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EFFECT OF A.C.T.H. AND CORTISONE THERAPY IN BLOOD DISORDERS

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Recently a panel appointed by the Medical Research Council (1952) has given a summary of the responses to A.C.T.H. and cortisone in 88 patients suffering from various blood disorders. The present paper reports in more detail the results of this form of therapy in 24 patients, many of whom were included in the M.R.C. report. The literature on this subject is not yet extensive and is dealt with by Wintrobe *et al.* (1951). The mechanism of action of these hormones is uncertain, and will not be discussed here.

The cases are considered under three headings: (a) patients with purpura, (b) those with haemolytic anaemia, and (c) those suffering from miscellaneous blood disorders. Where no clinical or haematological response occurred, the results of treatment are given only in tabular form.

Purpura

Fourteen cases of purpura have been treated with A.C.T.H. and/or cortisone, and of these 10 had thrombocytopenic purpura. The condition appeared to be idiopathic in all but two.

In addition there were four patients suffering from persistent non-thrombocytopenic purpura in whom the exciting factor was unknown. These cases have been grouped together under the heading of "idiopathic non-thrombocytopenic purpura" (possibly allergic).

There was some evidence of a response to hormone therapy in the following six patients with thrombocytopenic purpura.

Case 1

This patient, a man aged 55 with no previous history of bleeding, woke up one morning to find his mouth full of blood; this was due to bleeding gums, and continued up to the time of admission three weeks later. He also developed petechiae on the limbs and trunk, and bruised easily; these features, too, were limited to the three weeks before admission. There was no history of the taking of drugs, or of infection, or of exposure to chemicals.

Apart from petechiae, bruises, and bleeding gums, the only abnormality found on admission was that the spleen was palpable on deep respiration. The haemoglobin level was 13.3 g.%, platelets 40,000 per c.mm., bleeding-time 29

minutes. The bone marrow contained numerous megakaryocytes and was otherwise normal. A diagnosis of idiopathic thrombocytopenic purpura was made. The patient was transfused with a pint (570 ml.) of blood on the third day in hospital and again on the sixth and ninth days. There was no clinical or haematological improvement. On the thirteenth day treatment with A.C.T.H. was begun, 25 mg. being injected six-hourly for eight days. Within 24 hours bleeding from the gums ceased and no further petechiae appeared. After eight days of treatment with A.C.T.H., however, there was no improvement in the haematological findings, the haemoglobin level being 10.6 g.%, platelets 30,000 per c.mm., bleeding-time 29 minutes. A month after cessation of A.C.T.H. therapy the platelet count was 50,000 per c.mm. and the bleeding-time seven minutes, but when the patient was next seen, 45 days after treatment was discontinued, the platelet count had risen to 254,000 per c.mm. and the bleeding-time was two minutes. During a follow-up period of a year there has been no further clinical evidence of a bleeding disorder, and a normal blood count and blood picture have been maintained.

Summary.—This was a case of idiopathic thrombocytopenic purpura in which it seemed likely that blood transfusion produced no improvement. Treatment with A.C.T.H. for a period of eight days in a dosage of 100 mg. daily coincided with clinical recovery. No immediate improvement in the platelet count occurred, but the blood figures returned to normal 45 days after treatment was stopped.

Case 2

A man aged 33, who had no previous history of a bleeding tendency or of exposure to infections, drugs, or chemicals, woke with blood in the mouth and found petechial spots on the tongue on the day before admission. By evening petechiae had developed all over the body, and at the time of admission he had haemorrhagic bullae in addition. His haemoglobin was 16 g.%, platelets 40,000 per c.mm., and the bleeding-time more than 30 minutes. He was given transfusions of a pint (570 ml.) of fresh blood on the first, second, and third days in hospital, but there was no improvement, and very gross haematuria developed. On the second day he was also given 200 mg. of cortisone by mouth in divided doses, and thereafter he had 100 mg. daily for six days, the dose then being progressively reduced for a further four days and supplemented by small doses of A.C.T.H. by injection. On the third day of treatment with cortisone there was marked clinical improvement. Bleeding from the mouth and urinary tract ceased and no further petechiae

developed. The platelets, however, were now only 20,000 per c.mm. and the bleeding-time was more than 20 minutes. Two days later the platelets began to increase significantly and the bleeding-time fell to two minutes. By the seventh day of cortisone treatment the platelet count reached 295,000 per c.mm. No clinical or haematological relapse has occurred during the eight months since treatment with cortisone ceased.

