SWEAT SODIUM LEVELS

SIR,—May I clarify a few points that arise from the letter by Dr. Karlish and his associates (Feb. 24)?

First, my bronchitic patients were, as they suggest, unselected as regards family history.

Secondly, it should have been clear from the calculation of standard errors of difference between means of normal subjects and siblings of fibrocystic patients that there were differences in two of the four age-groups. The practical implication of this was noted (lines 18 et seq., p. 185). Similar comparison of normal adults and parents of fibrocystic children in the three groups which were comparable (i.e., 15–24, 25–34, 35–44) showed a significant difference in only one. To compare mean values of two series (for example normal adults and chronic bronchitics) each composed of different proportions in their age subgroups is an incorrect procedure.

Thirdly, I realise that my statement that it " . . . seems unlikely that the heterozygote state is manifest in alteration of the sweat sodium level . . ." was unfortunately worded. The study showed that heterozygotes as a group have a slightly raised mean sweat sodium level over the ages studied. My aim was to emphasise that sweat sodium levels (obtained by the iontophoresis of pilocarpine) are of little or no value in detecting individual heterozygotes.

Hospital for Sick Children,
London, W.C.1. T. MCKENDRICK.

SIR,—The recent contributions on this subject illustrate how little research has been done on the physiology of the sweat glands.

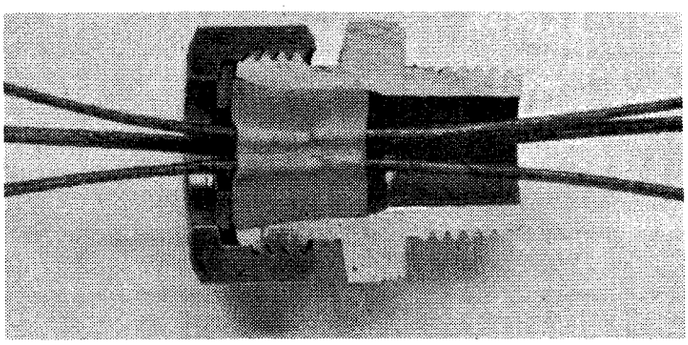
The excretion of electrolytes and sodium have a direct relationship to cortisone and other substances secreted by the adrenal cortex. It has been shown [1] that the amount of electrolytes and sodium excreted have a direct bearing on whether dry heat or wet heat is used to activate the sweat glands. The amount of sodium and electrolytes excreted when pilocarpine is used as an activator must depend upon the condition of the sweat glands, which are much less active in old age and in many systemic diseases.

Tunbridge Wells. E. F. ST. J. LYBURN.

THERMOCOUPLES FOR AUTOCLAVES

SIR,—The increasing need to measure temperature within packs inside autoclaves involves passing thermocouples through the chamber wall.

A way has been found of introducing six thermocouples requiring only an 0·5 in. B.S.P. entry. This consists of a 0·5 in. compression fitting as used for joining copper pipe to a B.S.P.



socket. A rubber bung drilled with holes in which the thermocouples are a tight sliding fit is inserted, instead of the copper pipe and cone, with a suitable washer as a retainer. For more permanent use the thermocouples are embedded in an epoxy resin in a short length of copper tubing and the cone is used in the normal way.

We find thermocouple wires wrapped and braided with glass fibre to form an oval section of about 3·0 × 1·5 mm. most satisfactory. The wires are impregnated with an epoxy resin, using toluene as a diluent. For insertion they are lubricated with silicone grease. The junctions are soldered into short lengths of hypodermic needle tubing for protection and ease of insertion through the rubber bung, and Roman numerals may be filed on these for identification.

These thermocouples withstand more than 200 cycles without serious deterioration if carefully handled to prevent mechanical damage.

Similar assemblies have been in use for 5 years and have given complete satisfaction. There is no leakage under either vacuum or steam pressure.

The accompanying photograph shows a vertical section through the unit.

Surgical Division,
Allen & Hanburys Ltd.,
London, E.2. G. R. WILKINSON
F. G. PEACOCK.

1. Lyburn, E. F. St. J. *J. Physiol.* 1956, **134**, 207.

XY/XO MOSAICISM

SIR,—Hirschhorn et al.[1 2] described a case of XY/XO mosaicism in a 3-month-old infant, who must be regarded as a true hermaphrodite on account of the histological structure of the gonads.

