ELISA, was recorded as positive by both observers. The frequency of false-positive reactions is being studied on a large panel of sera.

Because the assay may be used in developing countries, we invited three additional staff members to read the results obtained with panel 2. Two were western-trained medical laboratory scientists; the third was a visiting laboratory technologist who is responsible for all serological tests and screening of blood in his country. The degree of variability in the results is alarming (table III) and suggets that if this test is to be widely used in the field or in developing countries, considerable attention will need to be paid to staff training, the provision of control sera with visual aids (photographs) to assist in the interpretation of agglutination patterns, and the provision of proficiency panels.

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BORRELIA INFECTION AS A CAUSE OF PRESENILE DEMENTIA

SIR,—Tick-borne *Borrelia burgdorferi* is a known cause of meningitis, often associated with cranial nerve paresis and/or peripheral neuritis.[1] Acute encephalitis resulting in severe sequelae has also been reported.[2] We describe severe presenile dementia caused by *Borrelia*, which developed in a man without clinical signs of meningitis or encephalitis.

A 60-year-old man had been in good health until the summer of 1985. He was on drug treatment for mild hypertension. During the spring of 1986 he was brought to hospital by his relatives as he had lost his memory during the last few months and his personality had changed; he could not cope with daily activities; he did not know whether it was day or night; and he was incontinent for urine and faeces. He was admitted to a psychogeriatric ward with a diagnosis of rapidly progressing dementia. He had no paresis and computerised tomography of the brain was normal. Lumbar puncture revealed 285×10^6/l mononuclear cells, a high concentration of CSF protein, and a decreased glucose value. Serological analysis showed antibodies against *Borrelia* in cerebrospinal fluid (CSF).

During the summer of 1985, the patient had had an erythema on his right knee, but was not aware of any tick-bite. 1 month later, he saw his doctor because of severe pain in his left leg and in the upper right quadrant of his abdomen. The pain disappeared, but later he had pains in one shoulder which continued until penicillin treatment was given after admission in June, 1986. During the first 4 months of 1986, 6 months after what was probably an erythema chronicum migrans in the summer of 1985, the changes in his personality became increasingly obvious to his wife. This led to new contact with the hospital and in June the diagnosis of tick-borne borreliosis was established.

After a 14-day intravenous course of benzylpenicillin CSF analysis showed a reduction in the number of mononuclear cells and spinal protein. However, the titre against *Borrelia* in CSF had increased. The patient's general condition improved after treatment: he is now able to walk, he can control his bladder and bowels and his mental condition has improved. But 1 year after treatment his memory is still poor and he needs daily supervision and help from his wife.

CSF—ANALYSIS AND SEROLOGICAL TESTS BEFORE AND AFTER TREATMENT WITH PENICILLIN IN PATIENT WITH PRESENILE DEMENTIA CAUSED BY B BURGDORFERI

	Before treatment	After treatment
CSF:		
Mononuclear cells (10^6/l)	285	88
Protein (mg/l)	3600	700
Albumin (mg/l)	1905	376
Serum albumin (g/l)	32	29
Borrelia titre:		
CSF	128	570
Serum	2800	2200

The diagnosis of *Borrelia* infection as the cause of his brain disease was based on the finding of antibodies against *Borrelia* in CSF. The table shows titres against *Borrelia* in serum and CSF as determined by ELISA (National Bacteriological Laboratory, Stockholm).

Stiernstedt[3] has proposed a spirochaete index to separate antibodies that are produced intrathecally from serum antibodies that passively diffuse into CSF by barrier leakage. This index is calculated by dividing the CSF/serum ELISA titre by CSF/serum albumin. An index over 2 suggests intrathecal antibody production. In our case this index was 20 for *Borrelia* antibodies although the index for other viruses (eg, herpes simplex virus) was 1 after penicillin treatment. The high titres of *Borrelia* antibodies in CSF and the improvement on treatment with benzylpenicillin indicate that the central nervous disease of our patient was caused by *B burgdorferi* infection. Progressive dementia without concomitant signs of meningitis or encephalitis should be added to the list of manifestations of tick-borne borreliosis.

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NON-PARENTERALLY TRANSMITTED NON-A, NON-B HEPATITIS IN CHILDREN IN THE UK

SIR,—Non-parenterally acquired non-A, non-B hepatitis (NANBH) is a significant problem worldwide, and has been reported in Asia, the Middle East, Africa, the USSR, the USA, and France.[1-3] It is generally thought to be so unusual in the UK that patients in low-risk groups who present with hepatitis B surface antigen (HBsAg) negative hepatitis have usually been diagnosed clinically as having hepatitis A, without necessarily measurement of hepatitis A IgM levels.

A 9-year-old girl was admitted to this hospital with hepatitis, and she was still unwell 3 weeks after the onset of the illness. She was negative for HBsAg and for hepatitis B core (HBcAb), hepatitis A IgM, Epstein-Barr virus (EBV) capsid, and cytomegalovirus (CMV) antibodies. She died with fulminant hepatic failure, and necropsy revealed massive hepatic necrosis consistent with viral hepatitis.

6 weeks later a 13-year-old boy was admitted with a 3-week history of jaundice, clinically typical of hepatitis A. He too was HBsAg and HBcAb, hepatitis A IgM negative and had no antibody to EBV or CMV. 9 weeks after the onset of his illness severe aplastic anaemia developed.

These 2 cases, not linked epidemiologically, prompted us to do a retrospective survey of children under 16 years of age presenting to this hospital between 1983 and 1985, with a clinical diagnosis of hepatitis A. 133 children were identified, for 47 of whom serum was still available. 3 were found to be negative for hepatitis A IgM, and to have negative or insignificant antibody titres to HBsAg, HBcAb, EBV capsid, and CMV.

All cases presented with nausea, vomiting, anorexia, and jaundice, clinically typical of hepatitis A. None of the children had risk factors for parenterally acquired disease, and none had been out of the UK in the 6 months before the onset of illness. Two cases presenting at the same time were brothers; although no stored serum was available for one of them, it is probable that they were both affected by the same agent, hence the inclusion of both in the study. There was no known epidemiological factor linking the others.

In the past five years there have been at least 6 cases of NANBH in children in North-West England. None of these children—