relapse during the gluten challenge was followed by remission after the second withdrawal of gluten from the diet.

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INHERITANCE OF PSEUDOHYPOALDOSTERONISM

SIR,-The exact mode of inheritance in pseudohypoaldosteronism needs clarifying since few pedigrees have been reported. Dr Limal and colleagues (Jan. 7, p. 51) proposed autosomal dominance with variable expression. We have seen a female infant with pseudohypoaldosteronism family studies confirm Limal's suggestion.

The girl was born after an uncomplicated full-term pregnancy, birth-weight 2600 g. She was admitted at 3 months of age because of failure to thrive and severe dehydration. Electrolyte determinations revealed persistent hyperkalæmia. The plasma-aldosterone at 4.5 months was 600 ng/dl. The infant was discharged at this time weighing 4120 g. The results of plasma aldosterone, renin, and urine aldosterone at the age of 6 months are shown in the table. Her mother had been in hospital several times during infancy because of vomiting and failure to thrive and had received parenteral fluid. Since the age of 1.5 years she had gradually recovered, without any specific treatment. We examined five more members of the family who were symptom-free.

The propositus and her mother, maternal grandmother, and two brothers had high aldosterone and renin values. Five siblings of the mother who are also clinically normal were not available for investigation.

Blood-samples were drawn in the morning after the patients had got up. Aldosterone was isolated from the methylene chloride extract of plasma and urine by immunological purification before radioimmunoassay.² The normal ranges for plasma and urine aldosterone are 2-14 ng/dl and 2-14 μ g/24 h, respectively. The plasma-renin-activity (P.R.A.) was determined by angiotensin 1 radioimmunoassay kit (New England Nuclear). The normal base values range from 0.6 to 3.0 ng/ml/h.

The affected family members, except for one brother, have short stature. Moreover, the propositus was just below the 3rd percentile at the age of 6 months and remained so until we began a high-salt diet at the age of 8.5 months. The treatment resulted in a catch-up growth concomitant with the expected lowering of aldosterone and renin values.

Catch-up growth in salt-losing syndromes after high-salt diet is well known.²⁻⁴ However, the influence of this treatment

Donnel, G. N., Litman, N., Roldau, M. Am. J. Dis. Child. 1959, 97, 813.
Proesmans, W., Geussens, H., Corbeel, L., Eeckels, R. *ibid.* 1973, 126, 510.
Rosler, A., Rabinowitz, D., Theodor, R., Ramirez, L. C., Ulick, S. J. clin

Endocr. Metab. 1977, 44, 279.

on the linear growth of symptom-free persons with pseudohypoaldosteronism has not been described. As this syndrome is probably more common than previously thought, we suggest that plasma aldosterone and renin be measured in the evaluation of short stature, especially if there is a suggestive family history. Early salt treatment in proven symptom-free patients seems logical.

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PREVENTION OF CONGENITAL ADRENAL HYPERPLASIA

SIR,---According to Dupont et al.¹ and preliminary experience in our clinic, the 21-hydroxylase locus, defective in congenital adrenal hyperplasia (C.A.H.) is closely linked to the HLA B locus. This means that it might well be possible to use specific antibodies to eliminate the 50% of the paternal sperms^{2,3} which carry the marker HLA B antigen and then carry out artificial insemination with the remaining spermatozoa. Using this method the chances for the offspring of heterozygote (for C.A.H. and father for HLA B) parents, should be improved to almost 50% carrier-free and 50% heterozygotes. The same could be applied to HLA B27 fathers with ankylosing spondylitis.

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FINE-NEEDLE PERCUTANEOUS TRANSHEPATIC **CHOLANGIOGRAPHY**

SIR,—Our experience confirms your support for fine-needle percutaneous transhepatic cholangiography in the investigation of jaundice (May 27, p. 1139). In ninety-six consecutive cholangiograms done in this way there has been only one failure to outline the ducts, and there have been no serious complications. The diagnostic accuracy is excellent, as judged by definition of the level and cause of obstruction. Failure to enter the bileducts does not completely exclude obstruction as the intrahepatic ducts may be of normal fine calibre in some cases of gallstone obstruction.

This diagnostic technique is safe and sure, largely due to the use of the lateral approach, slim needle, and prophylactic antibiotics.

Drainage of the ducts is a therapeutic measure which requires the insertion of a much larger calibre flexible catheter. It is attended by greater risks such as bile leakage, of a degree that caused the older methods of transhepatic cholangiography

1. Dupont, B., Oberfield, S. E., Smithwick, E. M., Lee, T. D., Levine, L. S. Lancet, 1977, ii, 1309.

Fellous, M., Dausset, J. J. Nature, 1970, 225, 191.

3. Halim, A., Abbasi, K., Festenstein, H. Tissue Antigens, 1974, 4, 1.

RESULTS	OF	FAMILY	STUDY
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Subject (age)	Plasma-aldosterone (ng/dl)	Plasma-renin (ng/ml/h)	Urine-aldosterone (µg/24 h)	Height (cm)	High percentile (Tanner 1966)
Propositus (6 mo)	900	74.0	100	61	< 3
Propositus (13 mo, high-salt diet)	186	18.6	39.2	71.5	10
Brother I (70 mo)	168	10.3	20.6	107	10
Brother II (50 mo)	47.1	4.7	2.5	104	50
Mother (24 yr)	29.8	10.8	29.6	150	< 3
Father (31 yr)	10-4	3.5	9.5	170	25
Maternal grandmother (51 yr)	9.0			156	15
Maternal grandfather (63 yr)	67.7			160	< 3
	1	1	1	1	1

^{1.} Gomez-Sanchez, C., Kem, D. C., Kaplan, N. M. J. clin. Endocr. Metab. 1975, 36, 795.