

equipment. The method can also be used to concentrate urine samples for electrophoresis.

I am grateful to Mr J Millar for constructing the device and to Ms P Price for typing the manuscript.

References

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Letters to the Editor

Growth of *Neisseria gonorrhoeae* in a simple medium at 28°C

Although it is generally documented that *Neisseria gonorrhoeae* does not grow below 30°C there seems no more precise information on the minimum of its growth temperature range.¹ During some studies of simple media for the cultivation of this organism, it was observed that growth of all of a few strains tested did in fact occur at 28°C but not at 24°C. It was decided to examine a larger batch of isolates with a view to obtaining more data on the minimum growth temperature for this species.

The test medium was a semi-solid one which consisted of brain heart infusion broth (Difco) to which was added agar powder (0.1%) and soluble starch (0.1%). It was dispensed in 4 ml volumes in plastic tubes (100 × 12 mm) and autoclaved at 121°C for 15 minutes.

The organisms tested were a batch of 65 freshly collected clinical isolates. After ensuring purity of the isolates, growth was emulsified in nutrient broth and 2 drops of dense suspension were inoculated on to the surface of the test medium. One tube was incubated in a cabinet at 36°C and the other in an agitated water bath at a temperature of 27.7 ± 0.2°C (hereafter referred to as 28°C). The bath thermometer was calibrated against a standard instrument. Two chocolate agar plates were also inoculated; one was incubated at 36°C in 10% carbon dioxide and the other in air at room temperature (20-22°C).

The control plates at 36°C all yielded growth and those at room temperature

none. All tubes of the test medium showed growth after varying periods. Those at 36°C showed distinct turbidity within 24 hours whereas those at 28°C did so within 24 to 48 hours. Growth first appeared as a shallow surface sludge. When this was disturbed by gentle shaking and distributed into the deeper regions of the medium, growth continued and produced an evenly turbid suspension to a depth of about 15 mm. The final density of this growth varied widely among the isolates and in general correlated with the colony size of the isolates on chocolate agar. No critical survival study was made on the cultures but many, though not all of those tested, yielded growth when plated after about 30 days.

The semi-solid agar is proving a useful medium for growth and short term maintenance of gonococcal strains. Other semi-solid media such as that devised by Vera² and marketed as Cystine Trypticase Agar (Baltimore Biological Laboratories) have been used for many years for this purpose. Serum enrichment as advocated by some workers³ would appear to be unnecessary in media designed for the maintenance of the gonococcus.

It should be of interest to extend the investigations on low temperature growth of the gonococcus particularly to include observations on agar surfaces. Meanwhile it is now established that this organism does, under some cultural conditions, grow at a temperature of as low as, at least, 28°C.

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I should like to thank Mr M Blums, State Health Laboratory Services, Perth, Western Australia for kindly identifying and supplying the strains tested.

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IgM mesangial deposits in nephrotic syndrome

We have studied 14 patients (aged 8-39 years) with an idiopathic nephrotic syndrome. Seven patients were male and seven female. On light microscopy, moderate increase in cells and mesangial matrix were observed in all cases. The most characteristic findings by immunofluorescence microscopy were generalised and diffuse mesangial granular deposits of IgM in all the biopsies. There was C3 deposition in 12 cases, and C1q and C4

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were seen in two and three cases respectively. Ultrastructural examination showed electron-dense deposits localised in the mesangium in nine cases.

Patients were treated during the first month with prednisone 1 mg/kg per day. During the next two months the dose was reduced to 1 mg/kg on alternate days and then gradually decreased. Steroid-resistant patients were treated with 0.2 mg/kg per day of chlorambucil for three months. In no case was the total dose of chlorambucil greater than 25 mg/kg. One nephrotic patient experienced spontaneous remission and eight others were steroid-responsive. Only one of the five steroid-resistant patients treated with chlorambucil showed clinical remission. Five steroid-responsive patients relapsed. At present, four patients are "healthy," having not relapsed for the past two years.

We think it is important to determine if the presence of IgM in renal biopsies indicates a poor prognosis in idiopathic nephrotic syndrome. Cohen *et al.*¹ have suggested a relatively poor prognosis in five such patients after treatment, showing clinical remission in only one case. On the other hand, Bhasin *et al.*³ described six of eight patients who initially achieved clinical remission, although four of these later required cytotoxic drugs or were steroid-dependent. It therefore appears that whereas the short-term prognosis in IgM associated mesangial proliferative glomerulonephritis is good, the long-term outlook must be much more guarded.³

We do not know whether IgM mesangial nephropathy is really a single disease entity or whether the IgM deposition is simply a concomitant immunological finding not necessarily related to the pathogenesis of the nephrotic syndrome.⁴ Only a small number of cases have been reported of this ill-defined glomerular disease. A definitive statement regarding the course and prognosis is not possible. More studies are necessary to confirm that IgM mesangial nephropathy is indeed a separate entity.

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- ³ Lawler W, Williams G, Tarpey P, Mallick NP. IgM associated primary diffuse mesangial proliferative glomerulonephritis. *J Clin Pathol* 1980;**33**:1029-38.
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Dr Lawler and his colleagues comment as follows:

Thank you for inviting us to comment on the letter by Gonzalo *et al.* The cases which they describe appear to be very similar to ours¹ in structural and immunopathological terms, although criteria for selection were different; thus all their 14 patients had the nephrotic syndrome, whereas 9 of our 23 patients¹ had asymptomatic proteinuria.

In our experience, based on these 23 cases¹ and another unpublished group of 20 similar cases of IgM-associated primary diffuse mesangial proliferative glomerulonephritis, both clinical remission during steroid therapy and spontaneous improvement are uncommon, the majority of patients pursuing a chronic indolent course which, in a minority, progresses to end-stage renal failure. It may well be that what we and others,^{2,3} including Gonzalo *et al.*, have described is a heterogeneous group, and that the patients who improve, either spontaneously or with steroid therapy, may represent a different pathogenetic mechanism. Nevertheless, the fact that the majority do not improve suggests that they should be considered as a distinct clinicopathological group.

We agree that further long-term studies are required to confirm IgM mesangial nephropathy as a separate entity and, if so, to determine its course and ultimate prognosis.

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Misapplication of Russell's name

The paper by Bartolini *et al.* (October 1980;**33**:936) contains clerical slips in the spelling: Russell's bodies, but a more serious error in suggesting that the intracellular bodies described by Russell (1890)¹ occur within plasma cells. Russell stated that these fuchsinophil bodies occurred within and around cancer cells, but not in sarcomata or in simple tumours.

Modern staining methods make it fairly certain that the inclusions seen in carcinoma cells are of fibrin, and this gains support by the greater number of inclusions within carcinoma cells adjacent to fibrinous coagula. On the other hand, the inclusions within plasma cells are now linked to immunological changes, do not stain exactly as fibrin, and surely should never be called Russell bodies.

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Ethanol-induced vacuolation in red cells

The article "Cytoplasmic vacuolation of peripheral blood cells in acute alcoholism" (*J Clin Pathol* 1980;**33**:1193-6) found our interest. Working in the field of haemorrhology, we carry out studies of red cell deformability (RCD) using a filtration method based on a technique originally developed in our laboratory and described in this Journal.^{1,2} Studying healthy volunteers, we observed that RCD was reduced after alcohol intake during the night before the measurement (on average 13%). Subsequently we performed a series of in vitro experiments, determining the RCD of physiologically deformable red