Among these may be included foreign matter on the surface of the metal, inaccurate placing of the weld on the edge of the sheet of metal, maladjustment of the machine, etc. But even under ideal conditions sparking will occur in a certain number of cases. Such being the case, the problem of prevention is to devise a means of protecting the hands of the worker from these sparks. This might be achieved by one of three methods:

(a) By applying to the machine a guard which would protect the operator's hands or prevent them from being brought close enough to the welding point to be in danger. This would be the ideal method—being permanent and foolproof, and in no way demanding the co-operation of the worker. Unfortunately, no such guard yet conceived will permit of the welding machine being used.

(b) By the use of a jig for positioning the parts to be welded and thus removing the necessity for the operator's fingers to be close to the welding point. This is possible in many cases, but in some is technically impracticable. Also, in practice there is the very real danger that the operator will place ease before safety and discontinue the use of the jig.

(c) By the provision of tongs or protective gloves. This is analogous to the provision of goggles for the protection of the eyes, and possesses the same disadvantages—namely, that through laziness or carelessness on the part of individual operators they will be temporarily or permanently left off. As is well known by all industrial medical officers, protective clothing can easily be devised and provided, but it is most difficult to persuade operators constantly to use it.

It has been found that neither industrial cotton nor leather gloves afford sufficient protection, and we have experimented with various other materials. Those most satisfactory to date are cotton gloves coated with "neoprene" and asbestos fabric finger-stalls worn under the usual cotton gloves. We now have both of these in use, but are continuing our search for the ideal material.

We are grateful to Mr. J. C. Scott, Director of the Accident Service, Radcliffe Infirmary, for his help and encouragement in preparing this paper, and would like also to acknowledge the assistance given to us by the welding and laboratory staff of the Pressed Steel Company.

SYMMETRICAL GANGRENE IN THE AFRICAN

BY

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Gangrene in the extremities can generally be attributed to diabetes, arteriosclerosis, or thrombo-angiitis obliterans. Ergot poisoning is a relatively rare cause. Arteritis resulting in gangrene is described in acute infections, such as typhus and typhoid fevers and malaria. Gangrene has been noted in pellagra or the vitamin B deficiency state (Page, 1946). It is also seen in those cases where the arteries are suddenly occluded by an embolus, subacute infective endocarditis being a typical instance. Occasionally, in Raynaud's phenomenon, there are trophic changes in the tips of the fingers. Traumatic cases, forming a separate group, are also seen. Trench foot and the immersion-foot syndrome following exposure to cold constitute another class of cases in which gangrene of varying extent is the most important feature.

I have encountered gangrene in an extremity in the elderly African, presumably due to arteriosclerosis, and unilateral gangrene, which might have been attributed to syphilis. There appears to be very little written on Buerger's disease in the African. I have not encountered a case in the native. Ferro-Luzzi (1942) reports on 20

cases of Buerger's disease in European males living in Eritrea, but he failed to find the condition among the indigenous population. Goetz (personal communication), however, has seen a case in the African.

During the past four years I have seen six cases of symmetrical gangrene in the African, running a peculiar but nevertheless more or less constant course. In each the course and history were similar. (1) The onset occurred in a person who previously enjoyed more or less good health. (2) The disease was seen only in males. (3) It occurred usually between the ages of 20 and 35—one patient, however, was over 50. (4) The first sign was oedema of both feet; this was followed by pain. (5) The gangrene occurred simultaneously on each side. (6) No obvious cause common to each case could be found for the disease.

In four cases the gangrene was limited to the tips and pads of all the digits. In one the gangrene ascended to the mid-tarsal joints, being exactly symmetrical on both sides. In the sixth case the whole of both feet and up to 3 in. (7.6 cm.) above the ankle-joint on each leg was affected.

All the cases came from Mashonaland, but from widely separated regions. They were seen at different periods over the four years. In none was there anything to suggest that other cases with gangrene had occurred in the villages from where they originated. The patients denied having received any treatment from a witch-doctor or of having consumed any native drugs prior to the onset of the disease. They all stated that they had previously been well. In two of the six cases the Wassermann reaction was positive. In one of them the disease was immediately preceded by cerebral malaria, but in no other case was acute malaria found. No sugar was found in the urine of any of the patients. The blood pressure was normal in each. The clinical features of the two cases in which extensive gangrene occurred were as follows.

