approaches to seeking consent. In our study[8] of different procedures for obtaining consent to randomised treatment in cancer clinical trials we found that total disclosure of all information compared with an individual approach to seeking consent resulted in a better understanding of treatment, side-effects, and research aspects of the treatment, but less willingness to agree to randomised treatment, and increased anxiety. No significant differences were found in patient perception of the doctor-patient relationship. We hope that these results will stimulate similar controlled trials of consent practices at other hospitals.

Ludwig Institute for Cancer Research,
Sydney Cancer Therapy Branch,
University of Sydney,
Sydney, NSW 2006, Australia

R. J. SIMES
M. H. N. TATTERSALL

1. White DR, Muss HB, Michielutte R, et al. Informed consent: Patient information forms in chemotherapy trials. *Am J Clin Oncol* 1984; **7:** 183–90.
2. Reynolds PM, Sanson-Fisher RW, Poole AD, Harker J, Byrne MJ. Cancer and communication. Information-giving in an oncology clinic. *Br Med J* 1981, **282:** 1449–51.
3. Saurbrey N, Jensen J, Rasmussen PE, Gjorup T, Guldager H, Riis P. Danish patients' attitudes to scientific-ethical questions. An interview study focussing therapeutic trials. *Acta Med Scand* 1984; **215:** 99–104.
4. Elian M, Dean G. To tell or not to tell the diagnosis of multiple sclerosis. *Lancet* 1985; ii 27–28.
5. Cassileth BR, Zupkis RV, Sutton-Smith K, March V. Information and participation preferences among cancer patients. *Ann Intern Med* 1980; **92:** 832–36.
6. Fraser AG. Do patients want to be informed? A study of consent for cardiac catheterisation. *Br Heart J* 1984; **52:** 468–70.
7. Alfidi RJ Controversy, alternatives and decisions in complying with the legal doctrine of information consent. *Radiology* 1975; **114:** 231–34.
8. Simes RJ, Tattersall MHN, Coates AS, et al. Randomised comparison of procedures for obtaining informed consent in clinical trials of treatment for cancer. *Br Med J* 1986; **293:** 1065–68.

PAIN AND THE MOTHER-CHILD INTERACTION

SIR,—According to current theories of mother-child interaction, a child between the ages of about three and six months is in symbiotic relation with its mother. In this relation, it is said, unpleasant stimuli are projected outside the shared milieu and do not upset the fragile relationship.[1] One might therefore expect childen of this age to react more lengthily to pain experienced when not in direct contact with their mothers.

In Sweden, children between three and six months of age are given three shots of vaccine against tetanus and diphtheria (Duplex-vaccin pH 5,8-6). It is locally irritating and painful. Reactions to vaccination, measured as duration of crying, have been compared in two groups allocated at random, one sitting in the arms of the mother (n = 17), the other lying on a couch without contact with the mother (n = 21) while being injected. In the second group the child was given to the mother immediately after vaccination. Either way she was encouraged to comfort the child.

The children who were vaccinated while in the mother's arms cried for significantly longer than those vaccinated without maternal contact (table). This could mean that the child in the arms

DURATION OF CRYING AFTER VACCINATION

Duration (s)	Age (mo)		
	3	4·5	6
In mother's arms (n = 17)	91 *(53)*	51 *(39)*	50 *(25)*
On couch (n = 21)	34 *(20)*	28 *(12)*	25 *(16)*
p	0·001	0·001	0·01

Numbers in parenthesis are SDs.

of mother, when experiencing external pain, perceives it as coming from the mother herself. Thus the relationship is disturbed and comforting is made more difficult, which is contrary to current theories of mother-child interaction. It is noteworthy that as the children approached the age of six months the differences become smaller, so the relation between child and mother may be less easily "poisoned" with increasing age.

These observations provide an argument for vaccinating such infants with the mother apart, though standing by ready to give comfort, unless she particularly wants to hold the child.

