A COMPARISON OF CLINICAL AND BLOOD PICTURES IN ADULT SCURVY

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(WITH SPECIAL PLATE)

Cases of scurvy such as were described by clinicians in past generations are becoming very rare, and at the same time more and more accurate laboratory methods for diagnosing the malady in a latent or pre-clinical stage are being evolved. So accurately quantitative are some of these tests that there might seem to be little place for the clinician in the diagnosis of scurvy, and a consequent danger that he may miss the condition even in its more flagrant forms. For this reason, and because of the rather unusual and deceptive blood picture in one of our cases, we have ventured to present them in this paper.

Case I

This patient, a man aged 52, had led an active and energetic life, and apart from a period of war service spent in India and Mesopotamia had worked continuously on the railways since the age of 18. He came of healthy stock, with no familial tendencies, was married, but had no children. Twenty years ago he had had malaria, and about seven years ago a severe attack of bronchitis; otherwise he had enjoyed good health. For several years, however, he had noticed a tendency to excessive breathlessness on exertion, but could not date the onset of this symptom.

In August, 1937, he noticed a sudden pain in the right shoulder, which he ascribed to rheumatism. The shoulder was not swollen or discoloured; the pain persisted for about four weeks, and then passed completely away. In November he found on rising one morning that the right knee was swollen. The swelling was followed by pain, which gradually increased in severity over a period of fourteen days and then as gradually passed off. The pain kept him awake at night, but was not increased by movement and did not prevent him from getting about. In December he was admitted to the surgical department at the Postgraduate Medical School for treatment of the condition in the right knee. The knee was found to be grossly swollen, with effusion into the joint and much periarticular bruising and redness. Flexion was possible to a right angle only. It was thought that the condition might be of infective origin, although the Wassermann test, the gonococcal complement-fixation test, and culture of aspirated fluid yielded no positive information. While he was in hospital it was noted that he had anaemia with a colour index below unity and a red count of 3,200,000. Radiographs of the joint showed no recognizable abnormality. With symptomatic measures the swelling and pain in the knee cleared up in three weeks, and he was discharged to the out-patient department for treatment of his anaemia. The anaemia, however, progressed, and by January the red count had fallen to 2,240,000, with a colour index of 1.27. Accordingly he was admitted to the medical department on January 15, 1938, with a provisional diagnosis of pernicious anaemia.

CONDITION ON ADMISSION

The patient was a well-built, cheerful man, with pallor of the mucous membranes and a sallow muddy tinge to the skin; he was not in any pain or distress, and was not

dyspnoeic at rest. Temperature, pulse, and respiration rate were normal. The mucosa of the tongue was smooth at the edges but not atrophic. The jaws were edentulous and the gums healthy, with no tendency to sponginess or bleeding. The heart was normal apart from a slight apical systolic murmur. The blood pressure was 155/80. The lungs showed slight emphysematous changes, and the liver was soft and a trifle enlarged; there were no other findings in chest or abdomen. The central nervous system was normal. The right knee was stiff, full flexion not being possible; and there was infiltration and staining of periarticular tissues as a result of former blood extravasation. Multiple small raised petechiae into the hair follicles were present on the extensor aspects of both legs. The patient stated that these had been noticed for some time, but he did not remember when they first occurred as they had never troubled him.

INVESTIGATIONS

Blood Count (15/1/38).—Red cells, 1,900,000 per c.mm.; haemoglobin, 40 per cent.; colour index, 1.05. White cells, 3,000 per c.mm.; reticulocytes, 3.2 per cent.

Price-Jones Curve.—Definitely megalocytic (see chart).

Marrow Puncture.—Definite hyperplasia, the early red cells being more affected than the white cells. Percentages of total marrow cells: haemocytoblasts, 5.5; primary erythroblasts, 39; normoblasts, 14; megaloblasts, 0.5. These findings were compatible with a diagnosis of pernicious anaemia.

Bleeding Time.—Eight and a half minutes.

Coagulation Time.—1 minute 45 seconds (Dale), 12 minutes (Lee)—that is, high normal.

Blood Bilirubin.—0.5 mg. per 100 c.cm.

Urine.—Some red blood cells and leucocytes.

Gastric Juice.—Absence of free acid after histamine.

Gastroscopy (Dr. R. S. Aitken).—Moderately extensive gastric atrophy with several recent haemorrhages in the mucosa.

