

ture, and some dental equipment including the ultrasonic cleaner. Investigations into the effect of therapeutic radiation on pacemaker function are conflicting,² and the long-term effect of radiation on electronic components is unknown, but, provided the pacemaker is protected, radiation may safely be given to patients with pacemakers.

The patient faced with this daunting catalogue of potential environmental dangers might well feel insecure. He may be reassured that, though the list is long, the actual danger is, to use Sowton's term, minimal.² Nevertheless, patients with pacemakers should be aware of the more likely sources of interference so that they can learn to avoid them; manufacturers of electrical equipment should recognise the problem and provide warning notices if electrical interference is likely, and pacemaker manufacturers will, no doubt, continue to develop means of filtering and rejecting interference signals.

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¹ Frank G, Tyers O, Brownlee RR. Power pulse generators, electrodes, and longevity. *Prog Cardiovasc Dis* 1981;23:421-34.

² Sowton E. Environmental hazards for pacemaker patients. *J R Coll Physicians Lond* 1982;16:159-64.

³ Furman S. Spurious pacemaker programming. *Pace* 1980;3:517-8.

Evoked potentials in neurological diagnosis

The functional integrity of specific cortical areas and of pathways in the central nervous system can be assessed by recording the electrical potentials associated with specific sensory or motor events. The procedure uses electronic averaging techniques recording from surface electrodes placed over certain areas of the brain or spinal cord. These methods provide a non-invasive objective test which may disclose abnormalities not apparent on clinical examination.

Evoked potentials have now established their place in the diagnostic assessment of patients with several neurological disorders, and their contribution in other disciplines is continuing to be evaluated.¹ Visual, auditory, and somatosensory evoked potentials have been most widely used diagnostically, while olfactory, cognitive, and movement-associated potentials have yet to find routine application.

The main use of sensory evoked potentials has been in the diagnosis of multiple sclerosis, in which they have proved useful for confirming clinically suspected lesions of the visual, auditory, and somatosensory pathways and—more important—for detecting subclinical lesions. In so doing they may reduce the need for more invasive procedures such as myelography or angiography in some cases.² Most useful has been the pattern reversal visual evoked potential, which is abnormal in 80-90% of patients with a firm clinical diagnosis of multiple sclerosis with or without previous visual symptoms,^{3,4} and in 30-50% of patients with suspected or probable multiple sclerosis.^{5,6} The characteristic finding is an increase in the latency of the major positive component of the visual evoked potential, which only rarely returns to normal. Small but indistinguishable latency changes may, however, also occur in patients with refractive errors⁷ or other ocular abnormalities,⁸ in pernicious anaemia,⁹ in hereditary ataxias,^{10,11} in Charcot-

Marie-Tooth disease,¹² and in other forms of optic neuropathy.¹³

Auditory brain stem and somatosensory evoked potentials have been less useful than the visual evoked potential, but the combined use of the three techniques provides the highest yield of subclinical abnormalities in patients suspected of suffering from multiple sclerosis.^{14,15} Abnormalities of the auditory brain stem potentials have been found in up to 80% of patients with definite multiple sclerosis¹⁵ with a previous history or clinical signs of lesions in the brain stem, and in up to 50% of patients without such signs.^{16,17} A few patients with isolated optic neuritis are found to have abnormal auditory brain stem potentials pointing to an additional lesion in the brain stem and thereby increasing the likelihood of multiple sclerosis (C Storey, "Role of evoked potentials in the investigation of optic neuritis"; presented at neuro-ophthalmology symposium, Melbourne, November 1981). Abnormalities of latency have proved the most useful measures, especially comparisons of interpeak latency between the two sides. Nevertheless, changes in latency in auditory brain stem potential components are less definite than in the case of the visual evoked potential and the abnormalities are in general more labile; serial studies in patients with multiple sclerosis have shown bidirectional variations.¹⁸