Hallunda Health Clinic,
Box 104, S-145 01 Norsborg,
Sweden

GÖSTA ALFVÉN

1 Mahler M, Pine F, Bergman A. The psychological birth of the human infant. New York: Basic Books, 1975.

FETAL ALCOHOL SYNDROME IS NOW LEADING CAUSE OF MENTAL RETARDATION

SIR,—Fetal alcohol syndrome (FAS) is a pattern of birth defects in children born to alcoholic women, consisting of prenatal and postnatal growth retardation, facial anomalies, organ pathology, and central nervous system dysfunction, including mental retardation. We have reviewed nineteen epidemiological studies in which 164 FAS cases were reported from populations totalling 88 236 live births—a frequency of 1·9 per 1000.[1]

In a study of the frequency of chromosomal anomalies done at six hospitals in the United States on every live born infant (56 952 consecutive live births) the rate for Down syndrome was 1·25 per 1000.[2] In a thorough study in Canada, as part of the British Columbia Health Surveillance Registry, Baird and Sadovnick[3] found that the rate of spina bifida was 1 per 1000 and that about 50% of those identified had mental retardation. At the time the population of British Columbia was 2 7 million.

Shortly after they identified FAS as a clinical entity, Smith and his colleagues[4] stated that "in our experience, the fetal alcohol syndrome has become the third ranking recognised disorder in which mental deficiency is a feature. Only Down's syndrome and neural tube defects, such as meningomyelocele are more common causes". Since that statement was made ten years ago much more information, such as the findings we have presented, has become available. Smith and colleagues' conclusion needs to be amended. Mental retardation is a cardinal feature of FAS, and the syndrome, has emerged as the number one recognised disorder in which mental deficiency is a feature.

Department of Obstetrics and Gynecology,
Wayne State University,
Detroit, Michigan 48201, USA

ERNEST L. ABEL
ROBERT J. SOKOL

1. Abel EL, Sokol RJ. Incidence of fetal alcohol syndrome and economic impact of FAS related anomalies. *Drug Alcohol Dependence* (in press).
2. Russell D. Mental handicap New York: Churchill Livingstone, 1985.
3. Baird PA, Sadovnick AD Mental retardation in over half-a-million consecutive live births an epidemiological study. *Am J Ment Deficiency* (in press).
4. Smith DW, Jones KL, Hanson W Perspectives on the cause and frequency of the fetal alcohol syndrome *Ann NY Acad Sci* 1976; **243:** 138–39.

ACUTE TRANSVERSE MYELITIS AS PRESENTING NEUROLOGICAL FEATURE OF LYME DISEASE

SIR,—With up-to-date, specific serological tests a pathogenic role for *Borrelia burgdorferi* has been demonstrated in some neurological disorders lacking the characteristic triad of Lyme disease.[1] We describe here a case in which acute transverse myelitis (ATM) was the prominent neurological manifestation.

In August, 1986, a 19-year-old woman living in the Belgian Ardennes, an endemic area for *B burgdorferi* infection, complained of a pruritic swelling on her left ankle. Her general practitioner noted a 3 cm skin lesion resembling erythema chronicum migrans—a red ring surrounding a clear central part with the mark of a bite in the middle. She could not recall having been bitten by an insect. The only treatment prescribed was a topical anti-inflammatory preparation. 2 days later, she noted numbness on the right side of her chest, extending to affect the whole of her right leg in about 2 days. Thereafter, paraesthesias were noted in both legs. On day 15, when she complained of gait disturbance and weakness in her legs, she was referred to our hospital. Neurological examination revealed severe spasmodic paraparesis with hyperreflexia and bilateral extensor plantar responses. There was a T4 level for pin, temperature, touch and vibration. The small muscles of her left hand and dorsal muscles of the left forearm were weak. Motor reflexes were normal except for a reduced right triceps response. She had difficulty urinating and was constipated.

ATM was diagnosed. The CSF contained 10 white blood cells (90% lymphocytes, 10% monocytes) and protein 39 mg/dl, with a striking increase in gammaglobulins and more than twenty oligoclonal bands. IgG intrathecal synthesis was 28 mg per day (normal below 3·3 mg). Bacterial, fungal, and cryptococcal tests were negative. 1 week later the CSF contained 40 white cells (95% lymphocytes) and protein 60 mg/dl. Electrophoresis and isofocusing confirmed the same abnormalities. IgG intrathecal