Case 1

An African man aged about 30 first became ill on June 11, 1945, with general malaise and shivering. He vomited twice on the first day of the illness. During the night he developed severe headache and passed into coma. He was admitted to the Salisbury Native Hospital from Norton (about 25 miles away) on June 14. The coma continued for two days, after which he gradually regained consciousness (June 15).

On admission the blood smears were positive for *Plasmodium falciparum*, and quinine was given by injection 10 gr. (0.65 g.) twice daily for two days. The cerebrospinal fluid was clear. There was nothing abnormal in his legs. On June 16, the day after his recovery from the comatose state and acute malarial condition, the patient first complained that his legs were heavy to lift. It was noticed that his feet, ankles, and the distal halves of his legs were swollen and showed pitting oedema, but there was no pain. A day or two later his legs and feet were very painful to touch. Several days later it was noticed that the skin of both feet was hard and cold up to 3 in. (7.6 cm.) above each ankle-joint. The feet were discoloured black, this being most clearly seen on the soles and the pads of the toes. Three distinct zones were noted on each limb: The most distal one, from the toe-tips to past the mid-tarsal joints, was pitch-black, due to total gangrene. The middle zone, which reached to about 3 in. above the ankle-joints, was cold and swollen. Above this was a narrow zone, which was warm and faded gradually into the normal temperature of the leg. All the toes were affected by dry gangrene. No pulse could be felt in the anterior and posterior tibial arteries, but that in the popliteal and femoral region was easily palpated and was of equal force in each limb.

The patient was thin. There was no oedema in any part of the body until it appeared in the legs. Jaundice was not detected. The conjunctivae were pale, as was the tongue. The gums were slightly spongy. There were signs of a pellagroid rash

on the hands and feet. Abdominal distension was not present, nor was the spleen or liver palpable. Nothing of note was found on examination of the central nervous system. The cardiovascular system was normal, the blood pressure being 110/80. The Wassermann reaction of the blood and cerebrospinal fluid was negative. No sugar or albumin was present in the urine. The blood count was: red cells, 2,570,000 per c.mm.; haemoglobin, 40%; colour index, 0.78; white cells, 24,500; neutrophils, 74%; lymphocytes, 22%; monocytes, 4%.

Before admission to hospital the patient's diet consisted of maize and meat—quite an ordinary one for an African. Life and food were shared with other labourers in a labour camp, and the patient stated that so far as he was aware none of the other inmates of the camp had been afflicted with his complaint.

The gangrenous areas were treated with dry sterile dressings. However, after ten days moist gangrene set in, and amputation was recommended. The infection spread and the gangrenous area became swollen and offensive, but the patient could not be persuaded to consent to an amputation. He insisted on leaving hospital and was discharged on July 13. Trace of him was then lost.

Case 2

A native youth aged about 16 was admitted to the Salisbury Native Hospital on July 1, 1945, complaining of swelling and pain in both feet. He came from the Enkeldoorn District (about 100 miles away). He was quite well until two weeks previously, when he began to have lumbo-sacral backache. There was no preceding history of malaise, vomiting, headache, consumption of drugs, or eating of food other than that to which he was accustomed. He had not previously visited a witch-doctor. A week after the onset of the backache he noticed that both his feet had begun to swell. The oedema was followed by pain in the feet. The patient was quite certain that the swelling preceded the pain by two days. A week later he noticed that both feet had changed colour, becoming darker and blacker. His diet on the whole was a fairly satisfactory one, consisting of maize daily, meat four times a week, pumpkin three times a day, sweet potatoes every day, monkey-nuts occasionally, and certain wild fruits. He ate worms, caterpillars, locusts of the larger type, and white ants.

On examination he was fairly well covered, with no sign of nutritional disease. The lungs were clear and the heart normal. The blood pressure was 120/60. The spleen and liver were not enlarged. The blood count was: red cells, 3,750,000; haemoglobin, 71%; colour index, 0.94; white cells, 11,300; neutrophils, 68%; lymphocytes, 21%; monocytes, 6%; eosinophils, 5%. The blood Wassermann reaction was positive.

