correlation coefficient is 0.84, but there is a slight overall tendency for the tablets to give a high reading. In practice it was found that the mid-point between 100 and 150 mg./100 ml. gave a colour that could be recognized quite satisfactorily, and some readings of 125 mg./100 ml. are therefore shown. The mid-point 150–200 mg./100 ml. proved less easy to detect. Occasionally an impure colour was obtained and it was found necessary to repeat the test.

Discussion

A rapid and simple method for the performance of blood-sugar estimations in the out-patient clinic, consulting-room, or ward side-room would help the control of treatment of diabetes and in the management of diabetic emergencies.

This method cannot be recommended even as a screening procedure for the diagnosis or exclusion of diabetes. Definite diagnosis of this condition is of great significance to the patient and only methods of proved accuracy should be used. The tablet method is not really suitable for control of treatment in hospital diabetic clinics, where arrangements can usually be made for blood-sugar estimations by routine methods. It may be helpful for practitioners supervising the treatment of diabetics in their surgeries, an occasional check on the blood being desirable in all cases.

The method has proved very useful in the management of diabetic emergencies in hospital. In post-operative patients, in cases of ketosis, and in cases of suspected hypoglycaemia a rapid guide to treatment has been obtained. At night and at week-ends its use has often made it unnecessary to call on the services of the resident biochemist. In hospitals without a 24-hour biochemistry service this equipment would be very valuable.

I am indebted to the staff of the routine laboratories of the Courtauld Institute of Biochemistry of the Middlesex Hospital, where the control estimations of the blood sugar were made. I thank Dr. J. D. N. Nabarro, under whose guidance this paper was written, for allowing me to perform these tests on his patients, and the Ames Company (London) Ltd. for the provision of the blood-sugar kit.

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Medical Memoranda

Treatment of Ammoniacal Dermatitis with Folic Acid and Soya-bean Milk

The oral administration of either folic acid or soya-bean flour encourages the growth of lactobacilli and possibly inhibits the growth of ammonia-producing organisms in the stools. It would seem rational to try to cure ammoniacal dermatitis in infants by the oral administration of these substances.

The following report concerning the treatment of chronic napkin rash by the administration of (a) folic acid and (b) soya-bean milk may prove of value to others faced with a similar problem.

Five infants aged between 3 and 16 months, who had suffered from severe ammoniacal dermatitis in which there was desquamation and exudation for periods ranging from 1 to 12 months, and who failed to respond to routine methods of treatment, were given folic acid by mouth. The dosage was 10 mg. daily as an elixir. Routine treatment to napkins and buttocks continued unchanged, while in each case the infant's diet remained similar to that in use immediately before the start of folic acid therapy. After a month three patients showed slight improvement, but the other two were no better. In view of this unsatisfactory response to treatment folic acid was withheld and soya-bean

milk was given. The milk consisted of 1½ oz. (42 g.) of soya-bean flour added to a pint (570 ml.) of water. This was used to replace half the quantity of cow's milk normally taken daily, so that patients consumed 10 to 20 oz. (285 to 570 ml.) of soya-bean milk each day. The routine treatment of the napkins and buttocks continued as before and the infants' diets were not altered. Within five days of starting the treatment all the patients had shown some improvement in the condition of the buttocks, while four of them had become more contented. Within two weeks the appearance of the skin was normal in all cases. During the third and fourth weeks of treatment there was no deterioration, though very transitory erythema of the skin in the napkin area was sometimes noted.

In view of these findings further observations on the effect of treatment with soya-bean milk in cases of ammoniacal dermatitis would be useful, and it should be given a trial in all resistant cases of this condition. Soya-bean flour can be obtained from Soya Foods, Ltd., London, E.C.3, and possibly from grocers. Its food value compares favourably with cow's milk, so that it is possible to rear infants from birth on this food. The cost of soya-bean milk is approximately twopence a pint. It is possible that treatment with larger doses of folic acid than were used in these few cases might prove more satisfactory, and it may be that cases resistant to soya-bean therapy might respond to large doses of folic acid.