Electrocardiogram.—Normal.

COURSE AND TREATMENT

On the whole these findings appeared to justify the diagnosis of pernicious anaemia, and liver therapy was at once instituted, at first with anahaemin (8 c.cm. in all) and later with hepastab (14 c.cm. in all). In spite of the high dosage no response was elicited in the blood picture. On February 3 a 600-c.cm. blood transfusion, given with the idea of stimulating the marrow, had no such effect.

The reticulocyte count, while remaining persistently high, showed no crisis, readings fluctuating between 4 per cent. and 9 per cent. being obtained. The blood sedimentation rate corrected for the anaemia present on February 4 was 64 mm. at the end of one hour, and in view of this high reading, the reticulocyte count, and the absence of response to liver a neoplasm was suspected, a suspicion hardened by the finding of occult blood in the stools at this time. Accordingly, full x-ray investigations of the principal bones, the thorax, and the abdomen were carried out, but no evidence of neoplasm was obtained. Liver was discontinued and large doses of iron were tried. The blood picture showed at first some slight improvement with iron, but soon relapsed again (Table I).

On February 8 a clue was given to the condition underlying this puzzling blood picture when an effusion of blood occurred into the tissue of the left popliteal space and the lower thigh muscles. By the next day the effusion in the left thigh muscles had increased, forming a deep painful brawny swelling reaching half-way up the under surface of the thigh. On February 14 another effusion occurred into the right ankle, forming an extensive haematoma around the external malleolus, and the discoloration round the right knee increased in extent. On February 22 a haematoma on the right shin with overlying oedema was noticed, and there was a fresh and more extensive bruising of the left thigh muscles (Plate, Figs. 1, 2, and 3).

Clinically the condition now was definitely scorbutic, and inquiry elicited the information that since childhood the

TABLE I

Date	Red Blood Cells per c.mm.	Reticulo-cytes %	Hb %	Colour Index	White Blood Cells per c.mm.	Polys %	Lymphs %	Large Monos %	Eosins %	Basos %	Treatment
3/12/37	3,200,000		60	0.93	5,000	60	35	3		2	Nil
11/1/38	2,240,000*	2.4	56	1.27	6,000	71	22	3	3	1	Nil
15/1/38	1,900,000	3.2	40	1.05	3,000						Nil
21/1/38	2,100,000	6.0	46	1.09	4,000						<i>Liver</i> 17/1/38 Anahaemin 4 c.cm.
31/1/38	1,840,000	6.4	38	1.05	5,000	65	33		2		24/1/38 " " "
4/2/38	2,200,000	7.2	38	0.86	4,600	62	32	2	2	2	{ 31/1/38 Hepastab 8 c.cm. 2/2/38 to 7/2/38 Hepastab 2 c.cm. on alternate days
15/2/38	2,900,000		52	0.90	5,000	62	28	7		3	<i>Blood and Iron</i> 3/2/38 Transfusion 600 c.cm. blood
21/2/38	1,800,000†		34	0.94	3,400	49	45	3	2		7/2/38 to 19/2/38 Ferri et ammon. cit. gr. xxx t.d.s.
25/2/38	2,000,000	3.6	36	0.90	4,400	55	35		6	4	<i>Vitamin C</i>
1/3/38	2,200,000‡	5.6	40	0.90	2,400	56	37	1	5	1	22/2/38 to 6/4/38 Ascorbic acid orally 600 mg. daily
8/3/38	3,000,000*		54	0.90	3,200	50	46	3	1		
16/3/38	3,900,000		72	0.92	7,000						25/2/38 Extra dose of 1,000 mg. ascorbic acid orally
22/3/38	4,000,000	1.0	77	0.96	4,500	50	40	5	4.5	0.5	
29/3/38	4,400,000		86	0.98	9,000						
19/4/38	4,800,000		92	0.95	5,000						Orange juice also given from 8/3/38 onwards

* Anisocytosis.

† Considerable anisocytosis with many megalocytes.

‡ Anisocytosis and polychromasia.

patient had had a distaste for fruit and fresh vegetables amounting to a phobia, the sight of a tomato or an apple being enough to cause nausea. Most of the meat which he had at home was twice cooked in the form of puddings and pies, and his only source of vitamin C appeared to be potatoes. It was not at the time realized, however, that avitaminosis C could cause such a profound anaemia of a hyperchromic type, and it was thought that it was only a contributory factor in the production of this anaemia. The iron therapy was discontinued on February 19, and on February 22 and the ensuing days quantitative tests for scurvy were performed (Table III).

