8. Both ear drums of many showed abnormality immediately preceding death. This change is an antemortem phenomenon due to forcible ejection up to the eustachian tubes. When mastoid infection exists, it is the result and not the cause of the child's lowered condition.

9. Mastoid antrum puncture for diagnosis of latent mastoiditis is not to be recommended.

10. Bilateral mastoid operation as a cure for the disease was a failure. Operation is to be postponed as long as possible.

11. The autopsies of the two intensively studied series of cases of acute intestinal intoxication show that mastoid infection was not common.

12. There was no correspondence between the bacteriology of the infection in the upper respiratory tract and that in the intestinal tract.

13. Evidence is being accumulated to show that the disease is of intestinal origin.

CONCLUSION

Infection of the mastoid antrum is not the cause of acute intestinal intoxication in infants.

SCURVY IN ADULTS

ESPECIALLY THE EFFECT OF FOOD RICH IN VITAMIN C ON BLOOD FORMATION *

STACY R. METTIER, M.D.,
GEORGE R. MINOT, M.D., S.D.,
AND
WILMOT C. TOWNSEND, M.D.
BOSTON

Scurvy in adults in large centers of population is infrequent but not rare. Outbreaks occurred among both the civil and the military population of certain countries [1] during the World War, but since 1920 only reports of a single case or small groups of cases appear in the literature.[2] Öhnell [2] and Shattuck [2] remark that the disease is perhaps more frequent than is supposed and that cases, especially in latent form, often are not recognized. Nine cases of scurvy in adults, eight of which were males, have recently come under our observation. The occurrence and certain precipitating factors of the disease, together with observations on the alleviation of the anemia of scurvy, comprise the subject matter of this paper.

OCCURRENCE OF SCURVY AND CERTAIN PRECIPITATING FACTORS

With the single exception of Öhnell,[2] who records the occurrence of scurvy in sixteen females out of the twenty-two cases he observed in Sweden, the other recent reports [3] concerning scurvy in adults deal largely with males over 50 years of age; men who live alone and whose subsistence depends on their own culinary inclinations. Meulengracht [2] defines the condition as "bachelor scurvy." This was essentially the state of affairs for seven of our eight male patients who were all over 50 years of age. The diets of our patients had been ones prepared largely from flours and often composed of food purchased at a store in a form ready to eat. These diets contained little or none of the foods rich in vitamin C; i. e., vegetables, fruits and certain animal tissues.

Poverty plays an important rôle in the choice of diet, although aversion to foods rich in vitamin C may be the reason given for an abnormal diet. Of rather rare occurrence is the development of scurvy from a prolonged strict diet provided for an organic disorder such as duodenal ulcer. Davidson [2] has reported such an instance. Our female patient developed scurvy seven years after being placed on a milk and cracker diet for gastric ulcer when 27 years old. She developed a psychosis and, although the symptoms associated with ulcer vanished, she continued to take a diet similar to the one originally prescribed. Another patient chose for over a year an ill balanced diet sparing in fruits and vegetables. He then took of his own volition, because of a tendency to diarrhea, a diet consisting of only boiled milk and white flour biscuits ("Uneedas"). The patient continued this regimen for about a month, when he developed acute bronchitis. As the bronchitis subsided, slight but definite signs of scurvy developed. They rapidly disappeared when he was given an appropriate antiscorbutic diet. A single instance is reported in the literature in which scurvy followed persistent vomiting in pregnancy.[4]

It is important to appreciate that ill health may be produced by the deficiency of a food factor in much smaller amounts than that necessary to create the typical syndrome associated with its lack, as has been so well discussed by McCollum.[5] It is recognized that conditions due to vitamin deficiencies and due to a lack of certain hormones are brought forth or intensified under various adverse conditions. Infectious processes, chronic fatigue and excesses of various sorts may precipitate a deficiency disorder when states of nutritional instability are present. Individuals with nutritional instability may have the appearance of health but usually lack a normal sense of well being. Infections undoubtedly can precipitate in such persons typical syndromes of a deficiency disorder and probably more often conditions that are not well recognized as being due to abnormal nutrition. An instance of scurvy being

---

* From the Thorndike Memorial Laboratory, Boston City Hospital, and the Department of Medicine and Tropical Medicine, Harvard University Medical School.

