long storage. Legionellas of "species 1 and 2"¹¹ reacted in the test but the other legionellas examined did not, nor did *Brucella abortus*, *Bacteroides fragilis*, or *Pseudomonas aeruginosa*.

The test was easy to use, with very clear instructions, and was found to be extremely helpful in the preliminary sorting out of strains of legionellas (characterised initially as Gram negative rods growing on buffered charcoal yeast extract agar, with typical colonies showing a "cut glass appearance" and failing to grow in the absence of added cysteine and iron in the medium), which had been isolated both from clinical and environmental sources. As reported⁹ the monoclonal antibody also reacted strongly with legionellas in smears made from lungs from which *L. pneumophila* serogroup 1 had been isolated and also from lung from which serogroup 8 had been isolated. Economical use of the expensive reagent can be made by using a 2 mm platinum wire loop to add reagent to the wells in the slides (care being taken to ensure the reagent does not dry on the slide during incubation), and it was found that it was much easier to locate the stained film of organisms, or tissue, using the white coated Teflon slides in routine use in this laboratory (Hendley, Essex) than with the grey Teflon coated slides supplied with the kit. We find PVA mountant¹² more easy to work with in this laboratory than the buffered glycerol supplied with the kit.

This kit obviously has many applications in the preliminary identification of possible legionellas before further examination by reference laboratories, as well as in the examination by the DFAT of specimens for the presence of *L. pneumophila*. Where it is essential that the organism isolated and characterised should be differentiated from "species 1 and 2," however,¹¹ such cultures will have to be examined further to determine their precise identity.

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References

- Fallon RJ. Laboratory diagnosis of Legionnaires' disease. Association of Clinical Pathologists Broadsheet 99. London: Association of Clinical Pathologists, 1981.
- McKinney RM, Thacker L, Harris PP, et al. Four serogroups of Legionnaires' disease bacteria defined by direct immunofluorescence. *Ann Intern Med* 1979;90:621-4.
- Thacker WL, Wilkinson HW, Benson RF. Comparison of slide agglutination test and direct immunofluorescence assay for identification of Legionella isolates. *J Clin Microbiol* 1983;18:1113-18.
- Thacker WL, Plikaytis BB, Wilkinson HW. Identification of 22 Legionella species and 33 serogroups with the slide agglutination test. *J Clin Microbiol* 1985;21:779-82.
- Moss CW, Weaver RE, Dees SB, Cherry WB. Cellular fatty acid composition of isolates from Legionnaires' disease. *J Clin Microbiol* 1977;6:140-3.
- Karr DE, Bibb WF, Moss CW. Isoprenoid quinones of the genus Legionella. *J Clin Microbiol* 1982;15:1044-8.
- Brenner DJ. Classification of Legionellae. In: *Legionella. Proceedings of the Second Symposium*. Thornsberry C, Balows A, Feeley JC, Jakubowski W, eds. Washington DC: American Society for Microbiology, 1984:55-60.
- Gosting LH, Cabrian K, Sturge JC, Goldstein LC. Identification of a species specific antigen in Legionella pneumophila by a monoclonal antibody. *J Clin Microbiol* 1984;20:1031-5.
- Edelstein PH, Beer KB, Sturge JC, Watson AJ, Goldstein L. Clinical utility of a monoclonal direct fluorescent reagent specific for Legionella pneumophila: comparative study with other reagents. *J Clin Microbiol* 1985;22:419-21.
- Edelstein PH. Improved serum selective medium for isolation of Legionella pneumophila from contaminated clinical and environmental species. *J Clin Microbiol* 1981;14:298-303.
- Selander RK, McKinney RM, Whittam TS, et al. Genetic structure of populations of Legionella pneumophila. *J Bacteriol* 1985;163:1021-37.
- Heimer GV, Taylor CED. Improved mountant for immunofluorescence preparations. *J Clin Pathol* 1974;27:254-6.