Blank et al.[3] reported a second example of XY/XO mosaicism in a 55-year-old woman, who had a short stature and a short neck. Her chest was broad and her breasts were poorly developed. Axillary and pubic hair was present and of female distribution. The external genitalia were of female type. The clitoris was fairly long. A small cervix could be felt rectally. Sex-chromatin bodies could not be detected in buccal mucosal smears. Thirty years before, laparotomy had been performed, and a very small uterus and a very small ovary on the left side were found; no gonad could be identified on the right side. Sections of the left gonad were not made.

Miller et al.[4] described two more cases of XY/XO mosaicism in two female patients with enlarged clitoris, perineal urethra, vagina, uterus, bilateral fallopian tubes, absent gonad on one side, and a seminoma developing in the opposite intra-abdominal gonad, which contained abundant seminiferous tubules as well as ovarian stroma. One patient was mentally deficient, and her appearance suggested a variant of Turner's syndrome, with slight breast development and no masculinisation except enlargement of the clitoris. The other patient, highly intelligent, was strongly masculinised and had large clumps of Leydig cells in the gonad.

Jacobs et al.[5] made chromosome studies on 32 patients with primary amenorrhœa. Two patients were found to have an XY/XO chromosomal constitution. In one of these cases the cells from the skin culture showed only an XO stem-line, while in the other only 3 out of 50 cells could be regarded as having an XY complement. In both cases the majority of cells from the blood-cultures had an XY complement, although some XO cells were present. Clinically both patients had normal but underdeveloped genitalia, and in both cases laparotomy revealed " streak " gonads. Histological examination of the gonads showed some ovarian stroma but no follicles in one case, and mainly connective tissue in the other case, although here a cluster of theca-like cells was present in the right gonad. Neither case had evidence of testicular tissue. We believe we can add a seventh case to this series of XY/XO mosaics.

In a patient described by Plate [6] in 1957 as an example of male pseudohermaphroditism, we found the following distribution of chromosome counts in leucocyte cultures from peripheral blood:

	<44	*44*	*45*	*46*	*47*	*>47*	*Total*
Number of cells	0	0	52	48	0	0	100

In the cells with 46 chromosomes, 5 elements of the size of group 21–22+Y (Denver) were invariably found,

1. Hirschhorn, K., Decker, W. H., Cooper, H. L. *Lancet*, 1960, ii, 319.
2. Hirschhorn, K., Decker, W. H., Cooper, H. L. *New Engl. J. Med.* 1960, **263**, 1044.
3. Blank, C. E., Bishop, A., Caley, J. P. *Lancet*, 1960, ii, 1450.
4. Miller, O. J., Breg, R., Jailer, J. W. Human Chromosome Newsletter 1960, **1**, 6 (quoted with the author's permission).
5. Jacobs, P. A., Harnden, D. G., Buckton, K. E., Court Brown, W. M., King, M. J., McBride, J. A., MacGregor, T. N., Maclean, N. *Lancet*, 1961, i, 1183.
6. Plate, W. P. *Med. Tijdschr. Geneesk.* 1957, **101**, 1741.

whereas only 4 such elements were present in the cells with 45 chromosomes. The interpretation of these findings as another example of XY/XO mosaicism seems to us the more probable because of the clinical features.

The patient, born in 1940, never menstruated spontaneously. She is of a female phenotype but of a somewhat boyish build. Her length is 143 cm. During puberty hair began to grow on her face, and she had to shave once a week. The axillary hair was normal. Pubic hair was abundantly present, with a slightly male implantation. The breasts were totally undeveloped. Except for enlargement of the clitoris (4 cm.), there were no abnormalities of the external genitalia. Rectally no uterus could be felt.

The urinary hormone excretion was: gonadotrophins more than 150 units per 24 hours; 17-ketosteroids 6·2 mg. per 24 hours; œstrogens less than 25 units per 24 hours. There was a good response of the adrenals after intravenous administration of corticotrophin. Laparotomy revealed a uterus of 5 cm. with normal fallopian tubes. On the left side, in the position of the ovary, a gonad the size of a grape was found; on the right side, in the same position, a " streak " gonad.

Histological examination of the left gonad revealed seminiferous tubules without spermatogenesis, and numerous Leydig cells. In the right gonad, among connective tissue with nerve elements, a few tubules and clumps of Leydig cells were found. Sex-chromatin bodies were not present.

When we leave apart Hirschhorn's patient, who is a true hermaphrodite, and the patient described by Blank, in whom histological examination of the gonads was not performed, our case is very similar to those described by Miller and by Jacobs. The cases of Jacobs et al., however, are different from the others in their gonadal histology, since these workers declare that there is no evidence of testicular tissue in the sections.