Abnormal sensory evoked potentials recorded over the scalp or cervical spine, reflecting lesions of the dorsal column/lemniscal sensory pathway, have been found in over 75% of patients with clinically definite multiple sclerosis and in a third to a half of patients with probable or suspected multiple sclerosis, including some without sensory symptoms or signs.^{14,19,20} The combined use of upper and lower limb stimulation and the calculation of conduction times in the spinal and central portions of the somatosensory pathway has increased the rate of detection of lesions and has helped in locating them.²¹

Abnormalities of evoked potentials may also occur in disorders other than demyelinating diseases, though they are less often of diagnostic value.^{22,23} Of particular importance is the finding of normal evoked potentials in patients with hysterical sensory deficits or malingering. Abnormalities of the visual evoked potential may be found in patients with compressive lesions of the optic nerve or chiasm such as pituitary tumours or meningioma, and the site of compression may be defined more precisely by the use of multichannel recordings and hemifield stimulation.^{8,23} Characteristic patterns of abnormality are also found in patients with toxic²⁴ or hereditary forms of bilateral optic neuropathy.²⁵ Auditory brain stem potentials have an important application in assessing auditory acuity in infants or in retarded or uncooperative patients or those with psychogenic hearing loss.²⁶ They have also proved useful in the diagnosis of acoustic neuroma, abnormal responses being found in most cases,²⁷ and they may also provide objective evidence of damage to the lower auditory pathway in patients with ischaemia or a tumour of the brain stem and in the postconcussional syndrome.²⁸⁻³⁰ They have been used in evaluating brain stem function in coma and suspected brain death³¹ but have yet to find general application in these circumstances. Sensory evoked potentials may have a part to play in assessing patients with lesions of the brachial plexus or spinal roots³² and in determining the extent of traumatic spinal cord lesions. Evoked potential techniques have also been used to evaluate the severity of cerebral dysfunction after head injury^{33,34} and in patients with metabolic encephalopathies^{35,36} and coma.³⁷

Evoked potentials, therefore, provide a means of objective

assessment of central nervous system function and yield information which may help in diagnosis and in the management of patients with a variety of neurological disorders. The sensitivity of the techniques is likely to increase with further developments in instrumentation and methods for analysis of the response, with the development of new methods for assessing the temporal properties of conduction in specific pathways, and with improved definition of control groups.³⁸ The analysis of late cortical components in patients with dementia and cognitive disturbances³⁹ and recording of potentials associated with limb movement⁴⁰⁻⁴¹ are promising developments with the possibility for clinical application and warrant continuing investigation.