The decolorization time of intradermal 2:6-dichlorophenol-indophenol on February 22 averaged seventeen minutes, and the total urinary output of ascorbic acid in twenty-four hours was only 6.7 mg. Treatment was at once begun with ascorbic acid, 600 mg. daily by mouth. On February 25 urinary excretion was still deficient, and an extra dose of 1,000 mg. by mouth was given. The total output during the ensuing twenty-four hours, however, was only 12.9 mg. The red cell count had risen to 2,000,000 per c.mm.

On March 5 the haemorrhages into the hair follicles of the leg had completely disappeared, and the larger ones were absorbing satisfactorily. On this, the thirteenth day of treatment, the excretion test showed a greatly increased figure for the first time, the intradermal test having become normal on the fourth day of vitamin C therapy. On March 8 extra vitamin C, in the form of orange juice, was administered, and apparently saturation was achieved a week later, after three weeks' therapy and the administration of over 14 grammes of ascorbic acid; for on March 15 235 mg. of the test dose of 600 mg. were excreted. Throughout this period, on vitamin C therapy alone, steady improvement was made, the red cell count being 3,900,000 per c.mm. by March 16 and 4,400,000 by March 30. As by the latter date the patient's haematoma were completely absorbed, his joints normal, and his other scorbutic signs gone, he was discharged. With continued vitamin C therapy the blood count soon became normal.

Case II

This case makes an interesting comparison with Case I, for it presented more of the commonly described features of adult scurvy. But in spite of certain differences the two cases were obviously examples of the same disease.

The patient, a married man aged 65, had previously been very healthy. From 18 to 41 years of age he had been at sea, often on long voyages of eighteen months or two years. During this time he had seen men with scurvy, but his ships had always allowed rations of lime juice and fresh food. Only in the last four years had his diet become deficient in vitamin C, consisting mainly of porridge, milk puddings, bread and butter, fish, and eggs. His appetite had deteriorated, and at the time of admission was very poor, particularly for fruit, tomatoes, and the like. He felt generally unwell, but his chief complaints when first seen were of pain and swelling of the right knee for two months, and of progressive soreness and swelling of the gums for nine months. The latter had become so bad that he could eat soft food only.

CONDITION ON ADMISSION

On admission into Redhill County Hospital under the care of one of us (G. H. J.) the patient showed anaemia and wasting. The skin of the face, forearms, and hands was of a sallow muddy colour. The gums were striking (Figs. 5 and 6), being swollen, blue, and mauve, and bleeding very readily. The teeth showed marked caries and pyorrhoea, and there was a most unpleasant oral fetor. The right forearm showed a number of subcutaneous bullous haemorrhages, which had been caused by slight injuries a year before (Fig. 4). There were large ecchymoses on the knees, left shin, and left ankle. There were again many follicular petechiae on the shins and around and above the knees. The right knee was distended with fluid. Other symptoms revealed little of note, though there was some arteriosclerosis, and the blood pressure was 180/90. The tourniquet test was strongly positive, the whole forearm being smothered with large petechiae after five minutes of arm compression at 80 mm. Hg.

INVESTIGATIONS

Blood Count (8/7/38).—Red cells, 3,420,000 per c.mm.; haemoglobin, 67 per cent.; colour index, 0.98. White cells, 6,000 per c.mm.; reticulocytes, 0.8 per cent.

Marrow Puncture (15/7/38).—Megaloblasts, 1 per cent.; primary erythroblasts, 2 per cent.; late erythroblasts, 3.5 per cent.; normoblasts, 11.5 per cent.

Bleeding Time.—Less than five minutes.

Coagulation Time.—Three minutes (Wright).

It was unfortunately impossible to get a sample of gastric juice from this patient.