1. Harvier, P.: Epidemic of Scurvy, Paris méd. **20**: 394 (Nov. 10) 1917. Hess, A. F.: The Rôle of Antiscorbutics in Our Dietary, J. A. M. A. **71**: 941 (Sept. 21) 1918.

2. These include:
Hirschfeld, Isador: Three Cases of Scurvy in Adults, J. Am. Dent. A. **16**: 796 (May) 1929.
Esler, A. R.: Scurvy at Moyale, Kenya & East African M. J. **5**: 350 (Feb.) 1929.
Öhnell, Harald: Endemic, Manifest and Latent Scurvy, Acta med. Scandinav. **68**: 176, 1928.
Swanson, C. N.: Scurvy Complicating Vomiting of Pregnancy, J. A. M. A. **88**: 26 (Jan. 1) 1927.
Meulengracht, E.: Scurvy in Unmarried Men, Ugesk. f. læger **89**: 165 (March 3) 1927.
Salle, V.: Scurvy in Adults, Enzyklop. d. klin. Med., pp. 460-485, 1927.
Seckel, H.: Diagnosis of Sporadic Case of Scurvy, Deutsche med. Wchnschr. **53**: 790 (May 6) 1927.
Jones, D. R.: A Case of Scurvy, Lancet **1**: 232 (May 15) 1927.
Frecker, E. W.: A Case of Scurvy, M. J. Australia **1**: 274 (Feb. 19) 1927.
Roberts, L. J.: A Case of Scurvy, U. S. Nav. M. Bull. **25**: 126 (Jan.) 1927.
Hermans, E. H., and Horst, A. K.: Typical Case of Scurvy, Nederl. tijdschr. v. geneesk. **2**: 638 (Aug. 7) 1926.
Delamare, G., and Djémil, Said: Sporadic Scurvy, Bull. Acad. de méd. **94**: 1274 (Dec. 22) 1925.
Witherspoon, Jack: Adult Scurvy, J. Tennessee M. A. **17**: 271 (Dec.) 1924.
Norgate, R. H.: Three Cases of Scurvy, Lancet **2**: 511 (Sept. 8) 1923.
Madden, C.: Case of Scurvy, M. J. Australia **2**: 571 (Dec. 1) 1923.
Gottlieb, K.: A Case of Sporadic Scurvy, Wien. med. Wchnschr. **62**: 577, 1922.
McClelland, R. S.: A Case of Scurvy, Lancet **2**: 608 (Sept. 17) 1921.
Bullowa, J. G. M.: Self-Induced Avitaminosis, M. Clin. North America **10**: 959 (Jan.) 1927.
Shattuck, G. C.: Scurvy with Reference Especially to Adults, J. A. M. A. **90**: 1861 (June 9) 1928.
Carr, J. G.: Scurvy, M. Clin. North America **5**: 115 (July) 1921.
Davidson, P. B.: The Development of Deficiency Disease During Therapeutic Diets, J. A. M. A. **90**: 1014 (March 31) 1928.

3. Meulengracht, Norgate, McClelland, Bullowa, Shattuck and Carr (footnote 2).

4. Swanson (footnote 2).

5. McCollum, E. V., and Simmonds, Nina: The Newer Knowledge of Nutrition, ed. 2, New York, MacMillan Company, 1929.

brought forth by an infection has already been cited in the case of the patient who developed bronchitis. A similar reaction has been noted in the experimental scurvy of guinea-pigs.[6] Another clinical example is as follows: A man, aged 60, entered the hospital because of bronchopneumonia and a fractured rib resulting from a recent injury. He was somewhat overweight and did not have the appearance of chronic ill health. The day after admission there developed a large ecchymosis over the left flank extending to the region of the broken rib. At this time the ecchymosis was thought to be dependent on the recent injury. The following day, however, a similar large area of ecchymosis appeared in the right flank, and a few hemorrhagic lesions appeared on the gums and about the buccal mucous membrane. Similar lesions also developed on the lower part of the right leg. It was then learned that for years his diet had consisted chiefly of coffee, pastry, potato, and bread and butter, with only small amounts of meat or fish, and that he ate but rarely green vegetables or fruit. He was given about 400 cc. of orange juice daily, together with a diet composed chiefly of concentrated carbohydrate food of the variety containing little or no vitamin C, and within a week the lesions attributed to scurvy entirely disappeared. Cases of this sort, in which an infection brings forth a definite syndrome because of an inadequate supply of a dietary factor for a prolonged period of time, are unusual, but serve to emphasize the importance of a well balanced diet with optimal amounts of all food factors for the maintenance of health.

