A.C.T.H. A week later she had a sudden collapse with very weak pulse and some oedema of the legs; her blood sugar varied between 40 and 60 mg. per 100 ml., and her serum potassium was 32 mg. per 100 ml.

Summary

Three patients with hypopituitarism were treated with cortisone for 12 to 20 days. There was a pronounced clinical and biochemical improvement, though a peculiar psychological change occurred after about two weeks. This treatment seems to hold out much promise for the future.

Two patients with hypopituitarism were treated with A.C.T.H.—one for 7 days, the other for 15 days. There was a slight clinical and biochemical improvement, but after a few days both patients developed migraine and hemiparesis, and one became comatose. It seems advisable that A.C.T.H. should be employed only with the greatest caution in the treatment of hypopituitarism.

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During 1949 Unesco organized an experiment in West China to prepare a wide range of visual aids for use in fundamental education (*The Healthy Village*, Unesco, Paris). Dr. Clara Nutting writes about the work of the health department, and describes among other things how health filmstrips were made. The first topic chosen was smallpox. The greatest difficulty lay in explaining to the native artists how to teach the prevention of disease. Next they attacked the problem of trachoma, the cause of 80% of all eye infections in North China, most of which could be avoided if the people were educated to simple health habits. The artists were shown several cases, and followed the treatment; this enabled them to understand the medical side and thus to express their impressions in pictures. Filmstrips on tetanus neonatorum followed, then on safe food and water and on health habits. In the practical field vaccinations were performed in the township of Huang Ke Chen and the rural districts surrounding it; the people co-operated readily. Children under 16 formed 80% of those vaccinated. But there is still need for more education, says the report, as many people never reappear for further vaccination. Filmstrips on trachoma were shown, and all those who reported that they had the disease were registered; market days were chosen for giving examinations and treatment in the town, and the others were devoted to work in the rural districts. Education is still the most essential need, for until the people become trachoma-conscious treatment can never be really effective. Here, as in other diseases, filmstrips and pictures are of great value in teaching preventive medicine.

EFFECT OF CORTISONE ON CRISIS IN SIMMONDS'S DISEASE

BY

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AND

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After conversation with Professor Sheehan we decided that it was worth recording these notes on the response of a patient with Simmonds's disease, in crisis, to cortisone.

Case History

The patient developed Simmonds's disease after her third pregnancy in 1947. Her pregnancy and confinement were normal, but her milk supply stopped after a few weeks and was followed by an amenorrhoea. She then became very tired and lost interest in everything; there was complete loss of libido. On two occasions her 17-ketosteroids were estimated at 1.9 and 2.7 mg. a day. She had been treated with methyl testosterone, 10 mg. thrice daily, thyroid, 1 gr. (65 mg.) daily, and "doca linguets," three daily, with considerable improvement. Her normal blood pressure was 115/75. She was admitted on March 27 for implantation of hormones: the previous therapy was temporarily discontinued.

On admission she was listless but showed no abnormal physical signs. Her blood pressure was 100/60. On March 29 she had nausea and vomited during the night; her temperature went up to 103° F. (39.4° C.); she became very uncooperative and incontinent; and her blood pressure was 84/56. Her condition worsened and she became very drowsy and negativistic and would not answer questions. She lay curled up. Her blood electrolytes at this time were found to be normal: sodium, 137 mEq. per litre; potassium, 4.4 mEq. per litre; chlorine, 100 mEq. per litre; blood urea, 41 mg. per 100 ml. A saline drip was started and treatment with doca, 10 mg. followed by 5 mg. twice daily, was begun. "Eucortone," 15 ml. followed by 10 ml. four-hourly, was also given. Her blood sugar at midnight was found to be 53 mg. per 100 ml. The drip was changed to glucose saline. The blood sugar was still only 41 mg. per 100 ml. at midday on March 30. Her temperature remained high, rising up to 103° F. (39.4° C.). Her condition was otherwise unchanged.

On March 31, although the blood sugar had risen to 107 mg. per 100 ml., her condition was essentially unchanged. Her mental attitude was still childish and she was uncooperative and irritable. Her temperature was still up to 102.5° F. (39.2° C.). Cortisone was therefore started, 100 mg. being given at midday and at 6 p.m. In view of the pyrexia procaine penicillin, 300,000 units twice a day, was also started.

At midday on April 1 the patient's mental condition was much better and by 6 p.m. she was much more rational, though she was still incontinent. The highest temperature during the day was 100.5° F. (38.1° C.). Cortisone, 200 mg., was again given during the day. Next day she was quite reasonable and continence was returning. Cortisone, 100 mg. daily, was continued for a further four days and penicillin was discontinued. On April 3 her temperature was normal and she was well. Her subsequent progress was satisfactory.

Pellets of doca and testosterone were implanted on April 20, since when she has remained well.