COURSE AND TREATMENT

The patient continued with a diet poor in vitamin C, but from his sixth day in hospital 600 mg. of ascorbic acid were given daily by mouth. By the end of a week's treatment his gums were much better, his bruises were much less marked, and his knee less swollen. After a fortnight's treatment his knee was still slightly swollen, but the gums and the skin were almost entirely free of haemorrhages. The tourniquet test by now was much less positive, fifty tiny petechiae being produced. On the fourth day of treatment there was a reticulocytosis (6 per cent.), but the anaemia again only improved slowly (Table II). As in the first case, however, the anaemia was completely cured as a result of vitamin C therapy, in this instance after about a month's treatment. At the time of the last blood count the tourniquet test was almost negative, only five minute petechiae being produced.

TABLE II

Date	Red Blood Cells per c.mm.	Reticulo-cytes %	Hb %	Colour Index	White Blood Cells per c.mm.	Polys %	Lymphs %	Large Monos %	Eosins %	Baso-phils %	Treatment
7/7/38	3,420,000*	0.8	67	0.98	6,000	63	27	8	1	1	Nil
15/7/38	3,500,000*	6	70	1.0	8,500	71	19	9	1		12/7/38 to 9/8/38 600 mg. ascorbic acid daily; fruit juice also daily from 27/7/38
30/7/38	4,400,000†	1.5	76	0.85	6,500	74	22	4			
15/8/38	4,500,000		94	1.0	11,200	65	29	5	1		

* Slight anisocytosis, poikilocytosis, and polychromasia.

† Slight anisocytosis.

The treatment given to this patient was 600 mg. of ascorbic acid daily by mouth. All other vitamin-C-containing food and iron-containing food was excluded from his diet as much as possible until July 27, when a liberal allowance of fresh fruit was introduced. The excretion of ascorbic acid in the urine was estimated daily (Table IV), and it was found that saturation was reached on the twelfth day of treatment and after the administration of 6.6 grammes of ascorbic acid. As might have been anticipated from the shorter history of deprivation, this case was less desaturated in vitamin C than Case I. With the general improvement in the patient's condition his appetite improved, and when seen recently he reported that he was "eating like a horse" and taking plenty of fruit.

Discussion

There are many features of interest in these cases, and not the least is that they were married men with devoted wives. Schultzer (1936) and others have drawn attention to the liability to scurvy of single and solitary men, but the first patient was most unwilling to come into hospital because only his wife was able to give him "the food that suited him." She had indeed, with the best will in the world, severely and carefully limited the vitamin C in his diet for many years! His extreme aversion to fresh fruit and vegetables, which appeared to have increased with the severity of the scurvy, was another interesting feature. After his scurvy had been successfully treated he was able to take fruit juice with a certain amount of relish.

The clinical features in these cases were, of course, quite typical: the sallow muddy complexions; the extravasations of blood behind the knees (Figs. 1 and 3) and around the ankle (Fig. 2); the swollen knee-joints; the oedema; the petechiae in the hyperkeratotic hair follicles on the legs (Fig. 3); and the spongy gums in Case II (Figs. 5 and 6).

Bondurant (1934), Schultzer (1936), and Nisenson and Cohen (1937) have all recently reported similar cases, and among these and other recent ones the characteristic gum bleeding and mouth fetor occurred only occasionally. In the other cases teeth had been removed, and the most obvious evidence of scurvy was not therefore

prominent. In view of the recent importance attached to the discovery of latent scurvy as a cause of ill-health and its appearance after comparatively short periods of time in the dietary treatment of peptic ulcer (Bourne, 1938), it is interesting to observe from the first case the length of time during which scurvy will remain latent. It is fair to assume that for forty years at least this man had lived on a diet grossly deficient in vitamin C, and yet only for a few years had he had any symptoms. The first of these symptoms, dyspnoea, was presumably due to anaemia, and had been present increasingly for three or four years. In his case the haemorrhagic symptoms had only just appeared when he was seen, but Schultzer's (1936) cases show that severe haemorrhagic phenomena may make scurvy evident before anaemia has developed.

The anaemia in this case was of peculiar interest, and because it proved deceptive to us we now propose to consider it in detail. The blood counts and their relation to the treatment given are shown in Table I. The megalocytic and hyperchromic tendency of the anaemia up to the time of vitamin C therapy is evident from this table and from the Price-Jones curve (see chart) of January 11. On January 11, before any treatment, the mean corpuscular volume was 113.6 μ (normal, 75.7 to 96 μ), and there was anisocytosis. A little later the sternal marrow showed a hyperplasia chiefly affecting the early red cells, and the gastric juice revealed an achlorhydria. The blood cells at this time showed also a considerable variation in density of staining.