The presence of arteriosclerosis in individuals partaking of an ill balanced diet for a long time may perhaps favor the development of a deficiency disorder. There is some evidence at hand to suggest that pernicious anemia and pellagra patients with arteriosclerosis are apt to require greater amounts of effective substance to alleviate their deficiency than similar patients with normal blood vessels. It has been suggested[7] that arteriosclerosis may enhance the development of scurvy, particularly in elderly men partaking of suboptimal amounts of vitamin C, because it was noted to be a feature of many cases of scurvy in adults. Cardiovascular damage, chiefly arteriosclerotic in type, was present to a greater or lesser degree in all of our recent eight cases of scurvy in elderly males. Perhaps thickening of the blood vessels prevents the tissues from receiving nutritious elements as readily as when such a condition is absent.

It is, of course, difficult to diagnose cases of "latent" scurvy unless some infectious process or other circumstance occurs that precipitates the typical disease. It is suspected, however, that cases of scurvy in adults occur more frequently than is commonly recognized and will be detected if relatively minor symptoms, such as poor appetite and loss of energy, are looked for and the dietary history is carefuly evaluated.

THE ANEMIA OF SCURVY

A study of the literature shows that anemia of variable degree occurs in a large percentage of adults with scurvy. It also occurs in infantile scurvy and is of regular incidence in the experimental scurvy of guinea-pigs.[8] Scurvy in adults is apt to develop following a prolonged period of nutritional instability and thus manifest a chronic condition. Chronicity apparently favors the production of anemia. Our observations on the blood are in accord with those of numerous other physicians.[9] In a large group of cases of scurvy in adults one may expect to find about 35 per cent with red blood cell counts between 2 and 3 million per cubic millimeter, a similar number with between 3 and 4 million red blood cells per cubic millimeter, and the remainder showing slight or no anemia with red blood cell counts above 4 million per cubic millimeter. The hemoglobin is reduced so that a color index of about 1 is the rule. Achromia of the red blood cells is not a feature, but slight central pallor of these cells is frequent. If hemorrhage has been an important factor in the production of the patient's anemia, a greater degree of achromia and a lower color index will occur. Abnormal variation in size is more pronounced than variation in shape. Occasionally there may be a sufficient number of nonachromic cells slightly larger than normal to suggest the possibility of pernicious anemia. About 1 per cent of the cells are usually polychromatophilic, and nucleated erythrocytes may appear in the peripheral blood. The blood platelets occur in from normal to moderately increased numbers. The leukocytes in uncomplicated cases are usually between 4,000 and 6,000 per cubic millimeter. A slight lymphocytosis with about 3 per cent atypical lymphoid cells frequently occurs. Monocytes are not increased. A few myelocytes are often to be observed. Polymorphonuclear eosinophils are not absent but basophils are seldom present.

It is well known that an antiscorbutic diet causes the anemia to lessen and to do so frequently with amazing rapidity, but there appears from the literature to be some confusion as to the exact reason for anemia in scurvy. Undernutrition and intercurrent infection have been held to be,[10] and undoubtedly are, contributing factors. Loss of blood, especially from the intestinal tract, has been considered by some individuals as the most important cause for the anemia. However, since anemia occurs without evidence of hemorrhage of large amount, some other factor would seem to be fundamental. The observations given farther on lend support to the idea that the anemia may be due to insufficient red blood cell production, as Hojer[8] and others have suggested. Our observations furthermore suggest that the inadequate function of the bone marrow may be directly dependent on a chronic lack of vitamin C, although in any given case some other factor or factors may intensify the process.

