same difficulty confronts us in the diagnosis of other clinical conditions with almost identical symptoms. For instance, Cushing's syndrome associated either with pituitary basophilism or with adrenocortical hyperplasia and carcinoma; isosexual precocity in children, due either to hypothalamic tumour (among other causes) or to carcinoma, usually of a solitary adrenal gland.

The ketosteroid test has proved a great boon in differentiating the adrenal lesion in these allied conditions. It is remarkable that these children, essentially female, should tend to exhibit external genitalia of a degree more advanced than their sisters with adrenal virilism, and only their future development will show whether it is only a local manifestation or is part of a more general somatic change. They add further food for thought in this obscure, unnatural, and distressing episcene problem.

These three girls appear to be the juvenile form of a female pseudohermaphrodite brought up as a boy and described by Armstrong in the *British Medical Journal* of May 14, 1955, page 1175.

Summary

Three cases of intersexuality are described—girls with hypospadias of non-adrenal origin.

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USE OF M/2 SODIUM LACTATE IN MULTIPLE ADAMS-STOKES ATTACKS

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The prognosis for a patient suffering from multiple Morgagni-Adams-Stokes attacks is notoriously bad. Such attacks are often seen during a change-over from partial to complete A.V. block, but once a stable complete heart-block is established the outlook tends to improve. When, however, multiple Morgagni-Adams-Stokes attacks arise in a patient known to have had a stable idioventricular rhythm it must indicate a further stage of myocardial degeneration with little chance of recovery.

Many drugs have been used to increase idioventricular rates and to diminish the frequency of Adams-Stokes attacks when these result from ventricular asystole. These include adrenaline, isopropylnoradrenaline, ephedrine, thyroid extract, barium chloride, and atropine (White, 1951). In recent years artificial electrical pacemakers have been introduced with some success (Douglas and Wagner, 1955; Chandler and Rosenbaum, 1955). None of these measures is very dependable. Bellet *et al.* (1955) reported on the beneficial effects of intravenous sodium lactate (molar and half-molar strength) solutions in restoring ventricular beating during repeated Adams-Stokes seizures and increasing the ventricular rate in complete A.V. heart-block.

The case reported here is, we feel, of interest because of the apparently clear-cut relation between the administration of sodium lactate and the cessation of Adams-Stokes attacks.

Case Report

A 76-year-old man was admitted to hospital on July 30, 1955, having for the previous two days suffered from dizziness on standing. Examination revealed a regular heart rate of 17 a minute and a blood pressure of 150/60. There were no signs of heart failure. The heart sounds were not abnormal, nor were any murmurs present. Examination of

FIG. 1.—Effect of sodium lactate on frequency of Adams-Stokes attacks.

FIG. 2.—Effect of M/2 sodium lactate.

the respiratory system suggested moderate emphysema. No abnormality was found on abdominal or neurological examination. An electrocardiogram confirmed the presence of a complete A.V. dissociation and a slow idioventricular rhythm.

Shortly after admission the heart rate fell to 12 a minute. Isopropylnoradrenaline, 10 mg., was administered sublingually and oral ephedrine, $\frac{1}{2}$ gr. (32 mg.), four times daily was begun. These measures increased the heart rate to about 25 a minute, but the ephedrine had to be discontinued, as it appeared to be causing the patient to vomit. On the fourth day he began to have typical Adams-Stokes seizures. An electrocardiogram obtained in an attack confirmed that these were due to ventricular asystole. In each seizure the cardiac arrest lasted approximately 20 seconds. There was an interval of 15 seconds between the cessation of the heart beat and the beginning of the convulsive movements.

By next day there had been 29 attacks, and it was decided to try the effect of sodium lactate intravenously. Only M/6 sodium lactate was available, and 1 pint (570 ml.) of this was given over two hours, followed by a second pint five hours later. The cardiac rate was not affected by this treatment; neither was there any very significant reduction in Adams-Stokes seizures.

On the following day the Adams-Stokes attacks were becoming very much more frequent, and 200 ml. of M/2 sodium lactate was given intravenously over a period of 20 minutes. This resulted in a dramatic cessation of attacks.

although again the heart rate was unaffected. The remission lasted 10 hours, after which attacks returned and continued for a further nine hours, when a second infusion of 200 ml. of sodium lactate was given. Again this coincided with a complete cessation of attacks lasting five hours, and in fact only three more seizures occurred in the next 24 hours.

Fig. 1 shows the frequency of the seizures, the cardiac rate, and the relation of both to sodium lactate infusions over the entire period of the attacks. Fig. 2 shows the effect of M/2 sodium lactate drawn to a larger scale.

The total plasma carbon dioxide concentrations immediately before and after the first infusion of M/2 sodium lactate were 26 mEq/l. and 29 mEq/l. respectively. During the second infusion of M/2 sodium lactate the carbon dioxide concentration rose from 22 to 28 mEq/l. Sodium-, potassium-, and chloride-ion concentrations were not significantly altered.

In the ensuing two weeks there were no further seizures. The heart rate gradually rose from about 20 a minute to 35-40 a minute under the influence of thyroid extract and isopropylnoradrenaline. Once again ephedrine was not tolerated.

