



basal duodenal disease and pyloric channel disease may lead to pyloric reflux. The deformed fibrotic pyloric opening in advanced pyloric channel disease is shown in the Fig.—I am, etc.,

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Chlamydia in Chronic Prostatitis

SIR,—As you pointed out (1 July, p. 1), it is rarely possible to trace the causative agent of chronic prostatitis. Lately TRIC agent has been suspected as a fairly common cause of non-specific genital infections.^{1,2} We studied sera from 79 untreated patients with chronic or subchronic prostatitis and 72 age-matched registered male blood donors for complement-fixing antibodies to chlamydia. It is known that an antibody response to infections with chlamydia may be detected by complement fixation tests to shared group antigens. We used a psittacosis antigen (Wellcome). Complement-fixing antibodies in a titre of at least 1:5 were found in 33% of the patients with prostatitis but in only two of the blood donors (see Table)

Cultures of urethral swabs and of expressed prostatic and seminal fluids from the patients with prostatitis almost always gave growth only of such bacteria as normally constitute the flora in the distal urethra. None grew gonococci, nor did cultures of rectal swabs. But immunofluorescent studies of seminal smears suggested infection with gonococci in five of the cases. In a few cases it seemed that *Candida albicans*, *Corynebacterium vaginalis*, mycoplasmas, *Trichomonas vaginalis*, or a virus might be the causal agent.

Treatment with metacycline proved more successful ($P < 0.1$) in the patients with complement-fixing antibodies to chlamydia than in those without, as judged from the relief of symptoms. Chlamydia infections may be treated with antibiotics such as

tetracyclines, but recent work suggests that such treatment may not be so effective as supposed.³—We are, etc.,

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Rosette Inhibition Test and Renal Transplantation

SIR,—Despite the detailed report of the method employed by Mr. A. Munro and others (31 July, 1971, p. 271) to assay rosette inhibition, many difficulties may be encountered before reproducible results are obtained. This is to be expected in a test whose mechanism is ill-understood, and a recent report on the influence of physical factors on rosette inhibition¹ serves to emphasize these difficulties. Once reproducible results are obtained, however, in a test that utilizes 10 serial dilutions a shift of two or more dilutions from examination to examination should be regarded as significant.

Our group has found that unimmunosuppressed subjects exhibit a mean inhibitory concentration (M.I.C.) of antilymphocyte globulin between 1:16,000 and 1:32,000. Well suppressed transplant patients have a M.I.C. of about 1:128,000. We note that 24-48 hours before rejection may be diagnosed clinically the patient's M.I.C. sinks to 1:4,000-1:8,000. That is four to five dilution steps difference from "suppressed" to "unsuppressed." We believe that once a patient has been given the immunosuppressive drugs referred to a "normal" level is meaningless. The change in M.I.C. from a level indicating "immunosuppression" to one, two or more dilution steps lower, however, is important.

Our results with the rosette inhibition test seem to confirm the findings of Bewick's group. We are impressed by the early warning of impending rejection that the test appears to give.—We are, etc.,

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Diagnosis of Renal Agenesis

SIR,—Drs. N. C. De and J. R. Harper (16 September, p. 696) draw attention to the problem of resuscitation in infants subse-

quently found to have severe renal anomalies. With improvements in care the perinatal mortality and morbidity directly attributable to obstetric causes has declined and the recognition of infants with external manifestations of severe renal malformations assumes greater significance. We also have encountered instances where intensive resuscitative measures have been applied for considerable periods to such infants who had no hope of survival. Diagnosis is not easy, especially when pressures from labour confuse the characteristic facial appearances in the early neonatal period. Also somewhat similar features can be seen associated with the leaking liquor syndrome.¹ These infants, like renal agenetics, have pulmonary hypoplasia and consequently present problems in resuscitation, but it is important to distinguish them as they may be saved.

Many other factors besides the facial appearance should be considered. Help may be obtained from a careful obstetric history. In infants with severe renal malformations the presence of bilateral talipes and spade-like hands may be of assistance in diagnosis.² Birth weight tends to be low in renal agenesis but not in cases of severe renal cystic dysplasia or of congenital urinary tract obstruction such as urethral atresia or stenosis. Another factor which can prove of great value in diagnosis is macroscopic examination of the placenta, which in most cases of oligohydramnios associated with failure of fetal micturition will reveal the presence of amnion nodosum.^{3,4} Amnion nodosum may be present in association with the leaking liquor syndrome but is much less common in this condition.

Over-emphasis tends to be placed on the facial appearance of infants with severe renal malformations without due consideration of all the other features. Nevertheless, diagnosis will in a few instances present difficulties and resuscitative measures should be undertaken in doubtful cases lest a normal infant affected only by prolonged leakage of liquor be allowed to die.—We are, etc.,

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Immunological Responses in Pregnancy

SIR,—In their paper demonstrating depression of cell-mediated immunity during pregnancy, Dr. Ronald Finn and others (15 July, p. 150) predict that the function of the lymphocytes in the bursa-derived system would not be reduced and might even be increased during pregnancy. They refer to the work of Woodrow and others,² who found no reduction of humoral antibody production in pregnant rabbits.

We have examined the effect of pregnancy on insulin-binding antibodies present in the serum of insulin-treated patients. In all patients studied insulin antibody levels fell as pregnancy progressed so that by the third

Group	No. of Subjects	Complement-fixing Antibody Titres					
		1/5	1/10	1/20	1/40	1/80	
Prostatitis	79	53	3	12	6	4	1
Controls	72	70	—	1	1	—	—