Summary.—This patient with idiopathic thrombocytopenic purpura showed no immediate improvement to blood transfusion, but a dramatic clinical and haematological improvement occurred during the administration of cortisone for seven days; this has persisted for eight months.

Case 3

A single woman aged 20, with no previous history of a bleeding disorder, had an irritation of the skin seven weeks before admission which developed into a severe exfoliative dermatitis. The cause was unknown, although it was suspected that it might have been due to the taking of headache powders. While under treatment for this dermatitis and after she had been in hospital for one month and was improving, petechiae developed all over the body, and haematuria, melaena, and epistaxis occurred. She had received codeine, diphenhydramine hydrochloride ("benadryl") (for one day only, as it caused swelling of the face and neck), "sulphatriad," and various local applications for the skin irritation and dermatitis, but whether any of these drugs were responsible for the bleeding tendency is unknown.

At this stage she was transferred to our medical ward, and blood examination showed the following figures: haemoglobin, 6.9 g.%; red cells, 2,500,000 per c.mm.; white cells, 10,600 per c.mm.; platelets, 25,000 per c.mm.; bleeding-time more than 40 minutes. Capillary resistance was very low. A diagnosis of thrombocytopenic purpura was made, but the causative factor was uncertain.

Treatment with A.C.T.H. was begun in a dosage of 25 mg. six-hourly by injection. A total of 1.7 g. was given, but it was necessary to transfuse 2 pints (1.1 litres) of blood on the second day of treatment and again three days later, as the patient was exceedingly ill. There was much less bleeding immediately after the first blood transfusion, and

there was a distinct improvement after the fifth day of treatment, the petechiae disappearing and the external bleeding, including the haematuria, ceasing. The patient's general condition also improved, and the dermatitis became less severe. There was no immediate haematological response, the blood counts at the end of the A.C.T.H. therapy being: haemoglobin, 8.3 g.%; platelets, 20,000 per c.mm.; bleeding-time 20 minutes. Eight days later, however, the platelet count rose to 180,000 per c.mm. and the bleeding-time was 12 minutes. A further rise in the platelet count to 300,000 per c.mm. occurred, and the bleeding-time fell to three minutes. During a follow-up period of nine months there was no clinical or haematological relapse.

Summary.—This case of thrombocytopenic purpura was associated with exfoliative dermatitis of uncertain cause. Clinical and haematological "cure" followed treatment with A.C.T.H. and blood transfusion, and this "cure" was sustained for nine months.

Case 4

This patient, a woman aged 30, had a history of menorrhagia, petechiae, and bruising since the age of 15. A diagnosis of idiopathic thrombocytopenic purpura was made in 1947, and splenectomy was performed in August of that year. Unfortunately there was neither clinical nor haematological improvement from the operation. In May, 1951, the blood findings were: haemoglobin, 11 g.%; platelets, 45,000 per c.mm.; bleeding-time 14 minutes. A course of 25 mg. of A.C.T.H. was given six-hourly for 10 days by injection, but as there was no response this was followed immediately by the injection of 300 mg. of cortisone on the eleventh day, followed by 200 mg. on the twelfth day, in divided doses. The patient became psychotic, and the treatment had to be discontinued. The platelet count remained at 45,000 per c.mm., bleeding time 16 minutes. The bleeding tendency persisted.

Eight months later the patient was again given cortisone, this time by mouth, 50 mg. being administered six-hourly for 24 hours, and thereafter 25 mg. six-hourly for 13 days. Seven days after the treatment began the platelets rose from 30,000 to 155,000 per c.mm., and the bleeding-time fell from 48 to 5 minutes. This was the first haematological improvement that we had noticed during the five years she had been

Graph showing response of Case 5 to treatment.

under our care. Unfortunately, even during the time of treatment the platelets fell to 80,000 per c.mm. despite the continuation of cortisone therapy, which was discontinued because of the development of generalized oedema. There was no clinical or haematological improvement in the next two months.

Summary.—A patient with idiopathic thrombocytopenic purpura who did not respond to splenectomy. No response to treatment with A.C.T.H. occurred, but there was a temporary response to the oral administration of cortisone.