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Pregnancy in Addison's Disease

Adrenocortical insufficiency is not necessarily incompatible with fertility. In earlier days the hazards of pregnancy in such circumstances were well known and were such as to make conception inadvisable or therapeutic abortion necessary. Now with modern complete substitution therapy the prognosis of pregnancy in Addisonian patients is much better. Approximately 60 such cases are on record, and in 14 of these pregnancy has occurred in the past five years when potent adrenocortical hormones have been available (Hendon and Melick, 1955). Only 7 cases appear to have been reported since cortisone has been used for maintenance. Below is a further case in which pregnancy occurring during treatment of Addison's disease was brought to a successful conclusion.

CASE REPORT

A nulliparous 23-year-old woman who had been married for four years presented with generalized weakness, tiredness, loss of weight, oligomenorrhoea, and skin pigmentation. There was no gastro-intestinal upset and no past history of tuberculosis. She was dehydrated, the skin was inelastic, the tongue dry, and the oral mucous membrane pigmented. Her blood pressure was 70/50 mm. Hg. Serum electrolyte values were compatible with Addison's disease: sodium 130, potassium 5.7, chlorides 69 mEq. per litre. Blood urea 93 mg. per 100 ml.; haemoglobin 85% (Haldane). Urinary 17-ketosteroid excretion 2.9 mg. in 24 hours. Radiographs of chest and abdomen normal. On a standard water load a diuresis of only 50% was recorded in the first four hours.

Initial therapy consisted of deoxycortone acetate (D.C.A.) 10 mg. daily by injection, together with 6 g. of salt a day and an adequate fluid intake. Her general condition improved on this treatment; the blood pressure rose to 110/70 mm. Hg, the electrolytes returned to normal values, and she gained 10 lb. (4.54 kg.) in weight, thus confirming the diagnosis of Addison's disease. A month later it was decided to institute maintenance therapy consisting of an implant of 200 mg. D.C.A. and 3 g. of salt a day. After about six months additional salt was given to overcome increased weakness and lethargy. Her blood pressure and serum electrolytes continued normal. It was decided to

give a further implant of D.C.A., 200 mg., and to continue 3 g. of salt a day.

Two months later she was found to be eight weeks pregnant. She complained of morning sickness, weakness, and lassitude at the end of the third month of pregnancy in spite of continued maintenance therapy. She was admitted to hospital, and improved on bed rest, adequate fluid intake, and dimanhydrinate ("dramamine") to a total of 100 mg. daily. As previous maintenance therapy had been inadequate this was changed to cortisone, 25 mg. daily by mouth, with trimethyl deoxycorticosterone, 60 mg. intramuscularly monthly, and salt, 3 g. a day. The pregnancy progressed satisfactorily on this regimen.

Four weeks before term the patient was again admitted to hospital, and, in addition to the maintenance therapy of 3 g. of salt and 25 mg. of cortisone a day, was now given daily injections of 10-15 mg. of D.C.A., her general condition, blood pressure, and electrolyte levels being the main guide. It was considered inadvisable to give her a longer-acting steroid at this stage. When labour started she was given 100 mg. of cortisone. The second stage was prolonged over eight hours and low forceps under pudendal block analgesia were applied. She was delivered of a living female child of 9 lb. (4.082 kg.). Next day she was given 100 mg. of cortisone, 10 mg. D.C.A., and 3 g. of salt, and the day after a return was made to the previous level of cortisone 25 mg., D.C.A. 10 mg., and salt 3 g. daily.

She had a mild puerperal infection which responded adequately to chemotherapy. Two weeks later she had oedema of the ankles and a blood pressure of 160/110 mm., together with serum electrolyte values of sodium 148, potassium 3, and chlorides 108 mEq. per litre. D.C.A. and salt were therefore stopped but cortisone continued. Within a week the blood pressure was normal and the oedema had subsided. Smaller doses of D.C.A. with a varying salt supplement were started again, and a week later D.C.A. was replaced by trimethyl deoxycorticosterone. The skin pigmentation, which decreased with the initial treatment, increased again near term, but had become less marked by the end of the puerperium. Lactation was successful to start with, but because of cracked nipples it was artificially suppressed after the second week by ethinyloestradiol.