Chart of Case I: Price-Jones curve, showing the megalocytosis before the beginning of treatment.

These findings are similar to those described in pernicious anaemia, but there was a persistent reticulocytosis (2.5 to 9 per cent.), and neither the blood count nor the reticulocytes gave any response to liver therapy. Iron, as was anticipated, also had no effect. That a megalocytic anaemia of this type might occur in scurvy was first

suggested by Mettier, Minot, and Townsend (1930), who found that in the severe anaemia of some scurvy cases there might be enough red cells larger than normal to suggest a diagnosis of pernicious anaemia. Nisenson and Cohen (1937) have described a severe case of scurvy, very much like Case I, with marked anaemia and a colour index of 1.16. Among eight cases of infantile scurvy Parsons and Smallwood (1935) report one case of megalocytic anaemia, while two of their anaemia cases were normocytic and orthochromic, and two were hypochromic. Three cases had no anaemia at all. Mettier, Minot, and Townsend state that of cases of clinical adult scurvy a third are not anaemic, while half the remainder are severely anaemic and half moderately anaemic. As a general rule they found an orthochromic normocytic anaemia in scurvy, and this type of anaemia, shown by the second case, has also been found by Bondurant (1934) and Wood (1935).

The anaemia of scurvy is attributed by Parsons and Smallwood to deficient oxygenation of the marrow. Vitamin C takes up oxygen and releases it in the tissues, and when vitamin C is deficient in the marrow the maturation of red cells is retarded by lack of oxygen. While the stage of maturation of normoblasts to erythroblasts is most usually disturbed, the megaloblast maturation to normoblast may be chiefly affected in some cases, as in the first case just described. This case, with a very low level of vitamin C in the tissues, showed in the marrow a great interference with the maturation of the early cells of the red cell series, and a severe megalocytic anaemia resulted. Case II, with a less marked vitamin C desaturation of the tissues, showed no definite interference with the maturation of the early red cells and only a moderate normocytic anaemia. Such findings suggest that there is some relation between the extent of vitamin C lack in the tissues and the point at which marrow-cell maturation is chiefly affected.

In cases of scurvy anaemia, vitamin C administration provokes a reticulocytosis (see Case II), but the high reticulocyte count in Case I before treatment is a curious finding, though it was a feature to a lesser degree (2.5 per cent.) in Nisenson and Cohen's megalocytic case. The hypochromic anaemias found in scurvy result from blood loss and an associated iron deficiency, and in such cases a partial response to iron may be expected.

Achlorhydria may be a contributory factor in producing scurvy, as was pointed out by Schultzer (1933): for vitamin C is only stable in an acid medium. Injection therapy is therefore preferable in such cases.

We employed as confirmatory of the diagnosis of clinical scurvy three tests which have been used to make the diagnosis of latent scurvy. These were the ascorbic acid saturation test of Abbasy (1935); Rotter's intra-dermal test with 2:6-dichlorophenolindophenol (Rotter, 1937); and the capillary resistance test. The position of these tests in relation to scurvy in general and to our cases in particular may be summarized briefly as follows:

1. THE ASCORBIC ACID SATURATION TEST OF ABBASY

In this test the patient is kept on a diet free of vitamin C, and after a short control period 500 to 1,000 mg. of vitamin C (ascorbic acid) is given daily by mouth and the amount excreted in the urine is estimated. The saturation point is usually reached when half the quantity administered daily is excreted, but even on vitamin-C-free diets some ascorbic acid is excreted by severe cases of scurvy (Schultzer, 1936). All Schultzer's cases excreted as much as 17 mg. daily, and one excreted 33 mg. under these conditions.