Case 5

A married woman aged 40 had a five-years history of episodes of bruising and petechiae, due to idiopathic thrombocytopenic purpura. Her initial haematological response to small doses of A.C.T.H. on two occasions has already been published (Robson and Duthie, 1950), and is shown in the Graph. The benefit from this treatment was only temporary; purpura recurred and menstruation became excessive. The patient was admitted to our wards in the Royal Infirmary of Edinburgh in July, 1951, the blood figures on that date being: haemoglobin, 11.7 g.%; platelets, 35,000 per c.mm.; bleeding-time, 10 minutes. She was treated by injections of A.C.T.H. in a dosage of 100 mg. daily for eight days, followed by 200 mg. on each of the next two days. There was a clinical remission on the fifth day of treatment, and on the last day the platelet count was 125,000 per c.mm. and the bleeding-time four minutes. This remission was, however, only very temporary, the platelet count falling to 40,000 per c.mm. five days after the cessation of treatment, although the bleeding-time was not increased. A month after the cessation of A.C.T.H. therapy petechiae were again seen.

In November, 1951, it was decided to try the effect of treatment with cortisone by mouth, and this was given in a dosage of 100 mg. daily for nine days, followed by a progressive reduction of dosage for six days. As will be seen in the Graph, this resulted in a rise of the platelet count

from 30,000 to 370,000 per c.mm. on the ninth day of treatment, the bleeding-time falling from 7 to 3½ minutes. The platelet count began to fall, however, during the period of reduction of dosage, and was 80,000 on the last day, when only 25 mg. was taken. The bleeding-time had risen to 11 minutes. Two days later there was a recurrence of petechiae and bruising.

A further course of oral cortisone therapy again resulted in a clinical and a haematological remission, as shown in the Graph, the platelets rising from 50,000 to 250,000. The spleen was removed during this period of improvement. The platelets thereafter rose from 255,000 to 1,000,000 per c.mm., and then fell to 448,000 per c.mm. During a follow-up period of seven months there has been no clinical or haematological deterioration.

Summary.—This case of idiopathic thrombocytopenic purpura responded on three occasions to A.C.T.H. by injection, and later showed a better haematological response on two occasions to the oral administration of cortisone in a daily dosage similar to that of A.C.T.H.

Case 6

A married woman aged 32 had developed petechiae and bruising three years previously during the puerperium following her fourth pregnancy. At that time her platelet count was 50,000, and it had remained about that level ever since. While splenectomy was being contemplated, she became pregnant again for the fifth time. The pregnancy itself was uneventful, but she gave birth to a purpuric and thrombocytopenic son in November, 1950. This case has already been published by Robson and Walker (1951). The mother maintained comparatively good health after this, apart from occasional bruising, until June, 1952, when massive spontaneous bruising appeared on her legs. There were also a few petechiae, but no other evidence of bleeding. Blood examination showed a haemoglobin at 12.7 g.%, the platelets numbered 65,000, and the bleeding-time was 11 minutes.

TABLE I.—Cases of Purpura not Responding to A.C.T.H. or Cortisone

Case	Sex and Age (Years)	Spleen Removed	Before Treatment				After Treatment				Treatment	Days
			Hb (g. %)	Platelets (per c.mm.)	Bleeding-time (Minutes)	Clinical Features	Hb (g. %)	Platelets (per c.mm.)	Bleeding-time (Minutes)	Clinical Features		
Idiopathic Thrombocytopenic Purpura												
A	F 22	Yes	6.0	30,000	8-20	Epistaxis; petechiae; bleeding gums	5.3	40,000	30+	Bleeding gums	A.C.T.H. 25 mg. i.m. 6-hourly	1-7
B*	F 29	No	(1)14.7	60,000	7-30	Bleeding gums; petechiae	12.8	55,000	8	Bleeding gums	A.C.T.H. 25 mg. i.m. 6-hourly	1-10
		†No	(2)12.0	80,000	2½-23	Bruises	12.0	40,000	7-25	Petechiae	Cortisone by mouth 100 mg. daily	1-15
											Diminishing dosage	16-22
C†	F 58	No	11.2	40,000	20	Bruises; epistaxis; purpura; melaena	10.0	40,000	7	Bruises; purpura	A.C.T.H. 25 mg. i.m. 6-hourly	1-10
D*	F 5	..	13.0	65,000	6	Bruises; purpura	13.6	65,000	6	Bruises; purpura	Cortisone orally 100 mg. 50 " 75 "	1 2-9 10-19
Non-thrombocytopenic Purpura												
E	F 9	..	11.4	260,000	15	Purpura; haematuria; oedema	11.4	Normal	—	Haematuria; oedema	Cortisone orally 50 mg. 25 " 10 "	1-5 6-8 9
											Cortisone orally 200 mg. 150 " 50 "	1-11 12 13
F	M 43	..	15.0	304,000	2	Purpura	15.0	365,000	3	Purpura	A.C.T.H. i.m. 50 mg. 75 " 100 "	13, 22 14, 21 15-20
											Cortisone 200 mg. i.m. 100 " " 100 " orally 75 " "	1 2-5 6-16 17-28
G	M 48	..	15.2	472,000	2½	Purpura; colic; melaena	15.2	450,000	3	Purpura	Cortisone by mouth 200 mg. 100 " 50 " 25 "	1 2-11 12 13
H	F 37	..	13.1	250,000	3½	Purpura; ecchymosis	13.0	220,000	2	Purpura; ecchymosis		

* Subsequently responded to splenectomy. † Purpura started 6 months after gold injections. ‡ Six months later.

