and facilitate the removal of any scabs or scales that may be present. The occurrence of secondary infection is treated on general principles, but these cases were few in this series, which indicates the high standard of the school medical organization in detecting cases at an early stage.

With the factors given above, epilation has always been on the whole satisfactory. Occasionally small tufts of hair have remained in the centre of a well-epilated area or towards the periphery of the scalp. In most cases the hairs are loose and can be easily removed by a pair of forceps. Sometimes, however, these tufts resist extraction, but since infection of these hairs is not present, as shown from the following results, recontamination of the growing hair never follows and they can be ignored. No complications result from therapy except occasional headache and vomiting on the night of treatment, and there were no cases of alopecia, local or general.

Review of Cases

To check the results of treatment carried out on the lines described above, follow-up cards were sent to 103 patients whose epilation dated from eight months and over requesting them to attend the x-ray department for review. These 103 cases comprised: 71 with a laboratory diagnosis of *Microsporon audouini*, 27 with a laboratory diagnosis of ringworm fungus, and 5 diagnosed on clinical grounds and by Wood's light.

Forty-eight of these patients responded in person and were examined by us clinically and by Wood's light. They all proved to be free from infection and their hair and scalp were in excellent condition. Of the remaining 55 patients, 13 had left London and could not be contacted, and 42 were examined at their schools by the courtesy of the medical officer of the S.E. and S.W. divisions, who kindly forwarded reports on the condition of these patients. All without exception were completely well and their hair and scalp were described as being in a healthy state.

From these figures it can be stated that out of 103 patients with ringworm of the scalp who received no treatment other than x-ray epilation 90 (87.37%) showed no signs of relapse after an interval of time ranging from eight to twenty-four months. These 90 were made up as follows: 68 cases of *M. audouini* infection, 19 of ringworm fungus, and 3 diagnosed clinically and by Wood's light. It should be added that as no complaints have been received concerning the other 13 cases it can be assumed on fairly certain grounds that they too have remained free from relapse. This being so, the total percentage of cures may have reached 100.

Time Lost from School.—To date it has been our policy to keep the children away from school for about two to two and a half months, when the new hair showed signs of regrowth. We feel that in view of the above results an earlier return is possible, as the loss of a full term is often a serious handicap to these children. Perhaps, with a good epilation, six weeks would be sufficient in the great majority. Further work will be necessary, however, before a final decision can be taken on this point.

Summary

One hundred and three cases of tinea capitis treated solely by x-ray epilation are reviewed after an interval of eight to twenty-four months.

The results show that local application of fungicides is unnecessary—at least in cases of *M. audouini* infection. It may be added that the fact that the cases were consecutive and were free after the lapse of such an interval is ample justification of the view of the late Mr. G. Stebbing, who held that local

treatment was unnecessary, and that although x rays were not parasitocidal they either had the power to reduce the vitality of the fungus or brought about some tissue change which made it less easy for chance spores remaining to contaminate the new crop of hair.

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FULMINATING MENINGOCOCCAL SEPTICAEMIA

AN ACCOUNT OF THREE CASES, WITH ONE RECOVERY

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Fulminating septicaemia is one of the rarer manifestations of meningococcal infection. It occurred in 24 (3.4%) of Banks's (1948) series of 706 cases, and American authors give its incidence as about 1% (Sweet, Dowling, and Howell, 1947).

Though it may be preceded by a few days of mild infection of the upper respiratory tract, the onset is usually sudden, with fever, rapid respirations, and cyanosis, simulating early pneumonia. Petechiae appear within a few hours, and quickly enlarge to form purpuric blotches. Peripheral vascular failure occurs early, with a rapid feeble pulse and very low blood pressure. Meningism is absent at first, but may appear terminally. In untreated cases death occurs in 24–36 hours. Necropsy often reveals adrenal lesions, the most striking of which is massive bilateral haemorrhagic destruction (sometimes known as the Waterhouse-Friderichsen syndrome).

Since 1940, reports of successful treatment with sulphonamides and adrenal cortical extract have been published. With the additional help of penicillin Banks (1948) believes 50% of cases may recover.

We saw three sporadic cases within a year at a general hospital. One of these recovered.

Case 1

A female laundry worker aged 21 was quite well till 2.30 a.m. on Jan. 27, 1946, when she awoke with pains in her legs, which grew rapidly worse and spread to her whole body. Her throat became sore, and during the morning dark blotches appeared on her trunk and limbs.

She was admitted to hospital at 3.30 p.m., and was seen to be a well-built woman, gravely ill. The temperature was 102° F. (38.9° C.), pulse 112, and respirations 24. Purpuric blotches involved the face, neck, trunk, and limbs, and also the buccal mucous membrane and conjunctivae. The pulse was rapid, feeble, and regular. Blood pressure was 75/55. There were no signs of meningism and the urine was normal. White blood cells totalled 15,000 (83% polymorphs). Bleeding and clotting times and capillary resistance were normal.