FIG. 1

There was dry gangrene of all the toes and both feet up to the tarso-metatarsal joints, the affected parts being black and cold. The gangrene was symmetrical in extent (Fig. 1). Immediately above the gangrenous area in each limb was a zone of demarcation, 1 in. (2.5 cm.) in depth, above which again was

a band of hyperaemia which gradually faded into the normal temperature of the rest of the limb. The popliteal and femoral pulsations on each side were normal and equal. The dorsalis pedis and posterior tibial arteries could be felt pulsating.

Towards the beginning of August, spontaneous amputation was noticed at the line of demarcation in the left foot, and on Aug. 20 a similar line was seen in the right foot (Fig. 2). Next day the left foot was amputated at the line of demarcation

FIG. 2

in the tarso-metatarsal line, and on Sept. 7 the same operation was performed at the corresponding site in the right foot. The sites of amputation healed soundly and the patient was otherwise perfectly fit. He became anxious to leave hospital, was discharged, and returned to his kraal.

Discussion

The oedema is difficult to explain, since it is not usually a feature of arterial obstruction alone. Oedema in vascular disorders may usually be attributed to venous or lymphatic obstruction, local infection, or perhaps to disease of the capillary walls with resulting increased permeability. An example of the last type may be seen in Buerger's disease (Saint, 1945) or in ergot poisoning. It is possible that the oedema is the primary cause, producing secondary interference with the circulation of the limb, foot, or toes. The oedema appears rapidly in these cases and is of moderate degree. It may have been due to a thiamine deficiency. Case 1 was, in fact, given thiamine, 20 mg. daily, by intramuscular injection as soon as the oedema was noticed. It lasted only a few days and then disappeared, but by that time gangrene had supervened.

It will be seen that the primary cause of the admission of Case 1 to hospital was cerebral malaria, and that the limb disorder followed soon after. It would thus be reasonable to exclude ergot or other drugs which might possibly have been consumed before admission. The limb disorder may, however, be attributed to the severe malarial infection or to a spasm of the arteries in the legs following the administration of quinine.

From the above description it will at once be clear that Raynaud's disease and thrombo-angiitis obliterans are easily ruled out. Raynaud's phenomenon produces diffuse lesions, including small cutaneous trophic lesions with fibrosis in the fingers and toes (Saint, personal communication), but this occurs after a long interval and not, as in the cases described, after a single attack. Raynaud's disease confined to the toes is uncommon, whereas in the present cases the disorder was limited solely to the lower extremities. Thrombo-angiitis obliterans is easily excluded, for there is often a history of intermittent claudication when the muscle circulation is involved or of migratory phlebitis.

It is almost always unilateral to begin with, becoming bilateral sooner or later. Almost all cases have a polycythaemia (Saint, personal communication). An anaemia was first seen in both my cases. The only two points which Buerger's disease has in common with these cases are that the patients were relatively young adults and were all males.

Ergot poisoning has to be considered. There is usually a history of the patient's having taken the drug, or of other cases occurring in the community. In the cases recorded, however, no other sufferers with gangrene were observed in the areas from which they came. In ergot poisoning, in addition to the gangrene, nervous symptoms may appear. In my cases, however, there were no neurological changes. In ergotism the gangrene may affect not only the toes but also the fingers, and is usually delayed for weeks. In my cases the onset was rapid and only the lower extremities were affected.

Exposure to excessive cold may result in gangrenous changes in the extremities. Abramson *et al.* (1946) describe illustrative cases with symmetrical gangrene of the toes and feet bearing a close similarity to the cases described in this paper. Trench foot is usually seen in those compelled to remain in trenches for a prolonged period during which the extremities are exposed to a damp and very cold environment. There was nothing, however, in the history of the present cases to attribute the gangrene to such a cause.

Lewis (1936) describes two interesting forms of symmetrical or bilateral gangrene which bear a great similarity to my cases. The first form is referred to as "bilateral gangrene of the digits in the young," and the second as "bilateral gangrene in the digits occurring in the elderly."

