anaesthesia we report might reflect the innervation of the breast, with infiltration being lateral to the breast lump and not into the lump itself, as well as the period of time allowed after infiltration before FNA was undertaken. Local anaesthesia has no adverse effect on diagnostic accuracy,[4] and because the infiltration is lateral to the lump, palpation of the lump during aspiration is not obscured. We believe that the reduction of pain by local anaesthesia helps the tolerance of patients to the procedure and gives the operator a better chance of obtaining an adequate sample for diagnosis.

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Sir—Sutini Ngadiman and colleagues (Nov 18, p 1701)1 recommend use of a small needle (25G) for fine-needle aspiration cytology of the breast. By all means use small needles but, for holistic patient-centred care, always offer local anaesthesia.

I asked for pain relief before aspiration but was told I would "just have to put up with it". There was only one entry point for the needle, but it moved around in short stabs 20–30 times. The only feature of potential local recurrence that plays on my mind is the thought of having to endure fine-needle aspiration again.

A colleague underwent mechanical biopsy while her breast was clamped to a machine. No pain relief was offered and her screams were ignored.

"First do no harm?" Please put humanity into cancer care.

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Holmes-Adie syndrome and Lyme disease

Sir—Paolo Martinelli (Nov 18, p 1760)[1] provides an excellent clinical and historical overview of Holmes-Adie syndrome (tonic pupil and areflexia), more commonly known in the USA as Adie syndrome. Although the syndrome has not been associated with "infection of conventional bacterial or viral origin", it is occasionally linked to early syphilis, parvovirus B19, and herpes simplex virus infections.[2–4] We now report an association with neurological Lyme disease.

In a referral practice of about 140 patients with Lyme disease, we have seen three patients with predominant neurological symptoms who presented with Holmes-Adie syndrome (table). The diagnosis of Lyme disease was based on a history of tickbite, presence of an erythema migrans rash and positive serology for the spirochaete *Borrelia burgdorferi*. The duration of Lyme disease symptoms ranged from 2–16 years, and each patient developed a unilateral tonic pupil before the diagnosis of Lyme disease was made. All three patients had significant neuropsychiatric and cognitive defects, hyporeflexia, and facial dysaesthesia without anhydrosis. Rapid plasma reagin testing was negative in each case. Two patients had abnormal brain magnetic resonance imaging with white-matter lesions consistent with neurological Lyme disease. A decreased concentration of CD57 lymphocytes characteristic of chronic Lyme disease[5] was found in two patients before antibiotic therapy. Of note, Holmes-Adie syndrome persisted in each case despite intravenous antibiotic therapy and partial resolution of other neurological symptoms of Lyme disease.

Neurological Lyme disease has been associated with various cranial nerve, meningeal, and neuropsychiatric abnormalities.[5] Based on our clinical observation, it seems that Holmes-Adie syndrome might represent an early and unrecognised manifestation of Lyme disease. We suggest that patients presenting with this unusual neuro-ophthalmological syndrome should be tested for the Lyme disease spirochaete.

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Patient	Age/sex	Duration of Lyme disease (years)	Topic pupil	CD57 (cells/μL)	Brain MRI	Antibiotic treatment
1	39 F	16	Unilateral	49*	Negative	IV
2	42 F	8	Unilateral	31*	Positive	IV
3	39 F	2	Unilateral	111†	Positive	IV

MRI=magnetic resonance imaging; IV=intravenous ceftriaxone. *Before antibiotics. †On antibiotics. Normal CD57 range 60–360 cells/μL.

Characteristics of patients with Lyme disease and Holmes-Adie syndrome

Eyelid movement disorders and electromyography

Sir—Mikio Hirayama and colleagues (Oct 21, p 1413)[1] report improvement of apraxia of eyelid opening in two patients by wearing goggles.

They used F Lepore and R Duvoisin's criteria[2] to diagnose eyelid apraxia of a transitory inability to open the eyelid with no evidence of continuing orbicularis oculi activity, such as lowering of the brows beneath the superior orbital margins (Charcot's eyebrow sign of blepharospasm). Yet, in our opinion, a diagnosis is almost impossible to make on the basis of the clinical picture alone, and exact diagnosis requires electromyography.

We have reported three patients with eyelid opening disorders.[3] The first patient's symptoms were similar to those of the first patient of Hirayama and colleagues. We recorded electromyographic activity from the levator palpebrae and the orbicularis oculi muscles simultaneously. On the command to open the eyelids, our patients seemed unable to inhibit the discharges in the orbicularis oculi muscle (figure). This