Comment

This patient in severe Simmonds's disease crisis showed very little improvement when treated with eucortone, doca, and glucose saline, even after 48 hours, by which time the blood sugar had returned to normal. Within 24 hours of starting treatment with cortisone there was a marked improvement, which was thought to be associated with the cortisone. The cause of the patient's pyrexia is uncertain. There were no definite physical signs, apart from a few adventitious sounds in the lungs, and it seems unlikely that the toxæmia was due to infection and that the penicillin had anything to do with the dramatic recovery. It cannot, of course, be said with certainty that cortisone was responsible for the improvement. The other hormones had been given for some 48 hours previously, and it is possible that improvement was due to a delayed effect of the previous therapy, though the prompt response to cortisone following the very little change which had taken place during the previous therapeutic regime raised the strong possibility that the recovery was due to cortisone.

It is realized that this case is very far from proof that cortisone is a drug of choice in the treatment of acute Simmonds's disease crisis, but these observations have been recorded so that a wider trial can be given in future cases.

A CASE OF SIMMONDS'S DISEASE WITH MENTAL SYMPTOMS

BY

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The recent advances in endocrine therapeutics have revived interest in the disorder known as Simmonds's disease. In the past the missed diagnosis was often condoned, if not excused, by the fact that no effective treatment was available, but this no longer holds true. Furthermore, the diagnostic difficulties have been considerably lessened by the brilliant studies of Sheehan, whose portrayal of the typical case is unlikely to be surpassed. The earlier textbook descriptions were misleading in that undue stress was laid on such inconstant signs as progeria, cachexia, and hypotension, whereas insufficient notice was taken of the accompanying mental disturbance. Sheehan and Summers (1949), in a full review of the disease, write: "In the later stages a number of patients present mental symptoms and are removed to asylums, where attention is focused more on the psychological than the physical aspects of the condition."

The case described below is of especial interest in that it was the mental, not the physical, symptoms which brought the patient into hospital.

Case Report

A married woman aged 54 was admitted to hospital on February 3, 1950. She complained of depression, general weakness, and cramping pains in the calves. She was married in 1925 at the age of 29, and had had two uncomplicated pregnancies, in 1926 and 1929. In 1933 the birth of a still-born child was followed by a severe haemorrhage, for which she was given an infusion of saline but no blood transfusion. Permanent amenorrhoea followed the post-partum haemorrhage. Coitus ceased as a result of both a loss in sexual

appetite and a progressive shrinking of the vagina. Her axillary and pubic hair gradually disappeared, her head hair became dry and coarse, while her eyebrows grew steadily thinner. Her skin became dry and scaly. An insidious decline in her general health took place *pari passu* with the changes already described.

Since 1940 she had been subject to attacks of fainting and vague abdominal pain. From 1945 onwards her family noticed signs of increasing mental torpor and physical deterioration. She became slow in her speech and in the performance of simple routine tasks. She developed an inability to tolerate the vicissitudes of the climate, being prostrated by very hot or cold weather. Her cheeks, which were florid prior to the haemorrhage, assumed a remarkable pallor. Despite her failing health, she remained fairly cheerful until a few months before admission, when she suddenly became anxious and depressed. No marked loss of weight had taken place.

On admission she was noted to be in a fair state of nourishment. A striking degree of facial pallor gave rise to the suspicion that she had a severe degree of anaemia. Her eyebrows were scanty and the axillary and pubic hair absent (Fig. 1). Her skin was dry and wrinkled and showed a fine scalliness. Her fingernails were thin, dry, and brittle; they were not spoon-shaped. No abnormality was noted in the chest, abdomen, or central nervous system. The blood pressure was 140/80. The vagina admitted only the index finger. Her weight was 6 st. 4 lb. 10 oz. (40.2 kg.). Progeria was not in evidence. Pigmentation of the skin was absent, but erythema ab igne was conspicuous on the legs. The mental picture was one of slight retardation with depression and anxiety.

FIG. 1.—Photograph taken on March 22 (before treatment), showing scanty eyebrows and complete absence of pubic and axillary hair. Erythema ab igne is well shown on legs.

Investigations

On February 4 a blood count gave: red cells, 4,010,000 per c.mm.; haemoglobin, 85%; colour index, 1; white cells, 5,250 per c.mm. (polymorphs 28%, lymphocytes 67%, monocytes 3%, eosinophils 1%, basophils 1%). The corrected B.S.R. was 12 (Wintrobe). The W.R. was negative. X-ray studies of the skull (including the sella turcica), heart, and lungs showed no abnormality.

The 24-hour urinary excretion of 17-ketosteroids was 2.3 mg. The fasting blood-sugar and sugar-tolerance curve gave a fasting value of 100 mg. per 100 ml.; half an hour after 50 g. of glucose it was 100 mg.; one hour later, 90 mg.; one and a half hours later, 80 mg.; two hours later, 80 mg.; two and a half hours later, 80 mg.

On March 27 the following results were obtained by a biochemical study of the blood: blood urea, 20 mg./100 ml.; blood cholesterol, 234 mg./100 ml. Serum sodium, 285 mg./100 ml.; serum potassium, 17.7 mg./100 ml.; serum