Her condition deteriorated rapidly, the pulse became imperceptible, and the blood pressure too low for estimation. She sank into a coma, and a trace of neck rigidity was noted. The pupils were irregular and the limbs flaccid. At 5.15 a.m. next day the cerebrospinal fluid was turbid. Cells numbered 13,000 (99% polymorphs). Gram-negative intracellular diplococci

were found in deposit. Culture was sterile. She died at 6.40 a.m., 28 hours after the onset.

Necropsy was performed by Dr. D. J. O'Brien five hours after death. Widespread partly confluent intracutaneous haemorrhages from pin-point size to 15 cm. diameter were seen. There was thin pus over both cerebral hemispheres, but not at the base of the brain. Petechiae were present in parietal and visceral pleura, pericardium, and peritoneum. A small fleshy thymus gland was found. The spleen was twice the normal size. The right suprarenal was congested; the left had small haemorrhages in the medulla. No lesion of the muscles was noted.

Microscopical Examination.—The thymus was normal. The adrenals showed congestion of all capillaries but no frank haemorrhage. Cells of outer layers of cortex of both glands appeared necrotic. Diplococci were seen in films of the pus over the brain, and meningococci (Type I) were cultured from the heart blood.

Death was considered to be due to fulminating meningococcal septicaemia.

Case 2

A boy aged 3 was taken ill at midday on Oct. 11, 1946. At 7 p.m. he was febrile and breathless, and was sent to hospital as a case of early pneumonia.

On admission at 10.30 p.m. he looked severely ill, with grey cyanosis. The temperature was 104° F. (40° C.), pulse 160, and respirations 44. There was a trace of neck rigidity. The lungs were normal. A petechial rash was seen on the upper part of the trunk. During the next hour this rash developed with great rapidity until the whole trunk and limbs were covered with purpuric blotches. A clinical diagnosis of fulminating meningococcal septicaemia was made, and courses of penicillin, 30,000 units intramuscularly three-hourly, and oral sulphadiazine, 1 g. followed by 0.5 g. four-hourly, were begun. C.S.F.: crystal-clear; pressure, 120; no cells; protein, 20 mg. per 100 ml.; no organisms seen; culture sterile. Penicillin, 15,000 units, was injected intrathecally.

At 8 a.m. on Oct. 12 his condition was obviously much worse. The temperature was 106° F. (41.1° C.), pulse uncountable, and respirations 72. Blood pressure was too low for estimation. Neck rigidity was definite and Kernig's sign positive. C.S.F.: turbid; pressure, 140; cells, 3,200 per c.mm. (99% polymorphs); protein, 180 mg. per 100 ml.; no organisms seen; culture sterile. Penicillin, 15,000 units, was injected intrathecally. White blood cells numbered 33,000 (polymorphs 80%). Scrapings from a purpuric patch were cultured, but pathogenic organisms were not isolated. It was now thought that acute adrenal insufficiency might well have developed, and though the child appeared moribund it was decided to try the effect of administering adrenal cortical extracts (22 hours after the onset). The possibility of the cellular reaction in the C.S.F. being the result of previous intrathecal penicillin was also considered.

At 10 a.m. desoxycortone acetate, 5 mg., was injected intramuscularly. An intravenous infusion of 5% dextrose in normal saline was set up, and three-hourly intravenous injections were given by the infusion tubing of: 2.5 ml. of "eucortone"; 0.1 ml. of 1 in 1,000 solution of adrenaline hydrochloride; and 0.5 g. of soluble sulphadiazine. At 2 p.m. the temperature was 99.4° F. (37.55° C.), pulse 120, and respirations 48. Very great improvement was obvious. His colour was rosy, the pulse was easily felt, and the blood pressure was 100/60. Mild neck rigidity and Kernig's sign were still present.

On Oct. 14 there was no return of peripheral vascular collapse. A trace of oedema of hands and feet was present, which was considered to be due to excess of adrenal cortical extract and saline. Infusion and eucortone were therefore discontinued, after totals of 4 pints (2.3 litres) and 45 ml., respectively. Sulphadiazine, 0.5 g. three-hourly orally, and intramuscular penicillin were continued. On Oct. 16 progress was satisfactory, with only a trace of neck stiffness. C.S.F.: clear fluid, sterile; pressure, 180; cells, 360 (99% polymorphs); protein, 35 mg. per 100 ml. On Oct. 18 the purpuric patches were fading, but seven of the large ones on the back and

buttocks were sloughing. Chemotherapy was stopped after a total of 1,180,000 units of penicillin and 33 g. of sulphadiazine had been given.

Convalescence was delayed by infection of the sloughed purpuric patches and of the infusion wound, but this responded to a further course of penicillin.