In the former group children and young adults are affected, both sexes equally. The origin of the condition is obscure. It usually affects relatively young people with no previous history of vascular disease. At the time of onset, however, malnutrition or some other condition causing chronic ill-health is present. It may, on the other hand, develop during an acute infection such as pneumonia or typhoid fever. The onset is apparently sudden, without warning, and several fingers or toes are affected. These become discoloured within a few days. Tissue at the tips of the toes is lost, the gangrene usually being dry. Occasionally it may extend to the base of the distal or middle phalanx. The disease is bilateral and often strictly symmetrical. The main arteries are unobstructed. Lewis considers that the condition probably results from damage to the intimal coats of the digital arteries, with resultant thrombosis. My cases appear to differ from these in that they had a preceding history of pain and oedema in the affected areas, and that in two of them the gangrene extended well beyond the toes, thus being more extensive than in the disorder described by Lewis; also, the fingers were unaffected. The similarity between his cases and mine lies in the fact that a single attack with symmetrical distribution occurred in those generally poorly nourished or in a patient during the course of or following an acute condition.

In the second group described by Lewis—bilateral gangrene of the digits in the elderly—an associated disease, such as cancer, tuberculosis, high blood pressure, or diabetes was present with the gangrenous disorder. The fingers often become discoloured without warning and gangrene soon follows. The gangrene is often associated with much pain. Lewis supposes that the vessels show intimal thickening, due to nutritional changes. This leads to multiple arterial obstruction.

It is not possible to give a cause for the cases I saw over the past four years. The exact symmetry of the affection would suggest a toxic disturbance or some general disorder

such as a deficiency disorder. I doubt if ergot was the cause of the gangrene, since one of the cases was admitted with cerebral malaria, and developed the condition in hospital. Quinine, however, was administered to this patient and may have produced a spasm of the vessels.

Pellagra, ariboflavinosis, hypoproteinaemia, and other disorders of nutrition are common in the African. Case 1 showed signs of pellagra. It is possible that the gangrene may have resulted from vascular occlusion, a direct result of a nutritional disturbance in the vessel wall. This possibility is not altogether improbable in view of the reports on the development of gangrene in the extremities seen among prisoners of war in the Far East. Recently Page (1946) reported on the findings on 2,000 prisoners of war from Hong Kong. Among these a certain number with painful feet and signs of deficiency in the vitamin B complex developed gangrenous changes in the toes and feet. Page reports, too, that Prof. Kinoshita of Osaka University studied these vascular changes seen among Japanese soldiers returned from the South Pacific and found definite thickening of the vessel walls with narrowing or obliteration of the tissues of the small arteries of the feet and legs.

Summary

Six cases of gangrene of symmetrical distribution and extent occurring in the African are described. In four the gangrene was limited to the toes, but in two the gangrene was more extensive.

The condition sets in simultaneously on each side, first with oedema, followed by pain and finally gangrene.

My thanks are due to Prof. C. F. M. Saint for kindly examining the two cases described in detail and for his suggestions as to the differential diagnosis. I am grateful to Dr. R. M. Morris, Medical Director, Southern Rhodesia, for permission to publish this paper.

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Medical Memoranda

Acute Infectious Lymphocytosis

Steigman's (1946) account of infectious lymphocytosis has drawn attention to the importance of this condition in children, especially in the differential diagnosis of infectious lymphocytosis and acute leukaemia, and prompts me to report a case of the acute form, as very few have so far been recorded in this country. From a perusal of the literature infectious lymphocytosis would appear to be a disease of recent origin, though before Carl Smith recognized it as a separate disease entity in 1941 it was probably confused with infectious mononucleosis. No cases even suggestive of the condition seem to have been reported before 1939, and, indeed, only a handful of cases were to be found in the American literature, and apparently very few elsewhere, until Finucane and Philips (1944) described an epidemic of 21 cases in a children's sanatorium.

CASE REPORT

The patient, a boy aged 7, first attended the children's department of the London Hospital on Oct. 15, 1946, with a history of diarrhoea for five weeks. There was slight abdominal pain, he passed frequent motions containing a little mucus, and he complained of sweating at night. The only abnormality noted was moderate enlargement of the left cervical glands. Rectal swab and x-ray examination of the chest were negative. On Oct. 25 blood examination revealed a white cell count of 30,600 per c.mm. (75% normal small lymphocytes) and haemoglobin 85%. At the age of 4 he had had whooping-cough and two years later he developed mumps. An uncle had died of pulmonary tuberculosis when the boy was 1 year old, but there was no history of contact.