OBSERVATIONS ON THE RESPONSE OF THE RETICULOCYTES AND TOTAL RED BLOOD CELLS

Foods rich in vitamin C can rapidly effect erythropoiesis in scurvy for we have observed five cases in which a definite rapid increase of the reticulocytes occurred as a response to a diet rich in this vitamin. Data relative to the first patient, recorded in figure 1, show that the reticulocytes began to increase on the third day after he partook of a diet that was chiefly composed of fruit and green vegetables and that contained no animal food except boiled milk. They reached the peak

6. Nassau, E., and Scherzer, M.: Scurvy and Infection, Klin. Wchnschr. **3**: 314 (Feb. 19) 1924.

7. Minot, G. R.: Some Fundamental Clinical Aspects of Deficiencies, Ann. Int. Med. **3**: 216 (Sept.) 1929.

8. Meyer, A. W., and McCormick, L. M.: Studies on Scurvy, Stanford University Publications **2**: 199 (number 2) 1928. Liotta, D.: Experimental Scurvy: III. Modification of the Cell Elements of the Blood, Arch. di farmacol. sper. **36**: 76, 1923. Hojer, J. A.: Studies in Scurvy, in Almquist and Wiksell's Boktryckeri, Uppsala, 1924.

9. Meulengracht (footnote 2). Shattuck (footnote 2). Leitner, P.: Beiträge zur Haematologie und Klinik des Skorbuts, Wien. Klin. Wchnschr. **31**: 978, 1917. Eichorst, E. L. H.: Trattato di Patologie eterapia Speciale Medica (Milano. Soc. Ed.) **4**: 73, 1913-1914. Hess, A. F., and Fish, Mildred: The Blood, the Blood Vessels and the Diet, Am. J. Dis. Child. **8**: 385 (Dec.) 1914.

10. Hansmann, T.: Das Blutbild bei Skorbut, Ztschr. f. klin. Med. **93**: 346, 1922. Keefer, C. S., and Yang, C. S.: The Rôle of the Various Vitamins in the Production of Anemia, Nat. M. J. China **15**: 419 (Aug.) 1929.

of their rise on the eighth day and returned to normal by the fifteenth day. The response of this patient's reticulocytes was entirely similar to that observed in a second patient treated in a similar manner and in three other patients treated as described later on. Figure 2 shows the response in one of these other patients. The reticulocytes in all instances began to increase on the third or fourth day and then reached the peak of their rise on from the fifth to the eighth day. The height of

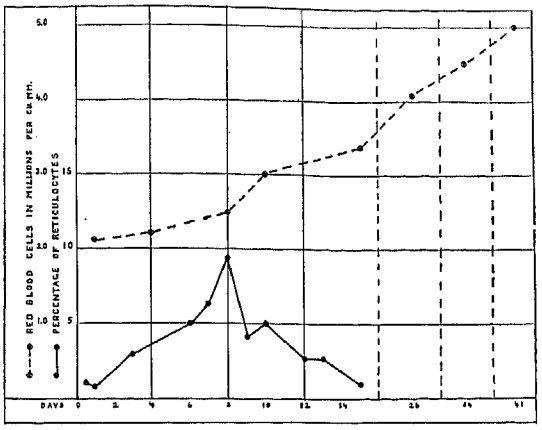

Fig. 1.—Response of the reticulocytes to a diet rich in fruit and green vegetables, without liver or iron, in a case of scurvy in a man.

their rise probably depends in some measure on the amount of potent material fed, the degree of anemia and the precise state of the patient. This state of affairs is comparable to what occurs with the administration of potent substances in pernicious anemia.[11] The response of the reticulocytes was coincident with cessation of hemorrhage in the scurvy patients. There was associated with this reaction of the bone marrow an increase of the red blood cells which usually continued rapidly to a normal level concomitant with a pronounced improvement in the health of the patient.

Certain anemias, appropriately called dietary anemias because they are associated with the patient partaking of an inadequate diet for a prolonged period of time, chronic chlorosis, and anemias of ill defined origin particularly occurring in women and often associated with gastric anacidity, as well as some other anemias, such as those due to chronic blood loss, lessen rapidly following the administration of large doses of iron. Therefore, it was thought that the anemia of scurvy might be alleviated by suitable iron therapy, and, like patients with anemia benefited by iron, might show a prompt response of reticulocytes to this element. Accordingly, two elderly male patients with typical scurvy were treated as follows:

They were given daily a diet deficient in vitamin C, composed of boiled milk and white flour biscuits. During six days there was no change in their reticulocytes. Then for six days in one case and for seven days in the other, 6 Gm. of iron and ammonium citrate (U. S. P.) were added to the diet. When the iron was omitted the same diet was continued but with the addition each day of 575 cc. of orange juice.