In May, 1947, he was quite normal for his age, the only sequelae being the scars of his infusion wound and sloughs. A radiograph of his abdomen showed no evidence of calcification of the adrenals.

Case 3

A schoolgirl aged 15 was well till Jan. 26, 1947, when she complained of a cold in the head. At bedtime she felt "shivery" and had an aching pain in the right shoulder and arm. At 1.30 a.m. on the 27th she vomited, and at 5.30 a.m. her parents observed her breathing to be rapid and shallow. She talked incoherently in her sleep. At 10.30 a.m. a rash appeared on the face and limbs consisting of "black spots which rapidly enlarged into blotches." At 2 p.m. the temperature was 102° F. (38.9° C.) and respirations were rapid. A purpuric rash was present on the face, neck, and limbs. There was no neck rigidity. Admission to hospital was requested.

On admission at 6 p.m. her temperature was 100° F. (37.8° C.), pulse imperceptible, and respirations 40. She was conscious and rational and complained only of pain in the right shoulder and weakness. Purpuric blotches and petechial spots were seen on the face, neck, arms, and legs, but not on the trunk. The heart sounds were faint—the rate was 140. The blood pressure was unobtainable. The lungs and abdomen were normal. There was dubious neck rigidity, Kernig's sign was not obtained, and the reflexes were absent.

A diagnosis of fulminating meningococcal septicaemia with adrenal haemorrhages was made, and 10 ml. of adrenal cortical extract was injected intramuscularly. Owing to the peripheral vascular collapse it was impossible to administer this intravenously; nor was it possible to obtain blood for culture and biochemical examination. Immediate preparations were made to set up an intravenous saline infusion and to begin therapy with penicillin and sulphadiazine. At 6.45 p.m., however, 20 hours after the onset of the disease, she died, remaining clear-minded to the end.

Necropsy was performed by Dr. Raeburn 20 hours after death. The body was that of a well-developed girl with an extensive purpuric rash on the face, neck, shoulders, arms, and legs. The body was free. The myocardium was pale, soft, and swollen on section. A few petechiae were seen beneath the endocardium. The entire respiratory tract was intensely congested. The mucosa of the trachea and bronchi was bright red. The lungs were markedly congested and semi-airless; minute haemorrhages could be distinguished in the parenchyma. The whole alimentary tract was congested, with numerous subserous petechiae on the stomach and small intestine. The thyroid gland, pancreas, ovaries, and kidneys were all congested. The adrenals were congested, but there was no gross haemorrhage, and the structure of the glands seemed intact. They were no more strikingly congested than the thyroid, pancreas, and kidneys, and less so than the lungs. The thymus was large and fleshy, weighing 42 g. The leptomeninges of both the brain and the spinal cord were hyperaemic, and there was a little fibrinous exudate in the sulci. The brain substance was hyperaemic and slightly oedematous. Blood obtained from the right ventricle was cultured and a profuse growth of meningococci (Type I) was obtained. The same organism was isolated from a swab taken from the sphenoid sinus.

Histological Examination.—The adrenals showed congestion of the vessels, with a number of minute haemorrhages. The spinal cord exhibited changes typical of leptomeningitis. The thymus showed normal glandular tissue, and the kidneys, pituitary, and thyroid congestion only. The lungs showed intense dilatation and congestion of the capillaries with secondary atelectasis.

Death was considered to be due to fulminating meningococcal septicaemia.

Discussion

Banks and McCartney (1942, 1943) classify fulminating meningococcal septicaemia into an encephalitic type, characterized by mental clouding progressing to coma; an adrenal type with purpuric rashes, peripheral vascular failure, and mental clearness; and a mixed type, having some of the features of the other two. Our cases are examples of the pure adrenal type, and the clinical picture is characteristic and easily recognized. Meningococci were not recovered from Case 2, probably owing to the early administration of penicillin and sulphadiazine.

In neither fatal case was there gross destruction of the adrenal glands, though congestion was seen in both and microscopic haemorrhage in one. Williams (1942) noted the absence of gross adrenal lesions in eight out of 17 clinically typical cases, and suggested that adrenal deficiency might not be an important factor in the pathogenesis of the syndrome.

Kinsman, D'Alonzo, and Russi (1946) describe a condition of "tubular degeneration" occurring in two fatal cases, without adrenal haemorrhages. The cortical cells appeared shrunken and fat globules were absent, so that the "cords" of cells usually seen looked like tubes containing fibrinous debris. They suggest that this condition is the result of adrenal exhaustion due to increased demand for the lipid hormone during the acute infection, and, further, that irreversible damage has not occurred at this stage.