In all, the patient had suffered 130 Adams-Stokes attacks.

Comment

It is felt that there was a clear enough response to M/2 sodium lactate on two occasions to warrant its further trial in similar cases. In this case we did not confirm the observation of Bellet *et al.* (1955) of an increase in the idioventricular rate. The mechanism whereby sodium lactate may exert its beneficial effect is not understood. Bellet *et al.* suggest certain theoretical possibilities. These are, first, that the lactate serves as a fuel that can rapidly be utilized by the myocardium; secondly, that the alkalosis induced by the lactate results in an increase of myocardial excitability and conductivity; thirdly, that the increase in sodium-ion concentration might do the same. Of the possibilities, they favoured the alkalosis as being the single most important factor.

We wish to thank Professor A. Kekwick for his permission to publish this case.

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Medical Memoranda

Haemolytic Disease of the Newborn

The following case of haemolytic disease of the newborn was successfully managed by early induction and exchange transfusion after three previous stillbirths due to hydrops foetalis.

CASE REPORT

A farmer's wife aged 33 attended the antenatal clinic at the Radcliffe Infirmary, Oxford, in August, 1950. She was pregnant for the fifth time and advanced to the twenty-eighth week. Her first pregnancy, in 1940, was normal, and a healthy child was born at term. She became pregnant again in 1941, 1943, and 1944, but each time was delivered of a macerated foetus. Her doctor, who had attended her throughout, could not account for these repeated disappointments. He could find no obvious maternal disease, nor was there any discoverable abnormality in the pregnancies. But the patient noticed on each occasion that foetal movements became less vigorous a week or two before the baby died *in utero*. When examined on August 3, 1950, the patient was in good health, her blood pressure was 130/80, and

her urine was normal. The uterus was compatible in size with a pregnancy of 28 weeks' duration. Vigorous foetal movements were felt. Serological examination showed her to belong to blood Group B, Rh negative. Incomplete anti-D antibodies were present in a titre of 1:2. The Wassermann and Kahn reactions were negative. The patient's husband belonged to blood Group B, Rh positive (R₁R₂).

It was thought that her previous stillbirths were due to rhesus incompatibility. It seemed that the prospect of a live birth in this fifth pregnancy was unlikely. In view of all the circumstances it was decided to induce labour at about the thirty-fourth week.

On September 6 labour was induced surgically. At this time antibodies were present in a titre of 1:4, but the foetus was lively and presented by the vertex. It was found that the cervix was long and tight; it was dilated sufficiently to introduce a Queen Charlotte's hydrostatic bag, which was then filled with 250 ml. of sterile water. A half-pound (225 g.) weight was suspended from this, so that pressure was made continuously on the cervix. After 12 hours regular contractions began, and recurred each five minutes; after 18 hours the bag came out. The cervix was found to be dilated three fingerbreadths; therefore the hind waters were ruptured in the hope that labour would be hastened. Unfortunately, subsequent progress was slow, but after a further 30 hours the cervix was sufficiently dilated to allow forceps delivery. On September 9 the patient was anaesthetized, a left occipito-posterior position was corrected manually, forceps were applied, and the foetus was extracted. The third stage of labour was normal.

The baby was slow to breathe, but responded after the air passages had been aspirated and oxygen had been insufflated into the trachea. Ten minutes later its immediate condition was satisfactory. The baby weighed 4 lb. 6½ oz. (2 kg.). There was no evidence of hydrops foetalis or of severe anaemia. Examination of cord blood showed the infant to belong to blood Group B, Rh positive (R₁R). The Coombs test was positive in 10 seconds. The serum bilirubin was 3.3 mg. per 100 ml. Weak incomplete anti-D antibodies were present in cord blood in a titre of 1:1. The baby's haemoglobin was 60% (9.5 g.%). Exchange transfusion was effected via the umbilical vein: 285 ml. of compatible blood (Group O, Rh negative) was infused, and 225 ml. was removed. The exchange was effected in 10-min. amounts. The operation proceeded easily and was completed in one and a half hours. At intervals throughout the operation the infant's venous pressure was checked by the simple expedient of holding the open "polythene" tube vertically, but there was no significant change in the level to which blood rose in it, and the baby's condition remained good. On the following morning her haemoglobin was 95% (15.3 g.%). She was not jaundiced, nor did jaundice develop in subsequent days. She left hospital on the thirty-sixth day weighing 6 lb. 4 oz. (2.8 kg.), and feeding well from the breast. Her haemoglobin did not fall below 80% (12.6 g.%).

Follow-up.—The infant continued to thrive and to develop normally. She was weaned at 5 months, and walked at 15 months. When visited in April, 1955, she was found to be lively and intelligent—in fact, a normal girl aged 4 years 6 months.

COMMENT

It is generally agreed that prematurity adds to the risk of mortality in babies affected by haemolytic disease of the newborn (Mollison and Walker, 1952). None the less, when there is a history of stillbirth, and when the father is homozygous rhesus-positive, it is clearly important to deliver the baby rather than let it die *in utero* (Davies *et al.*, 1953). It is generally inadvisable to allow pregnancy to continue beyond term when antibodies are present. Furthermore, it is an advantage to begin exchange transfusion as soon as