The results that occurred in the first patient given this treatment are recorded in figure 2. There was no significant quantitative change in the circulating reticulocytes within eight days following the commencement of iron therapy. However, after the patient had enjoyed the fruit juice for three days the reticulocytes began to increase from a stationary level of about 2.5 per cent. They reached a level as high as 9.7 and 9.9 per cent on the fourth and fifth day, respectively, and then rapidly fell to a low level during the following three days. During the twelve days that the patient took food lacking in vitamin C there was a drop in the hemoglobin from 73 to 55 per cent, and the red blood cells fell from 3.8 to 2.5 million per cubic millimeter. Striking, rapid improvement, however, occurred soon after the administration of orange juice, so that in twenty-four days after the patient first received this fruit juice the red blood cells were 4 million per cubic millimeter and the hemoglobin was 90 per cent.

The results obtained in the second case were similar. However, the anemia increased very little during the thirteen days that food lacking in vitamin C was taken by the patient. The reticulocytes increased from a stationary level of about 1 per cent, maintained for six days before and ten days after the commencement of iron therapy, to 3 per cent on the third day that orange juice was given. Their rise persisted so that there were between 4 and 5 per cent on the sixth, seventh and eighth days, followed by a return to a low level on the twelfth day. There was slow but progressive decrease of the anemia, and after forty-one days of an antiscorbutic diet the patient's hemoglobin had increased from 55 to 83 per cent and the red blood cells from 2.1 to 3.8 million per cubic millimeter. The relatively slight response of this patient's reticulocytes, especially in view of the relatively low level of his red blood cells and the continued retarded production of blood that followed, can be attributed to the fact that the patient suffered from cystitis, hypertension, arteriosclerosis and a mild degree of lead intoxication. Infections and poisons are known to inhibit the response of the bone marrow to substances potent for a given type of anemia. The four other patients whose reticulocytes were observed to respond to food rich in vitamin C had no complication except moderate arteriosclerosis. There was a rapid but tem-

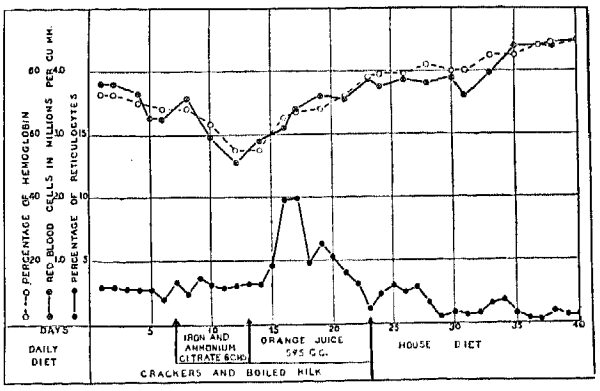

Fig. 2.—Absence of significant quantitative alteration of the reticulocytes to large doses of iron, but a distinct prompt response following the administration of orange juice, in a case of scurvy in a man.

porary increase of reticulocytes in these four patients with a quick regeneration of hemoglobin and red blood cells. Apparently a maximal production of reticulocytes occurred because they increased to the same approximate numbers as are found at comparable red blood cell levels in pernicious anemia following the administration of optimal amounts of effective material.

There are several reasons why one may attribute the reticulocyte response in the two patients given iron and later food rich in vitamin C to material contained in the

11. Minot, G. R.; Murphy, W. P., and Stetson, R. P.: The Response of the Reticulocytes to Liver Therapy, Am. J. M. Sc. **175**: 581 (May) 1928.

