have also been reported in *Haemophilus influenzae*1 and *coli* menigitis.2 3 The occurrence of normocellular bacterial menigitis, 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 normocellular findings.

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References


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.1–2 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 105 *staphylococci/ml* of nutrient broth after four hours at 37°C were 0.0018% and 0.00375%, respectively. Using the method of Van Furth *et al.*,3 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 electrode 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


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 adenomatous polyps 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 discrepency 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