Cortisone by mouth was given in a dosage of 200 mg. on the first day, followed by 100 mg. a day for eight days. During this period no further skin lesions developed; the platelet count rose to 145,000 on the seventh day of treatment, and the bleeding-time fell to 3½ minutes. Despite continuation of cortisone therapy in the same dosage the platelet count fell to 105,000 and the bleeding-time increased to eight minutes. Accordingly, the dose of cortisone was increased to 200 mg. for three days, but the platelet count did not rise again. Cortisone therapy was discontinued four days later, during which period the daily dosage was gradually reduced. During this time the platelet count fell to 20,000 and the bleeding-time rose to 10 minutes.

Summary.—This case of idiopathic thrombocytopenic purpura showed an initial temporary clinical and haematological improvement on cortisone by mouth. Haematological relapse occurred before cortisone therapy had been withdrawn.

Details of the eight cases of purpura in which hormone therapy was unsuccessful are shown in Table I.

Haemolytic Anaemia

In a previous paper (Davidson *et al.*, 1951) an account was given of three cases of haemolytic anaemia treated with A.C.T.H. Since then we have used hormone therapy in treating two further patients.

Case 7

A man aged 56 had been dyspnoeic on exertion for 18 months. Increasing tiredness was associated with the dyspnoea, but pallor was not noticed until a week before his first admission to hospital. There was no history of blood loss or of jaundice. No member of his family had, to his knowledge, been anaemic or jaundiced. On examination he was seen to be pale and slightly icteric. The liver was just palpable on deep inspiration and the lower pole of the spleen reached to within 2 in. (5 cm.) of the umbilicus. The urine contained a great excess of urobilinogen, and the serum bilirubin was 4 mg. per 100 ml. The haemoglobin level was 8 g.%, but an accurate red-cell count was difficult because of clumping of the cells. A reticulocytosis of about 15% was repeatedly found. The marrow was normoblastic. The direct Coombs test was positive and cold agglutinins were present; there was increased red-cell fragility. A sudden fall in the haemoglobin level to 5 g.%, a rise in the reticulocyte count to 26%, and a deterioration in the patient's condition indicated an increase in the haemolytic process. Accordingly he was given 2 pints (1.1 litres) of packed cells, which raised the haemoglobin level to 7 g.%. The reticulocyte count, however, remained at 26%.

Thereafter treatment consisted of injection of A.C.T.H. in a dosage of 25 mg. six-hourly for 10 days. This treatment brought a great improvement in his strength and well-being, a rise in the haemoglobin level to 10 g.%, the red cells now numbering 3,300,000 per c.mm., and a fall in the reticulocyte count to 15% and in the serum bilirubin from 4 to 1.7 mg. per 100 ml. The urine now contained only a trace of urobilinogen. The Coombs test, however, remained positive and cold agglutinins were still present.

The patient was discharged from hospital, but owing to deterioration of his condition he had to be readmitted 30 days after the A.C.T.H. therapy had been discontinued. The haemoglobin level had fallen to 6.5 g.% and the red cells to 2,530,000 per c.mm., while the reticulocyte count remained between 20 and 35%.

Treatment on this occasion was with cortisone given by mouth to a total of 925 mg. in eight days. During this period his condition deteriorated and 2 pints (1.1 litres) of blood had to be transfused. The reticulocyte count remained high, and even with blood transfusion the haemoglobin level did not rise above 6.4 g.%. Treatment was changed to A.C.T.H. given parenterally in a dosage of 25 mg. six-hourly for nine days, then 25 mg. eight-hourly for seven days. The patient showed a distinct clinical and haemato-

logical improvement, the haemoglobin level rising to 10.2 g.% and the red cells to 3,330,000 per c.mm., and the reticulocytes falling to 9.2%. A.C.T.H. therapy was continued and the spleen was removed.