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- ¹ Chiappa KH, Ropper AH. Evoked potentials in clinical medicine. *N Engl J Med* 1982;**306**:1140-50.
- ² Mastaglia FL, Black JL, Cala LA, Collins DWK. Electrophysiology and avoidance of invasive neuroradiology in multiple sclerosis. *Lancet* 1980;**i**:144.
- ³ Halliday AM, McDonald WI, Mushin J. Visual evoked response in diagnosis of multiple sclerosis. *Br Med J* 1973;**iv**:661-4.
- ⁴ Asselman P, Chadwick DW, Marsden CD. Visual evoked responses in the diagnosis and management of patients suspected of multiple sclerosis. *Brain* 1975;**98**:261-82.
- ⁵ Mastaglia FL, Black JL, Collins DWK. Visual and spinal evoked potentials in diagnosis of multiple sclerosis. *Br Med J* 1976;**ii**:732.
- ⁶ Shahrokhi F, Chiappa KH, Young RR. Pattern shift visual evoked responses. Two hundred patients with optic neuritis and/or multiple sclerosis. *Arch Neurol* 1978;**35**:65-71.
- ⁷ Collins DWK, Carroll WM, Black JL, Walsh M. Effect of refractive error on the visual evoked response. *Br Med J* 1979;**i**:231-2.
- ⁸ Carroll WM, Halliday AM, Kriss A. Improvements in the accuracy of pattern visual evoked potentials in the diagnosis of visual pathway disease. *Neuro-ophthalmology* 1982;**2**:237-53.
- ⁹ Troncoso J, Mancall EL, Schatz NJ. Visual evoked responses in pernicious anemia. *Arch Neurol* 1979;**36**:168-9.
- ¹⁰ Carroll WM, Kriss A, Baraitser M, Barrett J, Halliday AM. The incidence and nature of visual pathway involvement in Friedreich's ataxia: a clinical and visual evoked potential study of 22 patients. *Brain* 1980;**103**:413-34.
- ¹¹ Livingstone IR, Mastaglia FL, Edis R, Howe JW. Visual involvement in Friedreich's ataxia and hereditary spastic ataxia: a clinical and visual evoked response study. *Arch Neurol* 1981;**38**:75-9.
- ¹² Bird TD, Griep E. Pattern reversal visual evoked potentials—studies in Charcot-Marie-Tooth hereditary neuropathy. *Arch Neurol* 1981;**38**:739-41.
- ¹³ Carroll WM, Mastaglia FL. Leber's optic neuropathy: a clinical and visual evoked potential study of affected and asymptomatic members of a six generation family. *Brain* 1979;**102**:559-80.
- ¹⁴ Mastaglia FL, Black JL, Cala LA, Collins DWK. Evoked potentials, saccadic velocities, and computerised tomography in diagnosis of multiple sclerosis. *Br Med J* 1977;**ii**:1315-7.
- ¹⁵ Khoshbin S, Hallett M. Multimodality evoked potentials and blink reflex in multiple sclerosis. *Neurology (Minneapolis)* 1981;**31**:138-44.
- ¹⁶ Robinson K, Rudge P. Abnormalities of the auditory evoked potentials in patients with multiple sclerosis. *Brain* 1977;**100**, pt 1:19-40.
- ¹⁷ Chiappa KH, Harrison JL, Brooks EB, Young RR. Brainstem auditory evoked responses in 200 patients with multiple sclerosis. *Ann Neurol* 1980;**7**:135-43.
- ¹⁸ Robinson K, Rudge P. The use of the auditory evoked potential in the diagnosis of multiple sclerosis. *J Neurol Sci* 1980;**45**:235-44.
- ¹⁹ Small DG, Matthews WB, Small M. The cervical somatosensory evoked potential (SEP) in the diagnosis of multiple sclerosis. *J Neurol Sci* 1978;**35**:211-24.
- ²⁰ Eisen A, Stewart J, Nudleman K, Cosgrove JB. Short-latency somatosensory responses in multiple sclerosis. *Neurology (Minneapolis)* 1979;**29**:827-34.
- ²¹ Eisen A, Odusote K. Central and peripheral conduction times in multiple sclerosis. *Electroencephalogr Clin Neurophysiol* 1980;**48**:253-65.
- ²² Chiappa KH. Brainstem auditory evoked potentials. In: Stålberg E, Young RR, eds. *Clinical neurophysiology*. Vol 1. London: Butterworths, 1981:259-77.
- ²³ Halliday AM, McDonald WI. Visual evoked potentials. In: Stålberg E, Young RR, eds. *Clinical neurophysiology*. Vol 1. London: Butterworths, 1981:228-58.
- ²⁴ Kriss A, Carroll WM, Blumhardt LD, Halliday AM. Pattern- and flash-evoked potential changes in toxic (nutritional) optic neuropathy. In: Courjon J, Mauguire F, Revol M, eds. *Clinical applications of evoked potentials in neurology*. New York: Raven Press, 1982:11-9. (*Advances in neurology*. Vol 32.)
