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	824	647	1·27
	AB	141	135	1·04
A	O or AB or B	3496	3083	1·13
	A	5633	5560	1·01
O	A or B	3291	3194	1·03
	O	7520	6839	1·10
B	O or AB or A	1497	1467	1·02
	B	1243	1075	1·16
		23645	22000	1·07
AB Mothers		965	782	1·23
A + O + B Mothers		22680	21218	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 Hirszfeld 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 Hirszfeld, L., and Zborowski, H., *Klinische Wochenschrift*, 1925, **4**, 1152.
2 Sanghvi, 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 postoperative 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

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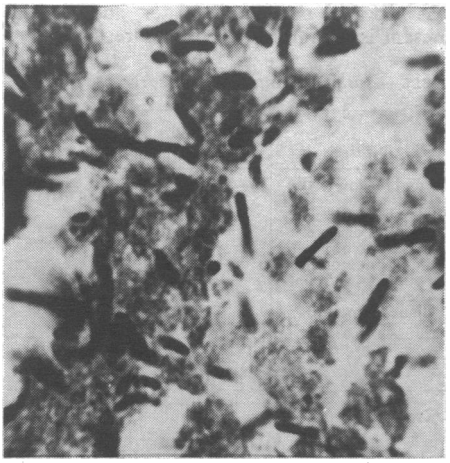
1 Aisenberg A. C., *Journal of Clinical Investigation*, 1962, **41**, 1964.

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 superolateral 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 haemotoxylin 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



size of the bacilli was 8 μ × 1 μ. No capsules were seen and many bacilli showed subterminal empty spaces probably representing spores which had not distended the bacterial outline. Typical rounded ends were present in all bacilli and many of them were slightly curved, others were at right angles to each other giving the impression of branching hyphal forms. Cultures from the drainage tube on the fourth postoperative day for anaerobic bacilli failed to produce any growth.

Immediately after receipt of the pathology report massive penicillin therapy was started with very favourable response. On the 16th postoperative day the patient was discharged after a minor lung infection had cleared up with broad spectrum antibiotic therapy. Liver function was normal except for slightly reduced serum protein (5·4 g/100 ml).

We were intrigued by the presence of the Gram-positive bacilli, since there was only mild bruising of the anterior abdominal wall and no bowel injury was found. Ascent of bacilli in the post-traumatic state up the common bile duct is the probable source of these organisms. Bacteria, especially anaerobes, have been found after death in human and animal livers at necropsy.[1] Cultures of liver biopsies are generally negative, but this is believed to be due to the bactericidal action of fatty acids. Bacteria entering the biliary tree or the portal circulation from the intestine have also been reported.[1] Experimental hepatic damage also leads to bacteria in the bile or lymph.

The exact classification of these bacteria was not possible because specimens for anaerobic cultures were not obtained during the surgical procedure. The morphology and gas formation would suggest *Cl. welchii* or organisms of the gas-forming anaerobic group.—We are, etc.,

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1 Popper, H., and Schaffner, F., *Liver Structure and Function*, New York, McGraw Hill, 1957.

Neonatal Conjunctivitis

SIR,—Dr. D. J. Hansman (18 March, p. 748) described a case of neonatal conjunctivitis due to *Neisseria meningitidis* and rightly stressed the need for thorough bacteriological identification if such cases are not to be confused with gonococcal infection. The following report of a case of purulent conjunctivitis, apparently caused by *N. catarrhalis*, may serve to reinforce Dr. Hansman's advice.

A healthy 11-day-old baby developed a purulent discharge from her left eye. Staining of the pus revealed large numbers of polymorphonuclear cells, some of which were packed with Gram-negative diplococci. Cultures yielded a heavy pure growth of *N. catarrhalis*, of which the identity was confirmed by the National Collection of Type Cultures, Colindale, London. The baby was treated with chloramphenicol eye drops and made a rapid recovery.

The possibility cannot be excluded that the conjunctivitis had some other underlying aetiology and that *N. catarrhalis* was a harmless superinfection. However, the organism was grown in pure culture from swabs taken on two separate days, and its location within pus cells might suggest that its presence was not welcome. Although *N. catarrhalis* is normally considered to be a harmless commensal it occasionally assumes a pathogenic role, particularly in the young. For example, Cocchi and Ulivelli[1] described a case of meningitis caused by *N. catarrhalis* and cited 17 similar cases from the literature, and Graber *et al.*[2] attributed three cases of urethritis to infection by this organism.

