The statistics for embolectomy, however, do show a reasonable chance of recovery in a person who is otherwise almost certain to lose a limb, and maybe his life. With this in mind it can be said that this form of treatment is the one that should be adopted in all cases of peripheral embolism, especially where the diagnosis is made early.

The recent advent of heparin (Murray and Best, 1938) may increase the chance of recovery still further, although a word of warning may be added here. For the first three days post-operatively the "specified dosage of 4,000 Toronto units" was injected intravenously daily, and at the end of this period the blood-clotting time was still only $3\frac{1}{2}$ minutes at 37°C . As its further use did not seem to be indicated the heparin was discontinued. Its effect in this case was obviously unsatisfactory, and it seems that the dosage advocated will have to be increased, and in addition very careful laboratory control must be used. There is no doubt that this drug is valuable, however, as shown by MacFarlane's (1940) case, in which he performed four separate embolectomies in the same patient with complete success; similarly it had a marked effect on the clotting time of the blood in Hamilton Bailey's (1944) case.

Summary

A brief history of the use of the operation of embolectomy is given, together with the results obtained in the past; a special note about the British cases on record is included.

The aetiology, diagnosis, and treatment of peripheral embolism are discussed.

A further case is added to the records: successful popliteal and femoral embolectomies 39 hours after the lodgment of the emboli.

I would like to express my thanks to Mr. Harold Dodd, F.R.C.S., for permission to record this case.

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The report for 1944 of the Kashmir Medical Mission of the Church Missionary Society has been received from Srinagar. The year is noted as an "absolute record" for this hospital, which, for many years under the guidance of Dr. E. F. Neve, has been regarded as one of the foremost medical missionary institutions in India. Over 48,000 visits of patients were recorded in the out-patient department and 2,707 were admitted to the wards. Though the valley of Kashmir enjoys one of the best climates in the world, the insanitary habits of the Kashmiris have not changed in the passing centuries. Outbreaks of cholera, dysentery, and typhoid persist and make possible the devastating epidemics which from time to time occur. The report is a good record of how war difficulties have been overcome—shortage of staff as also of drugs and dressings.

HIGH-COLOUR-INDEX ANAEMIA DUE TO VITAMIN C DEFICIENCY

BY

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During the spring and summer of 1943 four severe cases of adult scurvy associated with a high-colour-index anaemia of varying severity were admitted to St. Alfege's Hospital. They were all males, aged 70–80, old-age pensioners who were living alone. The diet in each case was markedly deficient in vitamin C. The most severe case ended fatally from heart failure soon after admission, but the other three responded satisfactorily to ascorbic acid. Cases 1 and 3 had hydrochloric acid present in the gastric juice in normal amounts. In the other two the gastric juice was not examined. They all received a mixed hospital diet, but were not given haematinics such as liver, iron, or vitamin B complex; yet the anaemia responded satisfactorily to saturation with ascorbic acid.

Case Histories

Case 1.—A man aged 72 was admitted on Aug. 24, 1943, with extensive bruising and purpura on the backs of his lower limbs. The spontaneous bruising had appeared three weeks previously and was becoming more extensive. He was edentulous, and his gums were unaffected. His diet had consisted almost entirely of bread, margarine, a little jam, and stewed meat, but almost no potatoes or greens. He was anaemic, and the capillary fragility (Hess) was raised. The blood count on admission was: R.B.C., 2.4 millions; Hb (Haldane), 60%; C.I., 1.25; average diameter of R.B.C., 8μ ; reticulocytes, 0.8%; platelets, 144,000 per c.mm.; W.B.C., 5,000 (polymorphs 82%, lymphocytes 10%, monocytes 8%); bleeding and clotting times normal. Hydrochloric acid was present in the gastric juice in normal amounts. He was saturated with 11.2 g. of ascorbic acid intramuscularly in 16 days. The bruising and purpura began to fade in a few days, and rapid improvement resulted. The capillary fragility test became normal in three weeks. The anaemia responded satisfactorily, as shown:

24/8/43: R.B.C., 2,400,000; Hb, 60%; C.I., 1.25; W.B.C., 5,000
 9/9/43: R.B.C., 3,250,000; Hb, 74%; C.I., 1.13; W.B.C., 5,600
 17/9/43: R.B.C., 4,300,000; Hb, 86%; C.I., 1.0; W.B.C., 6,000

The maximum reticulocyte count was on 1/9/43, when it was 1.6%.