Our first case was given 600 mg. of ascorbic acid by mouth on all the days shown in Table III, except on the fifth day (February 25), when an extra 1,000 mg. was given, also by mouth. Fruit juice was added from March 8 to 14, and was discontinued for the excretion test on March 15. The table reveals several interesting facts, the first being the comparatively large amount of ascorbic acid excreted when the body was still as markedly desaturated as it was on the day preceding therapy and in the early days of treatment. Also interesting was the long delay of eight days before there was any appreciable increase in the amount excreted. In the last place, if

TABLE III.—Results of Quantitative Tests for Scurvy in Case I

Date	Daily Amount of Ascorbic Acid Excreted	Volume of Urine	Daily Amount of Ascorbic Acid Administered
	mg.	c.cm.	mg.
21/2/38	6.7	1,180	Nil
22/2/38	14.9	950	600
23/2/38	9.3	790	600
24/2/38	15.9	1,000	600
25/2/38	14.6	1,305	1,600
26/2/38	12.9	1,220	600
27/2/38	—	—	600
28/2/38	14.5	1,310	600
1/3/38	13.5	1,230	600
2/3/38	12.9	750	600
3/3/38	27.8	1,310	600
4/3/38	44.7	970	600
5/3/38	110.0	1,160	600
6/3/38	85.2	1,270	600
7/3/38	59.7	1,030	600
15/3/38	235.2	1,690	600

TABLE IV.—Results of Quantitative Tests for Scurvy in Case II

Date	Daily Amount of Ascorbic Acid Excreted	Volume of Urine	Daily Amount of Ascorbic Acid Administered
	mg.	c.cm.	mg.
8/7/38	5.5	292	Nil
9/7/38	7.4	356	Nil
10/7/38	18.4	469	Nil
11/7/38	7.0	234	Nil
12/7/38	9.4	347	600
13/7/38	18.3	571	600
14/7/38	11.6	365	600
15/7/38	13.9	395	600
16/7/38	29.5	785	600
17/7/38	6.0	200	600
18/7/38	22.5	925	600
19/7/38	74.2	1,050	600
20/7/38	147.0	1,260	600
21/7/38	74.0	900	600
22/7/38	128.0	1,050	600
23/7/38	348.0*	675	600
26/7/38	270.0	820	600

* Only a half-day's specimen.

50 per cent. excretion of administered ascorbic acid indicated saturation, our first patient was apparently just short of this point after three weeks' treatment. By then 13.6 grammes of ascorbic acid tablets had been administered and much more had been given in fresh fruit, a considerable difference from the 0.5 gramme to 2.3 grammes which Wilkinson and Portnoy (1938) find will saturate a normal person.

Case II, as previously pointed out, was much less deficient in vitamin C, and was saturated after eleven days' treatment and the administration of 6.6 grammes of ascorbic acid (Table IV).

2. ROTTER'S INTRADERMAL TEST (1937)

In Table V the times of decolorization by Case I of an intradermal spot of the blue dye 2:6-dichlorophenolindophenol are charted. As described by Wilkinson and Portnoy (1938) 0.01 c.cm. of a solution of the dye (2 mg. in 4.9 c.cm.) was injected intradermally in four places. Normally the blue spots are decolorized within ten minutes, and this was the case in our healthy controls. But in cases of rheumatism, rheumatoid arthritis, and other infective conditions we found the time to be prolonged (up to thirty minutes in one instance). In scurvy it is usually much prolonged, and Case I at first showed average decolorization times of seventeen and twenty-one minutes. But after three days of oral ascorbic acid treatment the time became normal.

TABLE V

Date	Average Decolorization Time of Four Intradermal Tests	Treatment (Oral Ascorbic Acid)
	minutes	mg.
22/2/38	17	600
23/2/38	21	600
25/2/38	7½	1,600
26/2/38	8	600
1/3/38	8	600
2/3/38	7	600
12/3/38	4	600*

* Fruit juice also given from March 8.

It will be observed by comparison of Table III with Table V that the cutaneous dye test shows a normal result much more quickly than the excretion test—that is, it becomes normal in the presence of vitamin C deficiency. This fact was further exemplified by Case II, the patient giving a normal result (three to six minutes) before any treatment. Inquiry revealed that in the week before admission he had taken two bottles of lime juice, his only appreciable intake of vitamin C in four years. It is possible that this was the cause of his normal intradermal test time, though he was still very deficient in vitamin C and clinically a clear case of scurvy.

