EDITORIAL COMMENTARIES

Sural nerve biopsy

Sural nerve biopsy has been a well established diagnostic procedure for the investigation of peripheral neuropathies for over 30 years and the techniques and indications were described by Dyck and Loufgren at the Mayo Clinic¹ and Thomas.² Although indications and guidelines for sural nerve biopsy have been described³ and retrospective studies of its value have been published, the first prospective study of the procedure to determine its usefulness in influencing diagnosis and treatment and the complications is reported in the paper by Gabriel *et al*⁴ in this issue (pp 442-446).

Full laboratory investigations and neurophysiological studies should be undertaken before biopsy is considered and it is of primary importance that the surgeon is experienced in the procedure and that the tissue can be evaluated by a laboratory experienced in the techniques of light and electron microscopy, teased fibres studies, and the use of immunohistochemical methods of staining. Certain conditions have characteristic histopathological appearances including most cases of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), CMT1, amyloid neuropathy, vasculitis, sarcoidosis, giant axonal neuropathy, hexacarbon neuropathy, IgM kappa paraproteinemic neuropathy, metachromatic leukodystrophy, and Krabbe's and Fabry's diseases but in many conditions the appearance of axonal degeneration or mixed axonal degeneration and demyelination is non-specific and nerve biopsy assists the diagnosis only by exclusion.

Sural nerve biopsy has complications of pain, infection, sensory loss, and delayed wound healing and should only be undertaken in cases of peripheral neuropathy where there are good prospects of its significantly assisting in the

diagnosis, as was the case in the study of Gabriel *et al*,⁴ which examined the value of biopsy in 50 consecutive patients. In their series biopsy was performed only when it might have disclosed a treatable cause and therefore many hereditary neuropathies with characteristic pathology would have been excluded. In seven cases the prebiopsy diagnosis was altered by the biopsy and in 60% of cases an independent neurologist judged that it had been helpful, particularly in mononeuritis multiplex and demyelinating neuropathies.

It is clear that sural nerve biopsy has an important place in the diagnosis of peripheral nerve disease and, in the case of vasculitic neuropathy confined to peripheral nerves, it is the only certain way of making the diagnosis of the treatable condition. The value of biopsy needs to be weighed against the complications of persistent pain at the biopsy site (33%), infection (15%), and patient dissatisfaction with the procedure (27%).

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- 1 Dyck PJ, Lofgren NP. Methods of fascicular biopsy of human peripheral nerve for electrophysiologic and histologic study. *Mayo Clin Proc* 1966;41:778.
- 2 Thomas PK. The quantitation of nerve biopsy findings. *J Neurol Sci* 1971; 11:285-95.
- 3 Thomas PK. Biopsy of peripheral nerve tissue. In: Asbury AK, Thomas PR, eds. *Peripheral Nerve Disorders*, 2nd ed. 1998:281-300.
- 4 Gabriel CM, Howard R, Kinsella N, *et al*. Prospective study of the usefulness of sural nerve biopsy. *J Neurol Neurosurg Psychiatry* 2000;69: 442-6.

Fear can interrupt the continuum of memory

In the letter by Harvey¹ on page 562 of this issue, a personal instance of memory loss for the events of a life threatening accident is described. In the absence of any concussion or violent acceleration/deceleration forces, Harvey¹ concludes that this was "post-traumatic amnesia in which the trauma was wholly psychological." There are at least two interesting aspects to this account—one being what was remembered, and the other being what was not recalled.

In situations of extreme stress, some memories are enhanced, detailed, and may be recalled intrusively thereafter, whereas other items are forgotten. In the present account, Harvey notes that the front seat passenger was wearing a flowery hat, that the car was a small red Honda saloon, and that a black and white soft toy was dangling in the rear window, but he has forgotten what happened next. Fragments of vivid memories such as these seem to be common in life-threatening trauma, and they become intrusive in post-traumatic stress disorder. Similarly, in head injury, Russell and Nathan² referred to memories with the quality of "visions" arising from a brief "lucid interval"

before the onset of post-traumatic amnesia—for example, for the screech of brakes or being struck by a car. In head injury, amnesia predominates and such vivid memories are infrequent, whereas in post-traumatic stress disorder intrusive thoughts predominate and memory lapses are less common: nevertheless, the two seem to lie at the extremes of a continuum, and what is still poorly understood in both instances is why certain things are vividly remembered, whereas others are forgotten.

