected after the first injection had been given, and a few ova were seen in a specimen collected a week later, but no further adults or larvae. There was an increase in symptoms after the third and four injections, but his clinical condition then steadily improved. By July 28 he was asymptomatic and gaining weight, his total leucocyte count had fallen to 8,000, with 16% eosinophils, and his B.S.R. to 26 mm. after one hour.

He was seen again in August, when he complained that he was still getting attacks of asthma. He was repeatedly examined, but no abnormality was found in his chest, and his eosinophils had fallen to 10%. Arrangements were made to readmit him to hospital for observation, but he failed to return.

Case III

An African clerk aged 25 was first seen on May 22, when a large number of eosinophils were found in a blood film while he was suffering from an attack of malignant tertian malaria. He was otherwise well except for a slight dry cough. The white cells numbered 8,000 (P., 26%; L., 19%; M., 1%; B., 2%; E. 52%). Nothing abnormal was found in the urine, and no ova or parasites were present in the stools on seven consecutive days. The white cell count on June 6 was 15,800 (P., 10%; L., 18%; M., 0.5%; B., 1%; E., 69%; eosinophil metamyelocytes, 1.5%)—15% of the eosinophils showed cytoplasmic vacuolation. The B.S.R. (Westergren) was 9 mm. after two hours.

Clinical examination was negative except for a slight splenic enlargement which might have resulted from the malaria. He was given 0.26 g. of acetarsol twice daily for 15 days, starting on June 14. After two days his cough increased and a small quantity of sputum was expectorated. This was collected and examined for mites, but none was found. He was entirely free from cough three days later, but his eosinophils still numbered 46% at the end of the course. After two weeks' interval he was given six weekly injections of neoarsphenamine (one of 0.6 g. and five of 0.75 g.). On July 19 the blood count showed 6,200 white cells, with 12% eosinophils. On Oct. 18 his white cells numbered 6,000 (P., 51%; L., 40%; M., 2%; B., 1%; E., 6%). He has remained in good health.

Case IV

An Indian clerk aged 32 complained of recurrent attacks of dry cough for 12 months. The cough usually woke him in the early morning. In the past four months attacks had grown more frequent and more severe, and expiratory dyspnoea had become increasingly troublesome. Treatment had not given him any relief.

On examination on June 11 expiration was prolonged and a few rhonchi were heard at the right base and a patch of inspiratory rales at the left base. The white cells numbered 11,250 (P., 15%; L., 7%; E., 78%). The B.S.R. (Westergren) was 5 mm. in one hour. Examination of the urine and stools revealed no abnormality. A skiagram of the chest showed faint mottling of both lung fields. A course of acetarsol 0.26 g. twice daily was begun on June 12. Three days later his symptoms became so severe that he abandoned treatment for four

days, resuming it on June 19. Sputum was collected at the beginning of the exacerbation and one unidentified mite was found on microscopical examination. The following day he was symptom-free, and three days later his chest was clear on clinical and x-ray examination. Treatment was discontinued after a total of 50 tablets (13 g.) had been taken, the eosinophils at this time numbering 21%. In view of his high eosinophil count he was given five weekly injections of neoarsphenamine, starting on June 12 (0.6 g. followed by 0.75 g.). On July 28 his white cells numbered 8,600, with 13% eosinophils, and on Oct. 23 they had fallen to 6,240 (P., 85%; L., 10%; M., 3%; E., 2%). He remained fit, and stated that he was now feeling thoroughly well for the first time for over 12 months.

Case V

An African cook aged 24 complained of continuous dry cough since an attack of acute bronchitis a month previously. During the last two days his acute symptoms had recurred with cough, wheezing, and fever. On examination the temperature was 99.8° F. (37.7° C.). Numerous rhonchi were heard all over the chest, and a patch of leathery rales at the left base. Neither tubercle bacilli nor mites were found in the sputum. The urine was normal. A few ankylostome ova were found in the stools by concentration methods. A radiograph of the chest revealed no abnormality. The white blood cells numbered 14,600 (P., 20%; L., 15%; M., 1%; B., 1%; E., 63%). He was dewormed with chenopodium and tetrachlorethylene, and a blood count on July 9 showed: red cells, 4,650,000; haemoglobin, 94%; colour index, 1.01; white cells, 20,000 (P., 11%; L., 25%; M., 2%; E., 62%). Treatment was begun on July 9 with carbarsone 0.25 g. twice daily for 10 days. On the fifth day the cough and sputum increased and the eosinophils rose to 83%. All sputum was systematically searched for acari, but none was found. By July 19 he was symptom-free and his chest was clear, but his eosinophils numbered 75%. He was given three injections of neoarsphenamine (0.6 g., 0.75 g., and 0.75 g. at weekly intervals). The eosinophils fell to 29% at the end of treatment, and on Oct. 24 the white cells numbered 6,240 (P., 65%; L., 25%; M., 6%; E., 4%). He stated that he had had no cough since the beginning of the treatment and that his general health was better than it had been for many months.

Case VI

An African night-watchman complained of continuous dry cough with recurrent acute attacks of cough, wheezing, and pain in the chest for the last ten months. He asked to be admitted to hospital, as an acute attack had begun two days previously.