food rather than to a delayed reaction to iron. In the first place, observations on more than twenty-five patients in this clinic and studies by Schulten [12] and by Keefer and Yang [13] as well as by others indicate that when the red blood cells are below 3 million per cubic millimeter the response of the reticulocytes to even considerably smaller doses of iron than our patients received will commence within eight days and usually within five days after iron therapy is started. More than eight days had passed after iron had been given before the reticulocytes started to rise in either of the two scurvy cases. In the second place, if the response to iron is delayed, the reticulocytes do not reach the peak of their rise rapidly, as occurred in the first case. The relatively weak response in the second case sustained over a longer period of time was not a delayed response and is the type of response to be expected when suboptimal amounts of potent material act on the bone marrow or when the effect of optimal amounts are inhibited by infection or other complications. Furthermore, the response of the reticulocytes in these two patients is entirely comparable to that in each of the other two scurvy cases in which the reticulocytes were observed daily and which responded to food rich in vitamin C and to that in one case to be referred to treated with liver pulp.

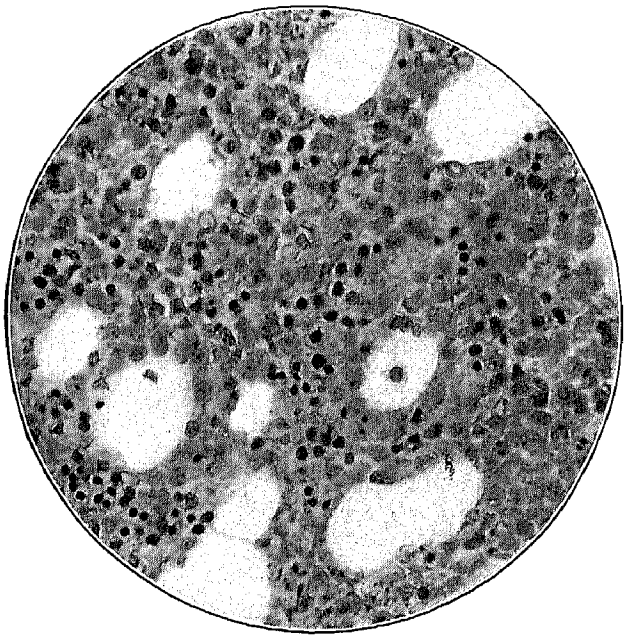

Fig. 3.—Appearance of bone marrow from an elderly man with scurvy; reduced from a photomicrograph with a magnification of 400 diameters. There are scattered foci of nucleated red blood cells and a moderate number of fat cells.

A liver extract potent for pernicious anemia was prepared in such a way that it could not contain vitamin C. This material was shown to be ineffective on the anemia of scurvy. Two patients, on a scurvy producing diet, with red blood cell counts of about 2 milion per cubic millimeter, showed no response of their reticulocytes and showed a slight decrease in the total number of red blood cells during ten days when they were each fed sufficient liver extract to cause a prompt response in pernicious anemia. The feeding of 240 Gm. a day of fresh raw liver pulp promptly produced a reticulocyte response with 8 per cent reticulocytes at the peak of their rise, and produced rapid regeneration of red blood cells in one of the cases, which is attributed to the fact that fresh liver is rich in vitamin C. The anemia of the other patient lessened rapidly when he took an ordinary well balanced diet containing plenty of vitamin C.

The observations recorded indicate that food rich in vitamin C can cause a prompt response of the reticulocytes and a rapid regeneration of blood in scurvy, and evidence favors the conception that neither large doses of iron nor the substance effective in pernicious anemia can do so. This information lends support to the idea that the fundamental cause of anemia in scurvy is the lack of vitamin C in the diet, although undoubtedly hemorrhage, infection, undernutrition and other factors may complicate the picture. It would appear, thus, as if vitamin C rather specifically affected the bone marrow of the patient with scurvy.

THE BONE MARROW IN SCURVY

There are only a few accounts in the literature of the state of the human scorbutic bone marrow. In their textbooks, MacCallum,[14] and Naegeli [15] described the bone marrow as having lost its blood-forming elements and as having become converted into an edematous fibrous tissue. Vedder,[16] on the other hand, states that the bone marrow is consistent with the type seen in secondary anemia from many causes. He writes that the fat disappears and that the marrow shows marked cellular hyperplasia.

