Sex Ratio of White Newborn Babies, by ABO Blood Group, in the Aggregate of the 15 Relevant Series of 1924-72^{1,4}

Mothers' Group	Babies' Group	Number of Babies		
		Male	Female	M./F.
AB	A or B AB	824 141	647 135	1.27 1.04
A	O or AB or B A	3496 5633	3083 5560	1.13 1.01
O	A or B O	3291 7520	3194 6839	1.03 1.10
B	O or AB or A B	1497 1243	1467 1075	1.02 1.16
		23645	22000	1.07
AB Mothers A + O + B Mothers		965 22680	782 21218	1.23 1.07
Mothers and Babies	Different	9108	8391	1.09
	The Same	14537	13609	1.07

ence which is reciprocal to it—that is, by a lower sex ratio for non-B babies of B mothers and non-O babies of O mothers than for non-A babies of A mothers. That is to say, the 1924-72 aggregate presents a clear-cut difference in sex ratio between the babies of two contrasted types of mother—namely, mothers whose babies are of the same ABO blood group as themselves, and those whose babies are of another group from themselves—the difference being significant for B mothers ($P < 0.05$), O mothers ($P < 0.05$), and A mothers ($P < 0.0005$). A difference parallel to the one observed in A mothers is seen in AB mothers—it is evident in the Table, and is present even in Hirsfeld and Zborowski's series—but here the difference is not significant ($P > 0.10$), whatever may eventuate when the aggregate is added to. A detailed account and discussion are being prepared in which the idea will be considered that the phenomena are the result of unknown effects of steroid hormones.

I am grateful to Professor D. F. Kerridge and Dr. H. B. M. Lewis for their stimulating comments.

—I am, etc.,

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¹ Hirsfeld, L., and Zborowski, H., *Klinische Wochenschrift*, 1925, 4, 1152.

² Sanghi, L. D., *Nature*, 1951, 168, 1077.

³ Cited by Allan, T. M., *British Medical Journal*, 1959, 1, 553.

⁴ Krauss, A., and Zimmermann, B., *Zentralblatt für Gynäkologie*, 1970, 92, 12.

Hypotension after Verapamil

SIR,—In view of Dr. M. E. Benaim's report of asystole after verapamil (15 April, p. 169) we should like to report another instance of an adverse reaction associated with the use of this drug.

A 46-year-old man in congestive cardiac failure resulting from fast atrial fibrillation probably due to viral myocarditis was given 10 mg verapamil intravenously over 30 seconds. Although his heart rate fell immediately to around 100 per minute from over 200 per minute, his systolic blood pressure diminished from 80 mm Hg to 50 mm Hg. This was accompanied by sweating and restlessness. Improvement took place over the next 30 minutes, his heart rate remaining at 100 per minute although still in atrial fibrillation. He was not digitalized at the time of receiving verapamil.

We should like to advise caution in the use of this drug in hypotensive patients.—We are, etc.,

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Post-gastrectomy Acidity

SIR,—In the surgery of duodenal ulcer, there seems to be a good deal of confusion over what preoperative or postoperative characteristics of gastric secretion are grounds for expecting a good clinical result. In particular, it is often understandably assumed that a low maximal acid output after operation is a good guide. But in our experience, using the augmented histamine test, this is of no prognostic value.

In a few (30) patients in Oxford undergoing both the augmented histamine test, with and without vagal block, and an insulin test, before and after operation, the most valuable prognostic guide was the post-operative basal acid output.

It is perhaps worth noting also that the postoperative repetition of the augmented histamine test with and without vagal block (by hexamethonium) seemed as good as the insulin test in predicting the clinical result—the absence of reduction in maximal acid output by vagal block, presumably indicating adequate vagotomy. Both were, however, prognostically inferior to the basal acid output.

In all tests, any collection period from maximum half-hour to first two hours, and titration to either pH 3.5 or pH 7, seemed equally good. The main pitfalls, as usual, were in failing to recover all secretions, and we found a sump tube far superior to other varieties.—We are, etc.,

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Need for Continued Oral Therapy in Diabetes

SIR,—Dr. J. W. Todd raises the problem of the obese diabetic who does not sustain a restricted diet (29 April, p. 295). Since I do not subscribe to the doctrine of original sin and since I believe prolonged hyperglycaemia to be harmful, I prefer not to allow such patients to stew in their own syrup. I agree that either insulin or the sulphonylureas may lead to further obesity. Phenformin or metformin should be prescribed. The biguanides have the double virtue of reducing the blood sugar and of reducing the weight.—I am, etc.,

ARNOLD BLOOM

London W.1

Skin Sensitivity in Au-antigen Carriers

SIR,—Australia antigen (Au) was detected in 32 out of 413 children age 1-16 years investigated by the Ouchterlony double-diffusion technique (Table).¹ Only one Au-positive patient had symptoms of hepatitis; 17 had been transfused previously. The study

was repeated two months later, and Au was present in samples from 25 children.

TABLE—Australia Antigen in Children

Disease	No. of Patients	Au-antigen positive
Acute lymphoblastic leukaemia	39	8 (8*)
Down's syndrome	24	3
Hodgkin's disease	10	6 (4*)
Lymphosarcoma	7	3 (3*)
Others (pneumonia, nephritis, asthma, rheumatic fever, diabetes mellitus)	333	12 (2*)
Total	413	32

* Transfused

In these 25 Au carriers the skin sensitivity to a chemical agent—dinitrochlorobenzene (D.N.C.B.)—was studied. The D.N.C.B.-sensitization procedure consisted of the application of 0.1 ml of a 1% acetone solution to a circular area 2 cm in diameter on the ventral surface of the forearm. After evaporation of the solvent the area was occluded by Band-aid for six days. Twenty-one days later the patients were retested with the same solution of D.N.C.B. Out of 25 tested only two Au-carriers developed delayed hypersensitivity to D.N.C.B. Control group consisting of 13 Au-negative children was similarly tested, and the D.N.C.B.-test was positive in all of them.

It seems from this that the delayed hypersensitivity type of reaction to a chemical contact sensitizing agent may be impaired in Au-carriers.—We are, etc.,

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¹ Aisenberg A. C., *Journal of Clinical Investigation*, 1962, 41, 1964.

Liver Injury

SIR,—Mr. L. H. Blumgart and Dr. T. Vajrabukka (15 January, p. 158) described liver injury in 20 cases, 17 of which were caused by traffic accidents. We present the case of an 11-year-old boy who was kicked in the abdomen by a donkey and brought to our hospital in a state of shock 14 hours after the injury. A plain upright film of the chest showed a bubble of gas under the right diaphragm.

At laparotomy, there was a foul odour as soon as the abdomen was opened. The liver was crushed and lacerated on the supero-lateral aspect (an area of about 10 × 5 × 5 cm). The peritoneal cavity was full of foul smelling dark blood. The gall bladder was distended but no bowel perforation was found. The liver was repaired by primary closure.

The material received for pathological examination consisted of about 5 g of necrotic brownish tissue fixed in formalin. Microscopic examination of haematoxylin and eosin and reticulum-stained sections showed necrotic liver tissue with many cystic spaces. Gram stain (Fig.) demonstrated numerous Gram-positive bacilli diffusely scattered throughout the section. The approximate