The daily dose of A.C.T.H. was then progressively reduced and hormone treatment was finally terminated nine days after operation. An immediate rise in the haemoglobin level and red cell count was noted after splenectomy; this was due to autotransfusion from manipulation of the spleen at operation. A large reduction in the haemolytic process occurred during the next 14 days. A further moderate recrudescence of haemolytic activity again developed, as indicated by a fall in the haemoglobin value and a rise in the reticulocyte count, together with an increase of urinary urobilinogen. Accordingly, A.C.T.H. was again given in divided doses, 1 g. being administered parenterally over 14 days. This resulted in a steady rise of haemoglobin level to 12.5 g.%, and the red cells to 4,020,000 per c.mm., with a fall in the reticulocytes to 2.8%. The patient felt very well. Three months after the cessation of hormone therapy, when he was seen as an out-patient, there was no clinical or haematological deterioration. Reticulocytes were now 1%. The Coombs test remained positive.

Summary.—A patient with idiopathic acquired haemolytic anaemia who improved temporarily with A.C.T.H. therapy but not with cortisone by mouth in a dosage of 100 mg. daily. A definite though delayed clinical and haematological improvement resulted from splenectomy supplemented with A.C.T.H.

Case 8

This patient was a woman aged 69 with rheumatoid arthritis and anaemia. As her case is of interest in relation to the arthritis, it will be published in more detail elsewhere. The anaemia was of a haemolytic type, with reticulocytes ranging from 9 to 18%. The urine contained an excess of urobilinogen. The direct Coombs test was positive, and there was evidence of cold agglutinins in the blood. When A.C.T.H. therapy was begun the haemoglobin level was 7.4 g.% and the red cells numbered 2,400,000 per c.mm. A.C.T.H. was given in divided doses by injection, to a total of 1.42 g. in 10 days. The haemoglobin level rose to 9.5 g.% and the red cells to 2,950,000 per c.mm., but fell slightly to 8.3 g.% and 2,750,000 respectively when the A.C.T.H. therapy was stopped. Cortisone was then given by mouth in a dosage of 100 mg. daily for three days and the counts returned to the highest level obtained with A.C.T.H. (9.5 g.% and 2,950,000 per c.mm.). The dosage of cortisone was now increased to 125 mg. daily for two days, followed by 175 mg. daily for four days and then 150 mg. daily for three days. The haemoglobin level rose to 13.3 g.% and the red cells to 3,900,000 per c.mm., the reticulocytes falling to normal levels. The Coombs test remained positive. A reduction in the dosage of cortisone resulted in an immediate fall in the level of the haemoglobin and red-cell counts.

Summary.—A case of acquired haemolytic anaemia in a patient with rheumatoid arthritis in which there was temporary benefit both with A.C.T.H. and with cortisone therapy.

Miscellaneous Group

The remaining patients consisted of four cases of aplastic anaemia, one of which was due to gold therapy, one case of myeloid leukaemia, two of acute leukaemia in adult patients, and one infant with eosinophilia associated with a grossly enlarged spleen. There was no clinical benefit in any of these patients, and, as will be seen from Table II, the blood picture was unchanged.

Discussion

In three cases of thrombocytopenic purpura of the present series there was a clinical and haematological "cure" following treatment with A.C.T.H. or cortisone supplemented by blood transfusion. The illness in all three