Before treatment a specimen of sternal bone marrow was obtained by bone marrow puncture from the second patient given iron. A photomicrograph of this bone marrow is reproduced in figure 3. The tissue shows moderate cellular hyperplasia and contains a few isolated fat cells. There are scattered small varying-sized groups of nucleated red blood cells. Many myelocytes are present, among which are several of the eosinophilic variety. Adult polymorphonuclear leukocytes and megakaryocytes occur in moderate numbers. There is no evidence of fibrosis.

A specimen of sternal bone marrow was also removed at the peak of the reticulocyte response from the second patient, treated with only a diet rich in vitamin C. This specimen, pictured in figure 4, in contrast to the one shown in figure 3, shows quantitatively more nucleated red blood cells. Mitotic figures among the precursors of these cells were not apparent in the specimen (fig. 3) obtained from the patient prior to any treatment, whereas, in the specimen obtained at the time of the peak of the reticulocyte rise, a few mitotic figures appear in each field of the microscope.

Thus the bone marrow alterations depicted by these two specimens are characteristic of what is spoken of commonly as secondary anemia. No definite conclusions, of course, can be drawn as to the comparative histology of the bone marrow before and during treatment; but the character of the bone marrow suggests the possibility that when vitamin C has been chronically deficient its replenishment in the body can promote in some fashion the development of nucleated erythrocytes. In pernicious anemia, potent material apparently permits maturation of the megaloblasts in the bone marrow,

12. Schulten, Hans.: The Treatment of Hypochromatic Anemia with Large Doses of Reduced Iron, München. med. Wchnschr. **77**: 355 (Feb. 28) 1930.

13. Keefer, C. S., and Yang, C. S.: Anemia of Undernutrition; Report of Cases with Results of Treatment, Nat. M. J. China **15**: 701 (Dec.) 1929. Yang, C. S., and Keefer, C. S.: Liver and Iron in the Treatment of Secondary Anemia, Nat. M. J. China **15**: 721 (Dec.) 1929.

14. MacCallum, W. G.: A Textbook of Pathology, Philadelphia, W. B. Saunders Company, 1928, p. 927.

15. Naegeli, O.: Blutkrankheit and Blutdiagnostik, Berlin, Julius Springer, 1923, p. 364.

16. Vedder, E. B.: Scurvy, in Tice: Practice of Medicine, Hagerstown, Md., W. F. Prior Company, Inc. **9**: 172, 1929.

which leads to a reticulocyte response. In scurvy and in anemias responding to iron perhaps the effective substance hastens maturation of the normoblasts, which, under comparable conditions, permits a reticulocyte response that we have noted tends to occur somewhat more rapidly than in pernicious anemia. The increased rate could be accounted for by the fact that the normoblast is a less primitive cell than the megaloblast, so that the former could maturate sooner than the latter. At all events, the increased activity of the bone marrow in scurvy is clearly reflected in the peripheral blood by the reticulocyte response to food rich in vitamin C.

Knowledge has been advanced rapidly in recent years concerning the importance of food in alleviating anemia and the influence of inadequate diets and digestive processes in the causation of anemia. Known vitamins play no rôle in the regeneration of the blood in pernicious anemia, and at least are not required for regeneration of blood in many cases of secondary anemia. If, however, an anemia can be related to a vitamin

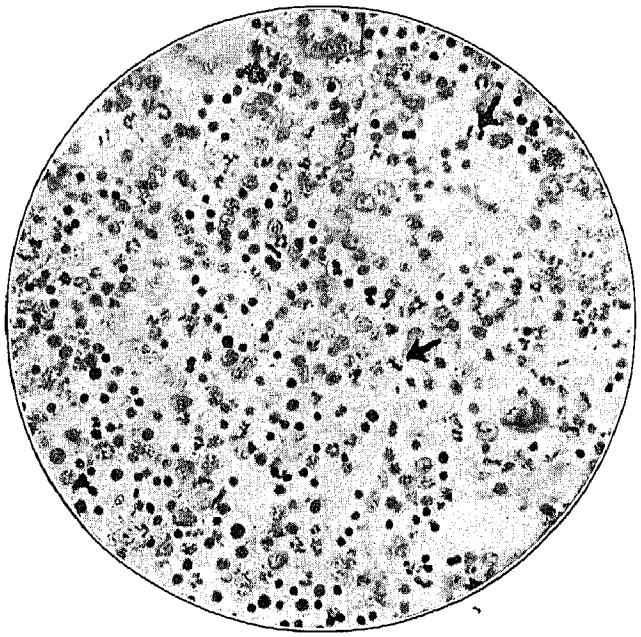

Fig. 4.—Appearance of bone marrow from an elderly man with scurvy at the peak of the reticulocyte response induced by orange juice; reduced from a photomicrograph with a magnification of 400 diameters. Note the large numbers of nucleated red blood cells. A few mitotic figures are present among the younger forms, some of which are indicated by arrows.