COMMENT

Before 1949 the maternal mortality among patients with Addison's disease who became pregnant was 35-45% (Brent, 1950), but since the introduction of replacement therapy with adrenocortical hormones and cortisone the prognosis is much better. Cases of successful pregnancy in Addison's disease have been recorded by Simpson (1946), Knowlton *et al.* (1949), Brent (1950), Richards (1952), Rolland *et al.* (1953), Lutwyche (1953), Hunt and McConahey (1953), Hendon and Melick (1955), and De Gennes *et al.* (1956). In cases reported so far in which maintenance has been by cortisone in addition to D.C.A. and salt there has been no maternal mortality and the incidence of associated complications has been much lower.

In the case now described Addison's disease was diagnosed and treatment started ten months before pregnancy occurred. The rapidity with which conception followed therapy suggests that the treatment was an important factor in the patient's fertility.

In the first trimester of pregnancy nausea, vomiting, and other gastro-intestinal disorders are more likely to occur, leading to electrolyte disturbance and thus precipitating an adrenal crisis (Brent; Hunt and McConahey; De Gennes *et al.*). In the present case there was only a slight upset at this stage, which was easily corrected. It was thought advisable to start cortisone at this point. In the second half of pregnancy in Addisonian patients there may be an increase in the remaining adrenal function which may ameliorate the adrenal insufficiency state, and substitution therapy may have to be reduced accordingly (Knowlton *et al.*; Hunt and McConahey; De Gennes *et al.*).

Before the introduction of cortisone 25-50% of patients being treated for Addison's disease had hypoglycaemic episodes (Soffer, 1946). In the present case there were no symptoms to suggest this, nor did blood sugar determination, which was carried out monthly throughout the pregnancy, give abnormal results at any time. During labour the added stress of physical exertion, haemorrhage, pain, surgical intervention, and anaesthesia may be too great, and in the early puerperium the physiological diuresis with excretion of sodium, and the removal of adrenal hormones provided by the foetal adrenals, may lead to development of an Addisonian crisis (Knowlton *et al.*; Brent; De Gennes *et al.*). Immediately before delivery, therefore, the patient should be treated as for an impending crisis and every measure taken to avoid undue stress. In this case the dosage of cortisone was increased before delivery and the second stage of labour curtailed by the application of low forceps under regional block analgesia with minimal sedation.

Before the advent of cortisone lactation rarely occurred in these cases, and it has been suggested (Rowntree and Snell, 1931) that this might be due to atrophy of the mammary glands. Breast-feeding used to be forbidden, as it acted as an additional stress on the already deficient metabolism of the mother. There are earlier reports of lactation being artificially suppressed, but with adequate replacement therapy successful lactation is possible without great risk to the mother (Lutwyche, 1953). In the present case lactation was successful, and was artificially suppressed only because of technical difficulties.

Hypertension, cardiac failure, oedema, or potassium deficiency may occur from overdosage with D.C.A., and such symptoms arising in the latter half of pregnancy might lead to confusion with pregnancy toxæmia (Brent). Alterations in blood chemistry and disappearance of symptoms on withholding steroid therapy for a few days will assist the differential diagnosis. Hence it is preferable at this stage to give short-term daily injections of D.C.A. rather than such long-term therapy as pellet implantation or trimethyl deoxycorticosterone injections. Theoretically less D.C.A. should be needed in the second and third trimesters because of the adrenal hormones supplied by the foetal adrenals and the high level of circulating sex hormones, which also have a salt-retaining effect (Soffer, 1946; Samuels *et al.*, 1943; Knowlton *et al.*, 1950). However, in the present case an increased dosage of D.C.A. was necessary during this period.

Signs of steroid overdosage appeared two weeks after the puerperium, but responded well to withdrawal of treatment for a week, after which a maintenance dose of D.C.A. and salt was easily achieved. Three weeks later the patient was discharged from hospital. Mother and child are both doing well.

I am grateful to Dr. I. Gilbert, consultant physician, and Dr. R. Crawford, consultant obstetrician, under whose supervision the patient was treated, for their permission to report this case.

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