3. THE CAPILLARY RESISTANCE TEST

This test has slight technical variations, and for the cases in this paper a pressure of 80 mm. Hg was sustained for five minutes. Dalldorf (1931) has shown that in the experimental scurvy of guinea-pigs a decreased capillary resistance is the first sign of the disease. Bourne (1938) has used the test to disclose latent scurvy during the treatment of peptic ulcers, but it must be remembered that normal people may at times give positive results to variable

small extents. The height of the blood pressure and the state of the vessels influence the results. For these reasons it is perhaps not surprising that Case II initially gave a far more extensive response to the test than did Case I. In Case II numerous large petechiae appeared over the whole forearm; in Case I there were about fifty petechiae in the antecubital fossa. In both cases the test results approached normal very early in treatment. In Case I the result was negative on the eighth day, and by then the skin petechiae elsewhere were almost gone. This early decrease in capillary permeability corresponding with the rapid clearance of the haemorrhagic manifestations has been commented on by Schultz (1936), and is the converse of Dalldorf's observation. In Case II the capillary response was a little slower, being almost negative by the fifteenth day and quite negative at the end of a month's treatment. Rather surprisingly in Case I it was found that on the thirty-fifth day of treatment the test was again definitely positive. Schultz (1936) has also commented on this variation in the result of the capillary resistance test in cases of treated scurvy. In this connexion it is interesting to note that Greene (1934) found a positive tourniquet test in 10 per cent. of healthy children and only a similar number of positive results in a group of children who might have been suspected of latent scurvy. He found variations in the test from time to time, and even a difference between the responses on the two arms of a normal subject.

Summary

Two cases of adult scurvy have been described which showed many of the classical features of the disease. The first case revealed a megalocytic anaemia not often associated with scurvy. A constant reticulocytosis, polychromasia, anisocytosis, and leucopenia were also observed in this case. A hyperplastic bone marrow showing many early red cells, a smooth tongue, gastric mucosal atrophy, and achlorhydria were further features of the case which simulated those of Addisonian anaemia. This case has been contrasted with a second case in which the anaemia was of the more usual orthochromic and normocytic type, and in which the characteristic gum lesions made the diagnosis evident, although the latter was otherwise a less severe case. An attempt has been made to find some relation between the clinical and haematological differences in the two cases and the different vitamin C levels demonstrated in their tissues by an excretion test. The initial differences of tissue vitamin C levels in the two cases seemed to be related to the lengths of their respective periods on scorbutic diets. The value of the intradermal test and the capillary resistance test in the diagnosis of scurvy has been discussed.

In the first case a megalocytic anaemia failed to respond either to liver or to iron, but in both cases the anaemia was cured solely as a result of vitamin C therapy. The clinical pictures improved more rapidly than the blood pictures. The comparative slowness of the responses in the blood might have been ascribed to defective absorption of the orally administered vitamin C as a result of an atrophic alimentary mucosa and lack of gastric acid. But the early disappearance of the superficial manifestations of the disease and the early normality of the intradermal test make this explanation unlikely. The great ascorbic acid desaturation of all the tissues, particularly in Case I, is the probable explanation of the slowness of the blood response. In Case I it is evident that the anaemia was much the earliest manifestation of the disease, so that it is not surprising that it should have been the last to disappear. The history of this case showed that

vitamin C deficiency must have been present for many years before the scurvy became clinically manifest.

We are indebted to Dr. Janet Vaughan, Dr. R. G. MacFarlane, and Dr. G. A. D. Haslewood for help with the investigation of the first case, and to Dr. J. H. Patterson for similar assistance with the second. The first case is published by permission of the Chief Medical Officer of the L.C.C., and the second by permission of the Chief Medical Officer of the Middlesex County Council.

** Through a clerical error, discovered too late, Surgeon Lieutenant A. J. Glazebrook's initials are incorrectly given on the plate.

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Clinical Memoranda

A Tumour of the Male Breast

This case, recently under my care, appears to be worth recording by reason of the very interesting differential diagnosis.

CLINICAL HISTORY

On October 4, 1937, I admitted a man about 35 years of age, complaining of a lump in his left breast. His attention had been first drawn to it eight months earlier by pain, though the pain had at no time been at all severe. Since then the size of the tumour had slowly increased. He also complained of pain in the right knee and ankle. He was thin, and had evidently lost a good deal of weight.