- ²⁵ Harding GFA, Crews SJ, Good PA. VEP in neuro-ophthalmic disease. In: Barber C, ed. *Evoked potentials*. Lancaster: MTP Press, 1980:235-42.
- ²⁶ Davis H. Principles of electric response audiometry. *Ann Otol Rhinol Laryngol* 1976;**85**, suppl 28:1-92.
- ²⁷ Parker SW, Chiappa KH, Brooks EB. Brainstem auditory evoked responses (BAERs) in patients with acoustic neuromas and cerebellar-pontine angle (CPA) meningiomas. *Neurology (Minneapolis)* 1980;**30**:413-4. (Abstract.)
- ²⁸ Starr A, Achor J. Auditory brain stem responses in neurological disease. *Arch Neurol* 1975;**32**:761-8.
- ²⁹ Stockard JJ, Rossiter VS. Clinical and pathologic correlates of brain stem auditory response abnormalities. *Neurology (Minneapolis)* 1977;**27**:316-25.
- ³⁰ Noseworthy JH, Miller J, Murray TJ, Regan D. Auditory brainstem responses in postconcussion syndrome. *Arch Neurol* 1981;**38**:275-8.
- ³¹ Starr A. Auditory brain-stem responses in brain death. *Brain* 1976;**99**:543-54.
- ³² Jones SJ. Investigation of brachial plexus traction lesions by peripheral and spinal somatosensory evoked potentials. *J Neurol Neurosurg Psychiatry* 1979;**42**:107-16.
- ³³ Hume AL, Cant BR. Central somatosensory conduction after head injury. *Ann Neurol* 1981;**10**:411-9.
- ³⁴ Lindsay KW, Carlin J, Kennedy I, Fry J, McInnes A, Teasdale GM. Evoked potentials in severe head injury—analysis and relation to outcome. *J Neurol Neurosurg Psychiatry* 1981;**44**:796-802.
- ³⁵ Mastaglia FL, Black JL, Collins DWK, Gutteridge GH, Yuen RWM. Slowing of conduction in visual pathway in hypothyroidism. *Lancet* 1978;**i**:387-8.
- ³⁶ Lowitzsch K, Göhring U, Hecking E, Köhler H. Refractory period, sensory conduction velocity and visual evoked potentials before and after haemodialysis. *J Neurol Neurosurg Psychiatry* 1981;**44**:121-8.
- ³⁷ Hume AL, Cant BR, Shaw NA. Central somatosensory conduction time in comatose patients. *Ann Neurol* 1979;**5**:379-84.
- ³⁸ Halliday AM, Barrett G, Carroll WM, Kriss A. Problems in defining the normal limits of the visual evoked potential. In: Courjon J, Mauguire F, Revol M, eds. *Clinical applications of evoked potentials in neurology*. New York: Raven Press, 1982:1-9. (*Advances in neurology*. Vol 32.)
- ³⁹ Goodin DS, Squires KC, Starr A. Long latency event-related components of the auditory evoked potential in dementia. *Brain* 1978;**101**:635-48.
- ⁴⁰ Deecke L, Grözinger B, Kornhuber HH. Voluntary finger movement in man: cerebral potentials and theory. *Biol Cybern* 1976;**23**:99-119.
- ⁴¹ Shibasaki H, Barrett G, Halliday E, Halliday AM. Components of the movement-related cortical potential and their scalp topography. *Electroencephalogr Clin Neurophysiol* 1980;**49**:213-26.

BCG vaccination scars: an avoidable problem?

Injections and vaccinations are most often given into the outer aspect of the upper arm—on the grounds of safety and ease of access. The use of this site may result in the formation of hypertrophic or keloid scars¹ sufficiently unsightly for patients to seek surgery. Smallpox vaccination was the most common cause of these defects, but now that it is obsolete, BCG (bacille Calmette Guérin) immunisation is left as the principal offender. The incidence of hypertrophic or keloid scars resulting from BCG immunisation in Britain is not known, but elsewhere in the world the incidence of hypertrophic scars has been put at 28-33% and of keloid scars from 2% to 4%.^{2,3}

Several factors influence the risk of scar formation and the final appearance.⁴ The skin in some areas of the body has a tendency to form hypertrophic scars—for example, the skin on the deltoid, the sternum, and the upper back.⁵ Any infection, especially if chronic, prolongs inflammation and increases the risk of a bad scar. Pigmented skins are also more liable to scar hypertrophy.

BCG inoculation results in the formation of a cell-mediated immune response to the bacterium. The vaccine is given intracutaneously (subcutaneous administration results in a cold abscess), and after three weeks a bluish red papule appears at