synthesis was 76 mg per day. Complete viral check-up was unremarkable. Motor and sensory nerve conduction velocities studies were normal in arms and legs. Pattern shift visual, brainstem auditory, and median nerve short latency somatosensory evoked potentials were normal. Both peroneal somatosensory evoked potentials were reduced in amplitude and slightly delayed. Multiple sclerosis (MS) was excluded when antibody tests, done because of the erythema chronicum migrans, revealed high-titre antibody to *B burgdorferi*. An immunofluorescence (IF) assay was positive at 128 (IgG) and the ELISA titre was 359 by spectrophotometry (the cut-off for our laboratory was 165). The patient was put on intravenous penicillin G sodium 20 megaunits daily for 15 days and her neurological state improved rapidly. At the end of the penicillin treatment the IF antibody titre was still at 128 (IgG) but the ELISA titre had fallen to 199. On discharge, 1 month later, the patient could walk unaided, but hyperreflexia was still present in her legs; the Babinski signs had disappeared.

This patient had ATM with involvement of sensory and motor tracts on both sides of the spinal cord. This neurological damage was probably a complication of infection with *B burgdorferi*: it developed soon after the appearance of the pathognomonic rash associated with this tick-born spirochaete; specific serological tests for *B burgdorferi* in the acute phase of illness were strongly positive; after penicillin therapy antibody titres fell slightly while the patient improved clinically; and no evidence for other viral, bacterial, or fungal causes of ATM was found.

The diagnosis of MS, another cause of ATM, might have been wrongly considered if the skin lesion had not been recognised, prompting the search for *B burgdorferi* antibodies. CSF analysis was confusing since both MS[2] and neurological complications of *B burgdorferi*[3] have the same CSF biochemical pattern with oligoclonal bands in the very alkaline region on isofocusing.

In contrast to the presentation in this case, myelitis, when noted in Lyme disease,[4] has seldom been the prominent or sole neurological manifestation. Myelitis has been an infrequent sign, associated with meningoencephalitis and cranial or peripheral radiculopathy, running a progressive rather than acute course.[5]

We suggest that *B burgdorferi* be considered in future as a potential (and curable) cause of ATM.

Departments of Neurology and Rheumatology, Centre Hospitalier de Sainte-Ode, 6970 Tenneville, Belgium — J. JACQUES ROUSSEAU, CATHERINE LUST, PIERRE F. ZANGERLE

Serology Laboratory, Cliniques Universitaires Saint Luc, UCL, Brussels — GEOFFROY BIGAIGNON

1. Pachner AR, Steere AC. The triad of neurological manifestations of Lyme disease: Meningitis, cranial neuritis and radiculo-neuritis. *Neurology* 1985; **35**; 47–53.
2. Delmotte P, Gonsette R. Biochemical findings in multiple sclerosis IV: Isoelectric focusing of the CSF gamma globulins in multiple sclerosis (262 cases) and other neurological diseases (272 cases). *J Neurol* 1977; **215**: 27–37.
3. Ryberg B, Hindfelt B, Nilsson B, Olsson JE. Antineural antibodies in Guillan-Barré syndrome and lymphocytic meningoradiculitis (Bannwarth's syndrome). *Arch Neurol* 1984; **41**: 1277–81.
4. Reik L, Steere AC, Bartenhagen NH, Shope RE, Malawista SE. Neurologic abnormalities of Lyme disease. *Medicine* 1979; **58**: 281–94.
5. Ackermann R, Gollmer E, Rehse-Küpper B. Progressive Borrelian-enzephalomyelitis. *Deutsch Med Wochenschr* 1985; **110**: 1039–42.

SPIROCHAETES AND LYME DISEASE

SIR,—Muhlemann and colleagues[1] found low titre antibodies to *Borrelia burgdorferi* antibodies in only 1 of 15 multiple sclerosis (MS) patients. Fumarola[2] recorded a low titre (1/64) in another case, rising during a relapse to 1/256. As outlined by Gay and Dick,[3] a central nervous system infection by *B burgdorferi* may cause an MS-like syndrome.

A 22-year-old man had a 5-month history of progressive spastic paraparesis and cerebellar syndrome. Computerised tomography (CT) and magnetic resonance imaging (MRI) were normal; visual evoked potentials were normal, brainstem evoked potentials showed mild abnormalities. The CSF contained 92 lymphocytes/μl, protein 2·55 mg/dl, glucose 0·22 mg/dl, and oligoclonal banding. Laboratory investigations revealed no evidence of bacterial, viral, fungal, parasitic, or immune-system disease. The patient had antibodies to *B burgdorferi* (indirect immunofluorescence assay) in blood (1/4000) and CSF (1/1024); syphilitic serology was negative. The patient was treated with penicillin G 20 megaunits daily for 10 days and then latamoxef 3 g daily for 3 months, and prednisone 50 mg per day for 2 months. The meningitis and cerebellar signs disappeared, the paraparesis improved partly, and the antibody titres decreased.