When examined on July 10 his nutritional state was poor and there was marked dyspnoea, with rhonchi all over both lungs. The urine was normal. A few ankylostome ova were found in the stools by concentration methods. A blood count showed: red cells, 4,590,000; white cells, 8,750 (P., 30%; L., 30%; E., 40%). He was dewormed with chenopodium and tetrachlorethylene, and a blood count on July 28 showed: red cells, 4,650,000; haemoglobin, 95%; colour index, 1.02; white cells, 19,600 (P., 11%; L., 13%; M., 1%; E., 75%). A skiagram of the chest showed mottling of both lung fields typical of the "eosinophil lung pattern." Treatment was begun on July 28 with acetarsol 0.26 g. twice daily for 12 days. On the fifth day he became extremely dyspnoeic and his temperature rose to 101° F. (38.3° C.). Sputum was examined during the acute phase and one mite (*Tyroglyphus*) was found. By Aug. 6 the patient was symptom-free and his chest was clear. The course of acetarsol was followed after a short interval by eight weekly injections of 0.6 g. of neoarsphenamine. Throughout treatment an eosinophilia persisted, counts varying between 28% and 50%, but on Oct. 28 his white cells numbered 8,250 (P., 50%; L., 44%; M., 3%; E., 3%), and skiagrams showed considerable clearing of the mottled pattern. Two months later he was still fit and had had no recurrence of symptoms.

Case VII

An Arab stone-mason aged 30 complained of chronic cough with occasional small haemoptyses. The first attack began 2 years 3 months before and had lasted 10 months. It was

followed by 10 months' freedom; then the present attack began 7 months previous to admission.

When examined on July 31 he was afebrile, expiration was slightly prolonged, and there were numerous coarse rales at both bases. Tubercle bacilli were not found in three specimens of sputum, and no parasites were discovered in three stool specimens. A skiagram of the chest revealed diffuse mottling of both lung fields typical of the "eosinophil lung pattern." A blood count showed: red cells, 4,950,000; haemoglobin, 98%; colour index, 0.99; white cells, 22,300 (P., 16%; L., 20%; M., 1%; E., 62%; eosinophil metamyelocytes, 1%). He was not seen again until Oct. 18, when he was traced and re-examined. It was found that his condition had again regressed spontaneously. A skiagram of his chest revealed no abnormality and white cells numbered 9,360 (P., 70%; L., 20%; M., 6%; E., 4%).

Discussion

The finding of six cases of tropical eosinophilia in a consecutive series of 34 patients suffering from chronic cough or asthma suggests that this condition is relatively common in the Dar-es-Salaam district. All the patients had resided on the Tanganyika coast for the previous two years, and none of them had been out of East Africa for eight years. The only features common to all cases were: a history of less than three years' duration, cough of variable degree, leucocytosis, and pronounced eosinophilia. In most cases there was no obvious symptom of physical sign to distinguish them from bronchial asthma or recurrent bronchitis, and it must be emphasized that unless a differential white count is done in the Tropics as a routine on all such cases tropical eosinophilia will constantly be missed.

The aetiology of tropical eosinophilia still remains obscure. It has been found at all ages and in both sexes. Weingarten stresses the importance of environment, finding it common on the coast round Bombay, but absent from the inland district of Rajputana. Although cases have since been recorded from inland districts the majority seem to have contracted the disease in a hot and humid atmosphere. In Dar-es-Salaam the mean temperature varies between 74° and 82° F. (23.3° and 27.8° C.), with a relative humidity of 60 to 90%.

Löffler (1936) described a syndrome characterized by fever, cough, and eosinophilia which resembles tropical eosinophilia but is essentially an illness of short duration in which rapid spontaneous cure occurs. The pulmonary infiltrations described by Löffler were transient ("flüchtige Infiltrate"), seldom lasting more than eight days, and were often confined to one lung. In tropical eosinophilia there is a tendency towards spontaneous cure, but if untreated the condition is apt to persist for several years. It is unlikely, therefore, that the two conditions are identical.

Vaidya (1943) and Lal (1945) do not consider the condition to be a separate disease entity, but think that it should be regarded as a form of asthma with a pronounced eosinophilic response. The relation between tropical eosinophilia and asthma is clarified by regarding asthma or bronchospasm as a symptom common to several diseases, of which this condition is one. Tropical eosinophilia differs from the primary allergic group of disorders in the absence of a family history and in the presence of fever, leucocytosis, splenomegaly, and characteristic shadows in the skiagrams of the chest. Moreover, arsenic is not particularly useful in the treatment of bronchial asthma, and no response was obtained when it was tried in a few of the cases with low eosinophil counts.