TABLE II.—Miscellaneous Blood Disorders not Responding to A.C.T.H. or Cortisone

Case	Diagnosis	Sex and Age	Before Treatment						After Treatment						Days	
			Hb (g%)	R.B.C. (mil. per c.mm.)	W.B.C. (per c.mm.)	Retics. (%)	Platelets (per c.mm.)	Other Findings	Marrow	Hb (g%)	R.B.C. (mil. per c.mm.)	W.B.C. (per c.mm.)	Retics. (%)	Platelets (per c.mm.)		Other Findings
S	Aplastic anaemia (after gold therapy)	M 34	8.0	2.41	2,500	<1	30,000		Reactive	5.3	1.35	1,750	1	15,000		A.C.T.H. 25 mg. i.m. 6-hourly Cortisone i.m. 300 mg. 200 " 100 " (2 pints blood transfused) Cortisone i.m. 300 mg. 200 " 100 " 1
T	Aplastic anaemia	F 49	6.3	1.85	1,700	<1	37,000		Aplastic	6.3	1.98	3,200	<1	60,000		2 3-13
U	Aplastic anaemia	M 61	3.7	1.13	2,200	4.2	—		Reactive	3.0	0.79	4,800	7.5	—		1 2 3-13
V	Aplastic anaemia	F 51	9.6	2.58	2,400	<1	20,000		Aplastic	9.9	3.56	2,200	<1	20,000		6-hourly Cortisone by mouth 50 mg. 100 " 200 " (4 pints blood transfused) A.C.T.H. 25 mg. i.m. 6-hourly Cortisone by mouth 200 mg. 100 " 200 " 1-6
W	Acute myeloid leukaemia	F 32	7.3	2.81	24,000	<1	25,000	Blast cells, 32%	Chiefly myeloblasts and myelocytes	9.2	3.73	69,000	<1	25,000	Blast cells, 40%	200 mg. 100 " (2 pints blood transfused) Cortisone by mouth 200 mg. 100 " 1 3-14
X	Acute lymphatic leukaemia	F 21	6.0	2.36	8,000	10	35,000		Lymphocytes and lymphoblasts	4.4	1.63	42,600	18.4	30,000		(7 pints blood transfused) A.C.T.H. 25 mg. i.m. 6-hourly Cortisone 20 mg. by mouth 38% 1-10
Y	Eosinophilic leukaemia	M 1	10.0	—	30,000	<1	—		Eosinophils, 53% Coombs test positive	10.0	—	25,000	—	—		Cortisone 20 mg. by mouth 6-hourly A.C.T.H. 25 mg. i.m. 6-hourly Cortisone i.m. 100 mg. 200 " 50 " 1-9
Z	Myeloid leukaemia	M 26	(1) 6.3 (2) 6.3	2.2 2.3	1,900 2,600	<1 <1	220,000		Some fibrous replacement	7.0	2.5	2,600	<1	195,000		Cortisone i.m. 100 mg. 200 " 50 " 1-2, 5, 6 3, 4, 7-10 11, 12

patients had been of short duration, and therefore it is not possible to be certain to what extent the hormone therapy was responsible for the improvement which occurred. Indeed, in one case the platelet rise did not occur until more than a month after A.C.T.H. therapy was terminated. A fourth patient, who had not responded to splenectomy for idiopathic thrombocytopenic purpura, showed no improvement with 100 mg. of A.C.T.H. daily for 10 days. She did, however, develop a temporary but significant increase of platelets from the administration of 800 mg. of cortisone by mouth over a period of seven days. A fifth case—a woman with idiopathic thrombocytopenic purpura—repeatedly responded to small courses of A.C.T.H. by injection, but when cortisone was given by mouth in the same dosage as the preceding course of A.C.T.H. the platelet response was much higher. It was noted in this same patient that withdrawal of cortisone led to an increase of the bleeding-time over its previous level. A sixth case showed a significant but temporary response to cortisone by mouth.

The remaining four patients with idiopathic thrombocytopenic purpura, and four with non-thrombocytopenic purpura, apparently of an allergic type, did not respond to hormone therapy. One of the latter group, however, who suffered from Henoch's purpura, was quite certain that his abdominal colic was rapidly relieved by cortisone although the purpura was unchecked. This patient, who was a doctor, was satisfied that muscular weakness was felt during cortisone therapy if he did not take the potassium citrate that had been prescribed.

There seem to be no satisfactory clinical or haematological criteria which would enable an accurate forecast to be made whether any particular case of thrombocytopenic purpura will or will not respond to hormone therapy. It is, however, clear that a failure to respond to such treatment does not indicate that splenectomy will not be successful (see Cases B and D, Table I). Moreover, as already mentioned, a case of thrombocytopenic purpura which has failed to respond to splenectomy can show a response to cortisone (Case 4).

In two patients with acquired haemolytic anaemia there was a response to A.C.T.H. or cortisone, but this improvement was transient.

No beneficial results were obtained in a small group of patients suffering from aplastic anaemia, acute leukaemia, or myeloid leukaemia.

Summary

A.C.T.H. or cortisone was administered to 14 cases of purpura, to 2 cases of idiopathic acquired haemolytic anaemia, and to a small group of patients suffering from aplastic anaemia, leukaemia, or myeloid leukaemia.

Three cases of thrombocytopenic purpura of short duration were apparently cured by hormone therapy.

Temporary beneficial effects were produced in three cases of idiopathic thrombocytopenic purpura and two cases of idiopathic acquired haemolytic anaemia. Such remissions may be of value in allowing splenectomy to be undertaken with greater safety.

No beneficial results were obtained in the remaining eight cases of purpura and in a small number of patients suffering from a diverse group of blood disorders.

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