Case 2.—A man aged 79 was admitted on July 2, 1943, with severe bruising and purpura, especially of his gums and the left lower limb, the latter being also swollen. The papillae of his tongue were atrophied. He was markedly anaemic, and the capillary fragility was raised. His diet had been deficient in greens and potatoes, and had consisted almost entirely of bread, margarine, cheese, and meat pies. After saturation with 9.1 g. of ascorbic acid intramuscularly in 13 days, rapid improvement resulted. The capillary fragility test became normal after one month. He refused a gastric-juice examination. The anaemia responded satisfactorily, as shown:

3/7/43: R.B.C., 2,080,000; Hb, 54%; C.I., 1.2; W.B.C., 4,500
 16/7/43: R.B.C., 3,500,000; Hb, 58%; C.I., 0.83; W.B.C., 3,800
 23/7/43: R.B.C., 4,100,000; Hb, 72%; C.I., 0.88; W.B.C., 4,700
 15/9/43: R.B.C., 5,650,000; Hb, 108%; C.I., 0.95; W.B.C., 9,000

Case 3.—A man aged 76 was admitted on May 29, 1943. Three weeks previously he noticed spontaneous bruising of his upper and lower limbs, which became more extensive and confluent. His gums were normal (he was edentulous), but he had numerous petechiae in the mucous membrane of his mouth. His diet had consisted almost entirely of bread, margarine, and cheese. He was markedly anaemic, and the capillary fragility was raised. He was saturated with 11.2 g. of ascorbic acid intramuscularly in 12 days. After a few days of this treatment the ecchymosis and purpura began to fade and rapid improvement took place. The capillary fragility test became normal in six weeks. Hydrochloric acid was present in his gastric juice in normal amounts. The anaemia responded satisfactorily, as shown:

31/5/43: R.B.C., 1,700,000; Hb, 38%; C.I., 1.11; W.B.C., 2,500
 10/6/43: R.B.C., 2,540,000; Hb, 50%; C.I., 1.10; W.B.C., 2,180
 5/7/43: R.B.C., 3,100,000; Hb, 74%; C.I., 1.20; W.B.C., 3,000
 14/9/43: R.B.C., 3,870,000; Hb, 84%; C.I., 1.07; W.B.C., 4,500

He died as a result of myocardial degeneration and congestive heart failure on Oct. 30, 1943.

Case 4.—A man aged 79 was admitted on May 5, 1943, with a history of three months' spontaneous bruising of his upper and lower limbs. His diet had consisted almost entirely of bread, margarine, and cheese. On admission he was found to have extensive bruising and purpura of his upper and lower limbs. His gums were spongy and bruised, and they bled easily. The capillary

fragility was markedly raised, and he was markedly anaemic. The blood count was: R.B.C., 1.35 millions; Hb, 28%; C.I., 1.04; W.B.C., 3,000. He died five days after admission as a result of congestive heart failure.

Comment

About 70% of cases of scurvy have an associated anaemia, according to Mettler *et al.* (1930). Although most cases described are normocytic, reports of cases of scurvy associated with macrocytic anaemia have been described previously by Parsons (1938)—two cases of infantile scurvy; Dunlop and Scarborough (1935), Nisenson and Cohen (1937), and Jennings and Glazebrook (1938)—one case each of adult scurvy. Perhaps the severity and duration of the clinical scurvy determine the nature of the anaemia. Thus a comparatively early and mild case would have a normocytic anaemia, and a more severe and long-standing case which may interfere with maturation of the red blood corpuscles at an earlier stage would have a macrocytic or even an aplastic anaemia. It is of interest in this respect to note that Wohlbach (1937) found the red bone marrow replaced by homogeneous amyloid-like material in guinea-pigs fed on a vitamin-C-deficient diet for a long time. The bone marrow was studied by Jennings and Glazebrook in their case, and they found the appearances to be similar to that of pernicious anaemia.

Mettler *et al.* (1930) proved conclusively that the anaemia of scurvy responded to orange juice with a maximum reticulocyte response on the fifth to eighth day. Moreover, in two of their cases iron and liver had no effect on the anaemia. Mettler and Chew (1932) cured the anaemia of scurvy in guinea-pigs by orange juice. Dunlop and Scarborough (1935) confirmed the specific effect of vitamin C on the anaemia of scurvy by giving their patients a diet similar to that which produced scurvy, and the anaemia responded satisfactorily to ascorbic acid. One of their patients had a high-colour-index anaemia.

Whether subclinical scurvy can produce anaemia or not is uncertain. Dyke *et al.* (1942) described three cases of "irreversible" pernicious anaemia which improved when ascorbic acid as well as liver was given. They did not, however, give details of the diet of their patients. It would, however, seem reasonable to treat cases of macrocytic anaemia having a history of vitamin C deficiency with ascorbic acid, particularly if hydrochloric acid is present in the gastric juice, before a diagnosis of refractory anaemia is made.