deficiency, it oftentimes will be decreased by the administration of material rich in the vitamin, the lack of which occurs in the given patient. Such appears to be the case in pellagra, in chronic vitamin A deficiency and in scurvy.

SUMMARY

Nine cases of scurvy in adults were studied. Eight occurred in elderly males.

Scurvy can be precipitated by infectious processes in individuals with certain types of chronic nutritional instability. Arteriosclerosis may favor the development of the disease.

In adults with scurvy, anemia is common and often pronounced. Fruit, green vegetables and fresh liver, foods which are rich in vitamin C, can cause in these patients a prompt response of reticulocytes and rapid regeneration of blood. Neither large doses of iron nor the substance potent in pernicious anemia appear to accomplish these effects.

The bone marrow in two cases of scurvy was examined microscopically. It appears to be of the type that occurs in what is often spoken of as secondary anemia.

Vitamin C apparently can have a specific effect on erythropoiesis when there has been a chronic lack of this vitamin.

PREGNANCY AND LABOR COMPLICATED BY GRANULOMA INGUINALE *

LESTER A. WILSON, M.D.
Professor of Obstetrics, Medical College of the State of South Carolina
CHARLESTON, S. C.

Granuloma inguinale is a disease found chiefly in the subtropics, but during the last ten years sporadic cases have been reported from many localities in this country, especially in the South. In Charleston, S. C., it is fairly common; the genito-urinary department of Roper Hospital informs me that it treats about twenty-five cases a year. Kenneth M. Lynch [1] of Charleston and Meredith F. Campbell [2] of New York are to be credited with the most comprehensive study of this condition that has been made in this country. The organisms that are supposed to be the cause of this disease were first described by Donovan in 1905 and are called Donovan bodies.

The method for demonstrating this organism is as follows: A portion of ulceration is selected where the ulceration appears to be spreading, not healing. Wet dressings of physiologic solution of sodium chloride are applied to this area for about twenty-four hours. The surface is then wiped thoroughly with sponges wet with salt solution, and with a dull curet or the edge of a glass micro-slide the surface is deeply scraped. This material is smeared out on several slides and allowed to dry thoroughly. Leishman's stain is applied to the smear, allowed to stand four minutes, then diluted with one half volume of distilled water and allowed to stand one minute. It is washed quickly with running water and dried with a blotter. The Donovan bodies are found scattered through the specimen but more particularly in the endothelial cells having a bluish bacillary or diplococcus appearance surrounded by a pinkish capsular zone.

The disease begins as a small moist papule, which rapidly ulcerates with progressive and eccentric invasion of the surrounding tissues by an elevated, reddish, often shiny, delicately skinned granulomatous proliferation, superficially ulcerating when moist, cracking when dry. Usually, however, the lesions are moist, and a thick mucoid exudation possessing a peculiar odor is characteristic. The disease shows no tendency to spontaneous healing. The genitals and perigenital tissues are most often involved. The disease apparently spreads by auto-inoculation and peripheral extension. Inoculation by transmission from clothing and friction is the probable explanation of its distribution. It is in no sense a venereal disease, as neither the husbands nor any member of the family of these patients give a history of having had granuloma inguinale. The occurrence of elephantiasis of the genitalia or at times of a leg signi-

---

* Read before the Section on Obstetrics, Gynecology and Abdominal Surgery at the Eighty-First Annual Session of the American Medical Association, Detroit, June 26, 1930.

1. Lynch, K. M.: Granuloma Inguinale, J. A. M. A. **77**: 925 (Sept. 17) 1921.

2. Campbell, M. F.: The Etiology of Granuloma Inguinale, Am. J. M. Sc. **174**: 670-679 (May) 1927.