MS-like manifestations (chronic[4-6] or even remitting-relapsing forms[7]) of central nervous system involvement by *B burgdorferi* infection have been reported, with moderate CSF pleiocytosis and oligoclonal banding. CT scans and MRI can suggest demyelination.[6,7] In our case the clinical presentation suggested a progressive form of MS but the pleiocytosis, raised CSF albumin, and low CSF sugar made this diagnosis unlikely. The diagnosis of borrelia infection usually rests on the detection in blood (or even in CSF alone) of antibodies to *B burgdorferi*.

In the cases of Muhlemann et al[1] and Fumarola[2] the CSF antibody titres were unknown.

Department of Neurology, Lille University Hospital, 59037 Lille, France — P. BENOIT

Central Laboratory, Claude Bernard Hospital, Paris — E. DOURNON

Department of Neurology, Lille University Hospital — A. DESTEE, P. WAROT

1. Muhlemann MF, Markby DP, Wright DJM. Multiple sclerosis associated with sinusitis. *Lancet* 1986; i: 1097.
2. Fumarola D. Multiple sclerosis and *Borrelia burgdorferi*. *Lancet* 1986; ii: 575
3. Gay D, Dick G. Spirochaetes, Lyme disease, and multiple sclerosis. *Lancet* 1986; ii: 685.
4. Ackermann R, Rehse-Kupper B, Gollmer E. Progressive Borrelia-Enzephalomyelitis: Chronische Manifestation der Erythema chronicum migrans Krankheit am Nervensystem. *Dtsch Med Wchschr* 1985; **26**: 1039–42.
5. Kohler J, Kasper J, Kern V, Thoden U, Rehse-Kupper B. Borrelia encephalomyelitis. *Lancet* 1986; i: 35.
6. Hansen K, Rechnitzer C, Pedersen NS, Arpi N, Jensen O. Borrelia meningitis and demyelinating CNS disease. Paper read at 2nd International Symposium on Lyme Disease and Related Disorders (Vienna, 1985): abstr 45.
7. Pachner AR, Steere AC. Tertiary Lyme disease: Central nervous system manifestations of longstanding infections with *Borrelia burgdorferi* In: Proceedings of 2nd International Symposium on Lyme Disease and Related Disorders (Vienna, 1985). (In press.)

^{99m}Tc-HEXAMETHYLPROPYLENEAMINE OXIME SCANS TO CONFIRM BRAIN DEATH

SIR,—Brain death is usually easy to diagnose on clinical evidence, when a known factor causing intracranial pressure is present, but brain death may be secondary to disease outside the central nervous system, and diseases such as encephalitis or drug intoxication can also confuse the picture. An aortocervical or carotid angiogram will confirm the termination of cerebral circulation[1] but angiography is expensive and time-consuming; a non-invasive method for confirming cessation of brain circulation would be more desirable.[2]

The lipophilic brain perfusion tracer ^{99m}Tc-labelled hexamethylpropyleneamine oxime (HMPAO) is a reliable indicator of brain blood flow, and is available on an everyday basis.[3] This makes the compound suitable for the non-invasive study of brain circulation when the diagnosis of brain death is uncertain.

Since December, 1985, we have investigated over 120 patients with HMPAO emission tomography (SPECT), including 13 unconscious patients. HMPAO SPECT and planar scintigraphy have demonstrated clear cerebral accumulation of the tracer in all cases (fig 1). We have now scanned a patient with suspected brain death (no brainstem responses, isoelectric EEG, no brainstem auditory evoked potentials) by HMPAO SPECT and planar scintigraphy. Both SPECT and planar scintigrams revealed the lack of cerebral circulation (fig 2), confirming brain death.

Our experience suggests, that HMPAO may be used in confirming the termination of cerebral circulation. Since both HMPAO SPECT and planar scintigraphy revealed the lack of