Normocellular bacterial meningitis

Case reports

CASE 2

A two year old girl was admitted with a three day history of mild diarrhoea and anorexia with occasional vomiting. Four hours after admission she developed a widespread purpuric rash, and a lumbar puncture was performed. The cerebrospinal fluid was acellular with protein and glucose contents of 0.16 g/l and 3.9 mmol/l (70.2 mg/100 ml), respectively. (Adult cerebrospinal fluid refer-

ence ranges 0.1-0.4 g/l protein and 2.2-3.3 mmol/l (39.6-59.4 mg/100 ml) glucose.) A Gram film was not examined. Despite treatment with intravenous benzyl-penicillin she died three hours later. *Neisseria meningitidis* group B was subsequently grown from both cerebrospinal fluid and blood.

CASE 2

An eight month old girl was admitted with a one day history of lethargy and occasional vomiting. There was minimal neck stiffness, and purpuric spots were noted over her back, legs, and buttocks. The cerebrospinal fluid was acellular with protein and glucose contents of 0.25 g/l and 2.1 mmol/l (37.8 mg/100 ml), respectively. No organisms were seen in the Gram film of a spun deposit. *N. meningitidis* group B type 8 was subsequently grown from both the cerebrospinal fluid and blood. She made a complete recovery with intravenous benzylpenicillin.

CASE 3

An 84 year old woman who had received a splenectomy was admitted with a 24 hour history of fever and confusion. Although no signs of meningism were present, a lumbar puncture was performed. The cerebrospinal fluid cell count was: polymorphs 2.0×10^6 /l, lymphocytes 2.0×10^6 /l, red cells 1.0×10^6 /l; protein and glucose contents were 0.25 g/l and 5.3 mmol/l (37.8 mg/100 ml), respectively. Numerous Gram positive diplococci were seen in the Gram film of a spun deposit, and *Streptococcus pneumoniae* serotype 12 was grown from both the cerebrospinal fluid and blood. She was treated with intravenous benzylpenicillin and made a full recovery.

CASE 4

A 28 year old woman was admitted with a one day history of sore throat and generalised arthralgia. Minimal neck stiffness and a widespread purpuric rash were noted. The cerebrospinal fluid cell count was: polymorphs 1.0×10^6 /l, red cells 3.0×10^6 /l, and protein and glucose contents were 0.39 g/l and 4.8 mmol/l (86.4 mg/100 ml), respectively. Numerous Gram negative diplococci were seen in the Gram film of a spun deposit and *N. meningitidis* group B type 2b was grown from the cerebrospinal fluid and blood. She was treated with intravenous benzylpenicillin and made a full recovery.

Discussion

Normal cerebrospinal fluid white cell counts

have also been reported in *Haemophilus influenzae*¹ and coliform meningitis.^{2,3} The occurrence of normocellular bacterial meningitis, however, is rarely emphasised in the standard infectious disease textbooks. We have experienced four such cases (three of meningococcal meningitis and one of pneumococcal meningitis) over two and a half years, representing 7% of our culture positive cases. In two of our patients (cases 1 and 3) lumbar puncture was performed before signs of meningism were present. The laboratory findings probably represented an early stage in the cellular response.

We recommend that a Gram stain and culture of a centrifuged deposit should be performed on all samples of cerebrospinal fluid, irrespective of cellular findings.

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References

- 1 Milne A, Hamilton W. Developing or normocellular meningitis. *N Z Med J* 1976; **84**:6-8.
- 2 Heckmatt JZ. Coliform meningitis in the newborn. *Arch Dis Child* 1976;**51**:569-73.
- 3 Christie AB. Acute bacterial or pyogenic meningitis. In: *Infectious Diseases: epidemiology and clinical practice*. Edinburgh: Churchill Livingstone, 1980:605-46.

Catalase negative *Staphylococcus aureus*

We report the isolation of a catalase negative strain of *Staphylococcus aureus* from a chronic paronychia in a 67 year old man attending a dermatology clinic. Catalase negative strains of *S aureus* isolated from human sources have rarely been reported.¹⁻³ The table lists the characteristics of this isolate.