As Menon (1945) showed, tropical eosinophilia bears a fairly close resemblance to infectious mononucleosis, and both its clinical appearances and its response to treatment favour the theory that it is a specific infection. Whether

or not the *Acarus* is the infective agent has not yet been satisfactorily proved. The finding of ova and larvae in sputum by Carter and his colleagues and of ova in the present series strongly suggests that the mites are living in the respiratory tract either as commensals or as the direct or indirect cause of the associated symptoms. Carter, Wedd, and d'Abrera (1944) have demonstrated that the mite *Tyroglyphus longior* is killed by exposure to arsenious oxide (1:1,000) for ten minutes. Carter and d'Abrera (1946a) have also produced a syndrome resembling tropical eosinophilia by introducing ova of tyroglyphid mites into the trachea of a toque monkey. Against the theory that the *Acarus* is the infective agent it may be argued that the mites found are not of any specific genus, nor did Carter and his colleagues find that they had entirely disappeared after the patient had been successfully treated. Further research is clearly necessary on this subject, particularly the examination of sputum from patients in the Tropics suffering from pulmonary disorders unassociated with eosinophilia.

Treatment with inorganic arsenic is dramatically successful. Until the aetiology of this condition is understood, and until more cases have been followed up, the minimum effective course of treatment cannot be determined. Vaidya and Lal both record cases which relapsed owing to inadequate treatment, and Soysa and Jayawardena found that the length of treatment should be determined by the blood picture and not by the pulmonary symptoms. In the present series treatment was probably continued in most cases longer than necessary. The most effective method appeared to be a ten-day course of carbarsone followed after a few days by four weekly injections of neoarsphenamine (0.6–0.75 g.). This was sufficient to produce complete clinical cure, and further treatment seemed to delay rather than accelerate the fall in the eosinophil percentage. No untoward reactions occurred, nor did any case relapse during the three months in which cases were followed up.

Summary

Seven cases of tropical eosinophilia are described from Dar-es-Salaam, East Africa.

Six of them were successfully treated with arsenic; the seventh regressed spontaneously.

It is thought that this condition must be relatively common on the coast of Tanganyika, and probably elsewhere in tropical Africa.

I wish to express my indebtedness to Dr. F. Vivarelli, who undertook most of the pathological investigations described, and to Dr. J. H. Macdonald for his assistance in following up the cases. My thanks are due to the Acting Director of Medical Services, Tanganyika, for permission, to publish this paper.

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Information on French spas is now available and may be obtained from the French National Tourist Office, 179, Piccadilly, London, W.1.

CAUSALGIA OF THE FACE TWO CASES SUCCESSFULLY TREATED BY SYMPATHECTOMY

BY

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While causalgia most often follows injuries to the median nerve and the medial division of the sciatic nerve, other nerves are sometimes similarly affected. I saw two cases of causalgia of the face, and as such cases are unusual it seems worth while to place them on record. Because of the good results obtained from sympathectomy in many cases of causalgia following injury to other nerves, it was thought that this line of treatment might relieve the facial pain in these cases. As will be seen from the case histories, interruption of the sympathetic chain proved successful.

Case I

An Indian N.C.O. aged 28 was wounded by shell fragments on Feb. 28, 1944. He sustained a small penetrating wound of the right cheek and a severe compound fracture of the right leg, for which an above-knee amputation was performed. Burning pain on the right side of the face developed on the day he was wounded and was still present when he was seen a year later. It was continuous, was felt in the area of distribution of all three divisions of the trigeminal nerve, and was strictly confined to this area. It became worse with warmth—when in the sun or on a very hot day—with eating, or with much talking. Marked hyperalgesia of the skin supplied by the three divisions of the trigeminal nerve was present. In the centre of the right cheek was the scar of a small penetrating wound. X-ray examination revealed a small metallic foreign body under the base of the skull in the region of the foramen ovale.

Paravertebral Procaine-alcohol Injection.—A procaine block of the stellate ganglion was produced by the paravertebral injection of 2 ml. of procaine on March 17, 1945. Within a few seconds, no longer than was required for the procaine to produce a nerve block, the facial pain was completely relieved. A further 3 ml. of procaine was injected and was followed by 5 ml. of absolute alcohol. The alcohol, unfortunately, produced a neuritis of the upper intercostal nerves, with pain in the back and front of the chest. This was fairly severe at first and lasted for several weeks. Three weeks after the injection the facial pain and tenderness returned, and the pain soon became severe. On examination it was found that the sympathetic block had been only temporary and that sweating had returned on the left side of the face. It was decided to excise the superior cervical sympathetic ganglion.

Operation.—On April 22, 1945, under local analgesia, the right superior sympathetic ganglion was exposed through a 3-in. (7.6-cm.) incision along the posterior border of the sternomastoid muscle. The sympathetic chain was divided below the ganglion, the superior cardiac nerve and communicating rami from the ganglion to neighbouring nerves were cut, and the lower half of the ganglion excised. The facial pain and tenderness were again immediately relieved, and there was no sign of their return when the patient was last seen three months later.

This patient also suffered from phantom pain felt in the amputated foot. This, however, was only slightly painful and he did not wish to have anything done for it.

Case II

An Indian N.C.O. aged 23 sustained multiple mortar wounds on April 1, 1944. There were several small penetrating wounds on the left side of the face and head, the left arm, and the left shoulder, and an extensive wound of the left thigh. Three days later the left leg was amputated above the knee for gas-gangrene. Pain in the left cheek began three months after the