Banks (1948) states that necropsy material from all his fatal cases showed "significant" adrenal lesions. We were impressed by the dramatic improvement which followed intravenous saline and cortical extract in Case 2. Since there was no dehydration and there had been no vomiting, it is difficult to believe that the infusion of 150 ml. of saline alone accounted for this improvement. We were convinced that the cortical extract was responsible. We agree, therefore, with Banks (1948) that, though toxic and other factors may be concerned, there is justification for regarding acute adrenal deficiency as playing a leading part in the pathogenesis of this syndrome and for treating it accordingly.

The thymus gland was found to be persistent in Case 1 and larger than normal in Case 3. In both cases it was of normal microscopical structure. A similar enlargement and persistence have been recorded by Magnusson (1934) and Holmes and Cowan (1945). The rapid course of the illness makes it evident that the enlargement preceded infection and suggests the existence of a constitutional type with a large thymus and small fragile adrenals in whom resistance to meningococcal infection is lower than normal.

When a clinical diagnosis has been made treatment must be immediately instituted for septicaemia, peripheral circulatory failure, and adrenal insufficiency.

Treatment of the Septicaemia.—The meningococcus is susceptible to sulphonamides and penicillin. It is advisable to employ both in order to control the septicaemia as rapidly and completely as possible. Any sulphonamide can be used, but, owing to the lower risk of producing renal complications, sulphamezathine is probably the drug of choice. A blood level of 15 mg. per 100 ml. should be maintained. The initial doses should be given intravenously, and for a young adult the dosage should be 4 g. immediately and 2 g. four-hourly until facilities for control by estimation of the blood level are available. Penicillin intramuscularly in full doses should be started immediately. Case 2 received 30,000 units three-hourly, but now that penicillin is more readily available much larger doses should be employed—e.g., 250,000 to 500,000 units immediately, followed by 100,000 units three-hourly. Even with such

doses it is hazardous to increase the time interval between injections, as a constant strongly bacteriostatic blood-penicillin level is essential in so serious an illness.

Peripheral Circulatory Failure.—An intravenous infusion of normal saline should be set up immediately. In acute adrenal deficiency the blood volume is reduced and the blood viscosity increased by loss of sodium and water into the tissues and by excretion into the urine. Logically the infusion should therefore be normal saline rather than plasma or whole blood.

Adrenal Deficiency.—Adrenal cortical extract B.P.C., or one of the proprietary preparations such as eucortone or eschatin, should be given intravenously as soon as possible. The initial dose should be 10 ml., and this should be repeated four-hourly until the systolic blood pressure is maintained above 100 mm. Hg. If the extract is not immediately available, desoxycortone acetate can be used in doses of 10 mg. eight-hourly, intramuscularly. Adrenaline hydrochloride, 3 min. (0.2 ml.) subcutaneously, six-hourly, can be given, but it is of doubtful benefit. Ascorbic acid, 500 mg. intravenously, twice daily, may be helpful. The adrenals contain much ascorbic acid, but its function is unknown, and its therapeutic use here is empirical. Vitamin K analogue—menaphthone—one 5-mg. ampoule, should be given initially to ensure a normal prothrombin level. The order of treatment should be: (1) begin intramuscular penicillin injections; (2) set up an intravenous drip-infusion of normal saline; (3) begin intravenous sulphamezathine, adrenal cortical extracts, and ascorbic acid via the infusion tubing; and (4) give adrenaline hydrochloride and menaphthone.

Summary

Three cases of fulminating meningococcal septicaemia are described. One of them, a child aged 3, recovered after treatment with penicillin, sulphadiazine, and adrenal cortical extracts.

This condition occurs sporadically as well as during epidemics, and presents as a severe illness of sudden onset, with a purpuric rash and profound peripheral circulatory failure. In untreated cases death occurs in 24–36 hours.

At necropsy, adrenal lesions are always found. Acute adrenal deficiency is probably a factor in the fatal outcome.

Diagnosis must be made on clinical grounds. Treatment should be started immediately with penicillin, sulphamezathine, intravenous saline, and adrenal cortical extracts, without awaiting laboratory investigations. If it is begun early enough there is a reasonable chance of recovery without sequelae.

We are indebted to Dr. P. Hamill, under whose care these cases were admitted to hospital, for permission to publish this report and for help and encouragement in its preparation.

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The Hospital Savings Association announces that Mr. Thomas Denman, its general secretary, retired on March 1. His early experience was in the office of a local authority, then on the passing of the National Insurance Act of 1911 he became clerk to an insurance committee. In 1919, seeking the greater freedom and flexibility of a voluntary organization, he joined the late Dr. Gordon Dil, of Brighton, in founding and organizing the Sussex Provident Scheme. It was from this scheme that he developed the idea of the H.S.A. He was appointed financial secretary on its incorporation in 1922 and from the first has been responsible for all the financial arrangements. In 1939 he became joint secretary and in 1945 general secretary. His successor will be Mr. C. S. Clarke, who joined the staff in 1931 and has been deputy general secretary since 1946.