In the group of cases described above vitamin C deficiency appears to have been responsible for the high-colour-index anaemia. It may be argued that some factor in the hospital diet could have been responsible for the improvement in the anaemia and not the ascorbic acid. I consider this unlikely, since the diet did not contain any liver; and iron and the vitamin B complex, the other recognized haematinics, were not present appreciably above minimal requirements.

Summary

Four cases of "bachelor scurvy" associated with a high-colour-index anaemia are described.

The scurvy as well as the anaemia responded satisfactorily to ascorbic acid given intramuscularly and a mixed hospital diet in three cases, without the addition of haematinics such as liver, iron, and vitamin B complex. Vitamin C deficiency is considered to have been the cause of the anaemia.

It is suggested that ascorbic acid therapy should be tried in cases of macrocytic anaemia whose diets are deficient in vitamin C, particularly if hydrochloric acid is present in the gastric juice before a diagnosis of refractory anaemia is made.

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Medical Memoranda

Treatment of Human Cutaneous Anthrax with Penicillin

In view of the fact that few clinical reports of the treatment of human anthrax by penicillin have been published the following case may be of interest.

CASE RECORD

An old, well-nourished, well-preserved ex-chief, a large cattle-owner, aged about 70, was admitted to hospital on May 4, 1945, with a history of a swelling in the neck of five days' duration. On examination there was a large fairly painful indurated swelling below the left jaw, covered by a black eschar about 3 by 1½ in.; much oedema and induration surrounded this area, spreading particularly round the front of the neck. A smear from the edge of the eschar showed anthrax bacilli, no other organisms being seen. The patient was very ill; his temperature was 102.2° F., the pulse was poor, and he was mentally very confused; the lower margin of the spleen was at the level of the umbilicus, was moderately soft, and was painful on palpation. The blood slide was negative on two occasions for malaria parasites.

In spite of the fact that, in general, anthrax lesions in this part of the world respond well to N.A.B., it was decided, in view of his age and his very poor general condition, to give penicillin. This was accordingly started, 20,000 units of the sodium salt being given intramuscularly, followed by 10,000 units three-hourly by the same route, to a total of 200,000 units. Fluids at the rate of about 4 pints a day were given—at first by a gastric drip, and finally by mouth.

The clinical response was remarkable. Sixteen hours after administering penicillin the temperature was normal, induration and oedema surrounding the lesion were markedly less, the spleen had contracted slightly, and the general condition was much improved. The eschar appeared to be drier but slightly bigger. The penicillin was stopped 54 hours after its start. By this time the patient's general condition was very good, the induration and oedema of the neck had completely disappeared, the temperature had remained normal, and the spleen had contracted by about 1½ in.; smears from the lesion revealed no anthrax bacilli, and no other organisms were seen. Over the next two days the eschar separated, leaving a large ulcer about 4 by 1½ in. with a clean but deep granulating base; the spleen had contracted by 2 in. from its initial size. Unfortunately, on May 12 the patient developed a pneumonic consolidation of the base of the right lung, associated with a recrudescence in temperature and accompanied by severe hiccup; this responded rapidly to sulphathiazole, the hiccup being controlled by morphine. At the time of writing, May 21, the patient was convalescent, and the ulcer had nearly healed.

COMMENT

Both the malignant pustule and small outbreaks of intestinal anthrax, contracted from eating infected meat, are fairly often met with in this district. In general, the malignant pustule does not appear to have the same severity as cases encountered in England; and, as has already been said, these cases respond well to large doses of N.A.B. The infection in question, however, was of a particularly severe type in an old man, and the prognosis seemed to be very poor. In addition it appeared that the condition was becoming generalized, although no anthrax bacilli were seen in any of the blood slides. The patient lives in a hyperendemic malarial area, and it was known that he had had splenomegaly previous to the onset of the disease. His blood slide on five occasions within the first three days of admission was negative for malaria parasites, and it was thought that the changes in the size of the spleen were more likely to be due to an anthrax septicaemia than to a malarial relapse. He was given no antimalarial therapy.

The improvement shown after starting penicillin was dramatic. Since treating this case I have read an extract describing the treatment of three cases of human cutaneous anthrax in the United States by Murphy *et al.* The American authors recommend that 200,000 to 400,000 units should be given at the rate of 100,000 units in 24 hours to the average adult, and they appeared to favour continuous intravenous infusion of the drug. In the case described above, 200,000 units, given over 54 hours by intramuscular injection every three hours, were quite sufficient to obtain a prompt and lasting response, the general condition of the patient and the local condition of the pustule being greatly improved after the first 100,000 units.

I would like to thank the Acting Director of Medical Services, Kenya, for permission to publish this note.

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