Memory loss after trauma is well described in the psychiatric and clinical psychology literature. It can be global, as in fugue states, or situation specific for a particular traumatic incident.³ If situation specific, the amnesia can be complete or partial ("fragmentary"). Fugue states are always precipitated by situations of severe stress, such as occur during marital or relationship breakdown, severe financial troubles, wartime, or being charged with an offence. They are very commonly associated with severe depression, and there is commonly a history of a transient, organic amnesia, which may have acted as a kind of "learn-

ing experience". Situation specific amnesia occurs in so called "crimes of passion", where the offence takes place in a state of extreme emotional arousal, is unpremeditated, and where the victim is (almost invariably) a lover, wife, or partner. Although it can be argued that such amnesia may be legally motivated, memory lapses are also reported in the victims and eye witnesses of offences.⁴⁻⁵ Amnesic gaps have also been reported in traumatised soldiers in the two world wars⁶ and subsequently.⁷ A recent review⁸ documents evidence of amnesia in the victims of lightning flashes, flood disasters, pipeline explosions, earthquake, concentration camp and holocaust survivors, and Bosnian refugees: these authors report amnesia or "memory disturbance" in 16 studies. Others have also cited cases of kidnap and torture,⁷ and I would add victims of the Herald of Free Enterprise disaster. In addition, Brown *et al*⁶ found some evidence of forgetting in all 68 studies that they reviewed on the fraught issue of memory for child sexual abuse. Although there are problems in evaluating self reports of amnesia for child abuse, some smaller scale studies have attempted to examine the evidence for both the trauma and the subsequent forgetting in some detail.⁹ Curiously unmentioned in this list are road traffic accidents, presumably because any memory loss is generally assumed to be organic: nevertheless, attempts to examine the interaction of neurological and emotional effects on memory after head injury are beginning.¹⁰

Many of the situations cited involved fear or threat to life. It has been claimed that such situations involve a narrowing of consciousness with attention focused on central perceptual details, sometimes evolving into amnesia⁵ or that emotional or traumatic events are processed differently from "ordinary" memories.⁷ In particular, emotional

memories may implicate amygdaloid circuits.¹¹⁻¹² It may also be the case that, when something extraordinary happens, we ask ourselves to recall far more detail than we would normally expect. The mechanisms involved are not well researched, but Harvey's¹ account will be invaluable if it restores attention to the putative contribution of emotion in at least some cases of accident or trauma.

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- 1 Harvey P. Fear can interrupt the continuum of memory. *J Neurol Neurosurg Psychiatry* 2000;**69**:562.
- 2 Russell WR, Nathan PW. Traumatic amnesia. *Brain* 1946;**69**:280-300.
- 3 Kopelman MD. The assessment of psychogenic amnesia. In: Baddeley A, Wilson B, Watts F, eds. *Handbook of memory disorders*. Chichester: John Wiley, 1995.
- 4 Loftus EF. *Eyewitness testimony*. Cambridge, MA: Harvard University Press, 1979.
- 5 Christianson S-A. The relationship between induced emotional arousal and amnesia. *Scand J Psychol* 1984;**25**:147-60.
- 6 Sargant W, Slater, E. Amnesic syndromes in war. *Proceedings of the Royal Society of Medicine* 1941;**34**:757-64.
- 7 van der Kolk BA, Finkelhor R. Dissociation and the fragmentary nature of traumatic memories: overview and exploratory study. *J Trauma Stress* 1995;**8**:505-25.
- 8 Brown D, Schefflin AW, Whitfield CL. Recovered memories: the current weight of the evidence in science and in the courts. *Journal of Psychiatry and Law* 1999;**27**:5-156.
- 9 Schooler JW, Bendixen J, Ambadar Z. Taking the middle line: can we accommodate both fabricated and recovered memories of sexual abuse? In: Conway MA, ed. *Recovered memories and false memories*. New York: Oxford University Press, 1997.
- 10 Williams WH, Williams JMG, Ghadiali EJ. Autobiographical memory in traumatic brain injury: neuropsychological and mood predictors of recall. *Neuropsychological Rehabilitation* 1998;**8**:43-60.
- 11 Rogan MT, Staebli UV, LeDoux JE. Fear conditioning induces associative long-term potentiation in the amygdala. *Nature* 1997;**390**:604-7.
- 12 LeDoux JE, Muller J. Emotional memory and psychopathology. *Philos Trans R Soc Lond B Biol Sci* 1997;**352**:1719-26.