On examination there was an obvious lump in the left breast, mainly in the right upper quadrant. It stood out about three-quarters of an inch from the chest wall, was roughly oval in shape (2½ in. by 2 in.), and the margins were ill defined. There was no change in the skin. On palpation the tumour was of firm consistence and did not fluctuate. It was attached to the deeper structures, being fixed to the chest wall, but not adherent to the skin, which was freely movable over it. The lump was not at all tender. In one part over the third rib there was just palpable a very narrow ridge of bone standing up one-eighth of an inch or less, and running across the rib. In the axilla were two enlarged, firm, painless, discrete, and freely movable glands, the largest of which, the size of the shell of an almond, it was easy to take up with the fingers. There were one or two similar but smaller glands in the subclavicular region, but the clavicle appeared free above. There were no abnormal physical signs in the chest, and the patient had no cough. On admission he had a temperature of 99.8° F., which responded to quinine at once. (I had treated him for malaria about two weeks previously.)

DIFFERENTIAL DIAGNOSIS

The diagnosis appeared to lie between tuberculosis and carcinoma, although a sarcoma and gumma had also to be borne in mind.

Against carcinoma there were the following points: (1) Carcinoma is reputed to be very rare amongst the natives,

although in my nine months out here I have had several cases. (2) The first symptom being pain, though at no time was pain a marked feature, and there was a complete absence of tenderness on examination. (3) The presence of the small bony ridge. In favour of carcinoma there were: (1) The general impression on examining the tumour. (2) The consistency and mobility of the glands. A supporting factor was the presence of pain in the knee and ankle, possibly of metastatic origin. There was no local abnormality to be discovered.

Against tuberculosis there were: (1) The absence of pyrexia save for that which responded to the quinine. (2) The absence of fluctuation. (3) The absence of any evidence of perilymphadenitis. (4) No evidence of any other focus of infection. In favour of tuberculosis there were: (1) The presence of the bony ridge, suggesting a bony origin of the tumour. (2) The extreme frequency of all types of tuberculous infection in this part of Central Africa. (3) The deep attachment with no skin involvement, though a sinus might have been expected. (4) Pain being the first symptom.

OPERATION

A radical operation was decided upon. The axilla and subclavicular region were cleared of infected glands. No evidence of periglandular reaction was found. On turning the pectoralis minor forwards and inwards a tuberculous abscess was found in the muscle, larger in size than a large walnut: it was in direct communication with an abscess in the third rib. There was a small bony ridge to the left margin of the opening into the bone. As the general condition of the patient contraindicated a partial resection of the rib the bony cavity was scraped and filled with bipp. There was an uneventful recovery.

Discussion

I consider that the presence of the bony ridge was of important diagnostic significance. I shall be interested to learn if the presence of such a ridge, due presumably to the hyperostotic reaction sometimes found in tuberculous infection of the bone, has often been noticed in these lesions of the ribs. A blood count might have helped in the diagnosis, and a radiograph certainly would have, but there are no facilities for this out here.

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Drainage of Pelvic Abscess in Acute Appendicitis

In cases of acute appendicitis with rupture of the appendix it is common to find, especially in late cases, that an abscess has formed in the bottom of the pelvis. This abscess is most safely and conveniently drained by the introduction of a tube through a small suprapubic incision.

I have devised a simple, quick, and easy method of doing this, as follows:

Place an ordinary sewing thimble on the forefinger of the left hand and introduce it through the wound made for the removal of the appendix, keeping it close to the anterior abdominal wall, till the tip reaches the central suprapubic position. The finger is now pressed against the anterior abdominal wall, and an incision, about three-quarters of an inch long, is made over and down to the thimble, which can be felt under the skin. The thimble is now pushed up through this incision and removed. A drainage tube is then placed on the tip of the left forefinger and guided by it down to the abscess cavity. By this method there is no danger of incising distended bowel, distended bladder, or the operator's finger.

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G. H. JENNINGS AND J. G. GLAZEBROOK: CLINICAL AND BLOOD PICTURES IN ADULT SCURVY

FIG. 1.—Case I. Adult scurvy, showing extensive extravasation of blood in right popliteal fossa and posterior part of thigh.

FIG. 2.—Case I. Extravasation of blood around right ankle and on right shin. Some oedema of foot.

FIG. 3.—Case I. Showing swelling of left knee with peri-articular and popliteal extravasations marked by tight bandage. Petechiae into hair follicles.

FIG. 4.—Case II. Forearm with bleb-like extravasations of blood resulting from slight traumata a year previously. Result of a recent tourniquet test also seen.

FIGS. 5 and 6.—Case II. Gums of upper and lower jaw showing considerable swelling and discoloration.