The susceptibility of this isolate to hydrogen peroxide was compared with that of *S aureus* strain Oxford (NCTC 6571). The concentrations of hydrogen peroxide required to kill an inoculum of 10⁷ staphylococci/ml of nutrient broth after four hours at 37°C were 0.0018% and 0.00375%, re-

Characteristics of catalase-negative strain of *S aureus*

Test	
Haemolysis	Beta (horse blood)
Tube coagulase	Positive
DNase ⁴	Positive
Staphaurex*	Positive
API biotype	6736151
Phage type	Not typable (RTD × 100)
Pigment	Golden-yellow (milk agar)
Penicillinase	Positive
Mannitol fermentation	Positive
Lysostaphin sensitivity	Positive

*Wellcome Diagnostics.

spectively. Using the method of Van Furth *et al*,⁵ we found that the susceptibility of this isolate and *S aureus* strain Oxford to neutrophil killing under aerobic conditions were also similar. A Clark oxygen electrode⁶ was used to confirm the absence of oxygen production from an overnight culture of this isolate in nutrient broth after hydrogen peroxide had been added.

The importance of catalase in determining the virulence of *S aureus*, particularly in conditions such as chronic granulomatous disease, needs to be clarified. This report illustrates that catalase production is not essential for survival of *S aureus* in vitro or in vivo.

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References

- 1 Carlson JR, Gorin DC. Catalase-negative *Staphylococcus aureus*. *Clinical Microbiology Newsletter* 1981;**3**:33-4.
- 2 Tu KK, Palutke WA. Isolation and characterization of a catalase-negative strain of *Staphylococcus aureus*. *J Clin Microbiol* 1976; **3**:77-8.
- 3 Lucas PR, Seeley HW. A catalase-negative *Micrococcus pyogenes* var *aureus*. *J Bacteriol* 1955;**69**:231.
- 4 Disalvo JW. Deoxyribonuclease and coagulase activity of micrococci. *US Armed Forces Medical Journal* 1958;**9**:191-6.
- 5 Van Furth R, Van Zwet TL, Leijh PCJ. In vitro determination of phagocytosis and intracellular killing by polymorphonuclear and mononuclear phagocytes. In: Weir M, ed. *Handbook of experimental immunology*. Oxford: Blackwell Scientific Publications, 1978: 32.1-32.19.
- 6 Rorth M, Jensen PK. Determination of catalase activity by means of the Clark oxygen electrode. *Biochim Biophys Acta* 1967;**139**:171-3.

Ploidy studies in adenomatous polyps of the colon

We are surprised that Whitehead *et al* failed to find aneuploid cells in a series of 16 adenomatous polyps of the colon. We detected aneuploid cells in 18% of similar polyps in a larger series, using Feulgen staining and microdensitometry. In view of the well established finding of increased proliferative activity associated with dysplasia in polyps² we are further surprised that evidence of proliferation was present in only two of the 10 polyps with moderate or severe dysplasia.

We obtained our cells by cytologic brushings, but we used a similar disaggregation technique for fixed paraffin wax embedded tissue from the breast. In this experiment we were able to show aneuploidy in dysplastic lesions, and therefore cell preparation techniques do not seem to be responsible for the discrepancy in results.

Further evidence that aneuploidy occurs before frank invasion comes from a study of cellular DNA in chronic ulcerative colitis. Aneuploid cells were found in 62.5% of biopsy specimens showing severe dysplasia, contradicting the main hypothesis of Whitehead *et al*.

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References

- 1 Whitehead F, Jarvis LR, Skinner JM, Whitehead R. Ploidy studies in adenomatous polyps of the colon. *J Clin Pathol* 1985;**38**:1261-4.
- 2 Deschner EE. Experimentally induced cancer of the colon. *Cancer* 1974;**34**:824-8.
- 3 Cuvelier CA, Morson BC, Roels HJ. The DNA content in cancer and dysplasia in chronic ulcerative colitis. *Br J Surg* 1985;**72**:5133.