The two cases described by Miller, and ours, may represent a syndrome characterised by a female phenotype with signs of masculinisation, primary amenorrhœa, rudimentary gonads with testicular features, and XY/XO chromosome mosaicism. The symptoms suggest an intermediate form between gonadal dysgenesis and testicular feminisation. It seems to us that this syndrome can be distinguished from Turner's syndrome with XO chromosomal constitution and from the syndrome of testicular feminisation with XY chromosomal constitution.

A detailed description of the findings will be published elsewhere.

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C. H. WILLEMSE.
JANNY M. VAN BRINK

Institute of Genetics, University of Utrecht.
P. L. LOS.

PHENINDIONE JAUNDICE

SIR,—The paper by Dr. Perkins (Jan. 20), which includes a review of the literature, suggests that jaundice due to phenindione sensitivity usually develops in the fifth week of therapy. An interesting variation was seen recently in a patient in Edgware General Hospital under the care of Dr. G. H. Jennings.

He was a man of 52 who had been admitted in October, 1961, with a deep-vein thrombosis for which he was treated with phenindione for twenty days and made a good recovery. No allergic reactions were noted. After his discharge he remained well for twelve weeks and was then readmitted in January, 1962, with a recurrence of the thrombosis. Phenindione was again given, and on the ninth day an urticarial reaction developed and on the fifteenth day jaundice. At this time the serum-bilirubin was 5·2 mg. per 100 ml., the thymol turbidity test was positive, the alkaline-phosphatase level was 31 King-Armstrong units, and serum-glutamic-pyruvic-transaminase 200 units per ml. The jaundice began to fade after a few days without specific treatment, and liver-function tests are now returning to normal.

Presumably this patient was sensitised to phenindione on his first admission (although there was no evidence of this) and had an accelerated sensitivity reaction on his second admission. The patient would not agree to a liver biopsy, so the histological reaction could not be determined.

Edgware General Hospital, Edgware, Middlesex.
B. E. HEINE.

NAMES *AND* ADDRESS

SIR,—May I plead for a full postal address at the end of each article—as our American friends do? The recent G.P.O. " work to rule " reminds us that it is unfair to expect delivery of reprint requests to addresses such as " Dr. X, General Practitioner, Birmingham."

London, W.5.
A. HOLLMAN.

*** Though we do not at present want to change the way in which authors are described in the headings of articles, we shall try to ensure that the descriptions always include adequate addresses.—ED. L.

EPIDEMIOLOGY OF SKIN DISEASES IN AFRICA

Dr. JAMES MARSHALL, head of the department of dermatology in the University of Stellenbosch, writes:

" A pilot survey on this subject has yielded such interesting material that I am preparing, with the assistance of the South African Council for Scientific and Industrial Research, to make a large-scale investigation of the distribution and incidence of skin diseases on the continent of Africa. The ultimate aim is to establish a central office for information and a reference library of literature and photographs.

" I wish to approach not only those in scientific and academic institutions, but also anyone who practises or has practised in Africa who could supply information, even if it were only in a limited area of the subject. Questionnaires will be sent on request to anyone willing to help. Please reply to: Simon's View, Sorrento Road, St. James, C.P., Republic of South Africa."

Parliament

Commonwealth Immigrants Bill

ON Feb. 22, at the report stage of this Bill, Mr. KENNETH ROBINSON moved an amendment to ensure that no immigrant could be refused admission on mental or medical grounds on the judgment of an immigration officer alone. Mr. DAVID RENTON, Minister of State to the Home Office, said that it would be most unusual for an immigration officer to refuse entry on medical grounds without referring the matter to a medical officer. But circumstances could arise when the power would be needed. For instance, an immigration officer, after consultation with the ship's doctor, might warn a passenger who was taken seriously ill during the voyage that he would not be allowed to land. The passenger would then be saved the trouble of coming ashore for a medical examination. Coming to the question of mental disorder, Mr. Renton said that in no doubtful case would the immigration officer take it on himself to refuse leave to land, but there were occasions, admittedly rare, when mental derangement was obvious. Mr. ROBINSON remained unconvinced that the immigration officer should be given blanket powers of this kind.

Dr. DICKSON MABON wanted to see the immigration officer's powers restricted to gross mental disorder or to " serious transmissible disease ", because patients with acute physical illness would presumably have to land because they would need medical treatment, and many patients with a chronic disease, such as diabetes, could prove useful citizens. Mr. RENTON gave examples of physical conditions which would not be admitted: crippling orthopædic disease or serious eye