It seems likely, therefore, that *N. catarrhalis* may at times be responsible for neonatal conjunctivitis and this problem will only be elucidated by careful investigation.—I am, etc.,

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1 Cocchi, P., and Ulivelli, A., *Acta Paediatrica Scandinavica*, 1968, 57, 451.
2 Graber, C. D., Scott, R. C., Dunkelberg, W. E., and Dirks, K. R., *American Journal of Clinical Pathology*, 1963, 39, 360.

Asymptomatic Bacteriuria—a Serious Disease?

SIR,—Asymptomatic bacteriuria is a common finding in children[1] as well as adults.[2] Its clinical significance, however, is presently unknown, but might be elucidated by a few observations from our studies of urinary tract infections.

The frequency of *E. coli* in the R phase varies between different categories of patients with urinary tract infection. The Table shows that R phase bacteria are uncommon in primary infections, but increase in frequency with recurrences to be common in chronic pyelonephritis. In 48 children with asymptomatic bacteriuria we found R phase bacteria in 22%. In addition 28% of the strains showed a broadened agglutination pattern suggesting partial S-R degradation.[3] This suggests that asymptomatic bacteriuria in childhood might be a finding indicating that the patient has had previous attacks of urinary tract infection. Further, the frequency of *E. coli* of O groups less common in urinary isolates also was higher in the children with asymptomatic bacteriuria than in those with their first known infection, which is possibly a result of repeated infections which have induced immunity to the more common O antigens allowing only less common bacteria to invade the urinary tract.

Type of infection	% R phase E. coli	No. of urinary isolates
First known infection, girls	3	119
Recurrent infection, girls	6	63
Infection in pregnant women	29	302
Asymptomatic bacteriuria, women	40	121
Chronic pyelonephritis, women	44	34
Asymptomatic bacteriuria, girls	22	48

In our experience one third of the patients with a first recurrence after acute primary pyelonephritis are asymptomatic, whereas twice as many of those with further recurrences are asymptomatic.[4] Thus, the frequency of asymptomatic infections increases with the number of recurrences. This would suggest that asymptomatic bacteriuria mainly occurs in patients with previous attacks of urinary tract infections, although many of these might have passed unrecognized.

In our material more than 15% of the children with asymptomatic bacteriuria have signs of renal damage, in accord with the observation of Kunin.[1] We believe that the renal damage is not the forerunner of the urinary tract infections in most of these cases, but a sequel of the infections since such lesions develop in about 10% of the children who have a history of acute pyelonephritis.[5,6] The highest frequency is seen in patients who have had many, often asymptomatic, recurrences.

These findings suggest that asymptomatic bacteriuria might be considered a potentially dangerous disease in many cases and not a harmless invasion of the urinary tract, although in a few of our cases the bacteriuria has vanished spontaneously. Longitudinal studies of patients with asymptomatic bacteriuria may hopefully define the characteristics of the patients at risk.—We are, etc.,

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1 Kunin, C. M., Zacha, E., and Paquin, A. J., *New England Journal of Medicine*, 1962, 266, 1287.
2 Sussman, M., *et al.*, *British Medical Journal*, 1969, 1, 799.
3 Bettelheim, K. A., Taylor, J., *Journal of Medical Microbiology*, 1969, 2, 225.
4 Bergström, T., *Acta Paediatrica Scandinavica*, 1967, *Suppl.* 177, 45.
5 Bergström, T., in preparation.
6 Lindblad, B. S., and Ekengren, K., *Acta Paediatrica Scandinavica*, 1969, 58, 25.

D.T.P. Immunization by Intradermal Jet Injection

SIR,—With reference to the article by Professor J. P. Stanfield and his colleagues (22 April, p. 197) the following may be of interest. I described in 1944[1] a small investigation into the feasibility of using the intradermal route of administration of diphtheria aluminium precipitated toxoid (A.P.T.). The series consisted of 75 children mostly under four years of age, who were all originally Schick-positive. They were all treated one week after the test with a 0·1 ml syringe-administered intradermal dose of A.P.T. This was followed 28 days later by an identical injection. Schick tests performed