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.<sup>38</sup> The analysis of late cortical components in patients with dementia and cognitive disturbances<sup>39</sup> and recording of potentials associated with limb movement<sup>40 41</sup> are promising developments with the possibility for clinical application and warrant continuing investigation.

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- <sup>1</sup> Chiappa KH, Ropper AH. Evoked potentials in clinical medicine. N Engl J Med 1982;306:1140-50.
- <sup>2</sup> Mastaglia FL, Black JL, Cala LA, Collins DWK. Electrophysiology and avoidance of invasive neuroradiology in multiple sclerosis. *Lancet* 1980; i:144.
- <sup>3</sup> Halliday AM, McDonald WI, Mushin J. Visual evoked response in diagnosis of multiple sclerosis. Br Med J 1973;iv:661-4.
- <sup>1</sup> 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.
- <sup>5</sup> Mastaglia FL, Black JL, Collins DWK. Visual and spinal evoked potentials in diagnosis of multiple sclerosis. Br Med J 1976;ii:732.
- <sup>6</sup> 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.
- <sup>7</sup> Collins DWK, Carroll WM, Black JL, Walsh M. Effect of refractive error on the visual evoked response. Br Med J 1979;i:231-2.
- \* 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.
- <sup>9</sup> Tronosco J, Mancall EL, Schatz NJ. Visual evoked responses in pernicious anemia. Arch Neurol 1979;36:168-9.
- <sup>10</sup> 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.
- <sup>11</sup> 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.
- <sup>12</sup> Bird TD, Griep E. Pattern reversal visual evoked potentials—studies in Charcot-Marie-Tooth hereditary neuropathy. Arch Neurol 1981;38: 739-41.
- <sup>13</sup> 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.
- <sup>14</sup> Mastaglia FL, Black JL, Cala LA, Collins DWK. Evoked potentials, saccadic velocities, and computerised tomography in diagnosis of multiple sclerosis. Br Med J 1977;i:1315-7.
- <sup>15</sup> Khoshbin S, Hallett M. Multimodality evoked potentials and blink reflex in multiple sclerosis. *Neurology (Minneap)* 1981;31:138-44.
   <sup>16</sup> Robinson K, Rudge P. Abnormalities of the auditory evoked potentials in
- <sup>16</sup> Robinson K, Rudge P. Abnormalities of the auditory evoked potentials in patients with multiple sclerosis. *Brain* 1977;100, pt 1:19-40.
  <sup>17</sup> Chiappa KH, Harrison JL, Brooks EB, Young RR. Brainstem auditory
- <sup>17</sup> Chiappa KH, Harrison JL, Brooks EB, Young RR. Brainstem auditory evoked responses in 200 patients with multiple sclerosis. *Ann Neurol* 1980;**7**:135-43.
- <sup>18</sup> Robinson K, Rudge P. The use of the auditory evoked potential in the diagnosis of multiple sclerosis. J Neurol Sci 1980;45:235-44.
- <sup>19</sup> 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.
- <sup>20</sup> Eisen A, Stewart J, Nudleman K, Cosgrove JB. Short-latency somatosensory responses in multiple sclerosis. *Neurology (Minneap)* 1979;29: 827-34.
- <sup>21</sup> Eisen A, Odusote K. Central and peripheral conduction times in multiple sclerosis. *Electroencephalogr Clin Neurophysiol* 1980;**48**:253-65.
- <sup>22</sup> Chiappa KH. Brainstem auditory evoked potentials. In: Stalberg E, Young RR, eds. *Clinical neurophysiology*. Vol 1. London: Butterworths, 1981: 259-77.
- <sup>23</sup> Halliday AM, McDonald WI. Visual evoked potentials. In: Stålberg E, Young RR, eds. *Clinical neurophysiology*. Vol 1. London: Butterworths, 1981:228-58.

- <sup>21</sup> Kriss A, Carroll WM, Blumhardt LD, Halliday AM. Pattern- and flashevoked potential changes in toxic (nutritional) optic neuropathy. In: Courjon J, Mauguiere F, Revol M, eds. *Clinical applications of evoked potentials in neurology*. New York: Raven Press, 1982:11-9. (Advances in neurology. Vol 32.)
- <sup>25</sup> Harding GFA, Crews SJ, Good PA. VEP in neuro-ophthalmic disease. In: Barber C, ed. *Evoked potentials*. Lancaster: MTP Press, 1980:235-42.
- <sup>26</sup> Davis H. Principles of electric response audiometry. Ann Otol Rhinol Laryngol 1976;85, suppl 28:1-92.
- <sup>27</sup> Parker SW, Chiappa KH, Brooks EB. Brainstem auditory evoked responses (BAERs) in patients with acoustic neuromas and cerebellar-pontine angle (CPA) meningiomas. *Neurology* (*Minneap*) 1980;**30**:413-4. (Abstract.)
- <sup>28</sup> Starr A, Achor J. Auditory brain stem responses in neurological disease. Arch Neurol 1975;**32**:761-8.
- <sup>29</sup> Stockard JJ, Rossiter VS. Clinical and pathologic correlates of brain stem auditory response abnormalities. *Neurology (Minneap)* 1977;27:316-25.
- <sup>30</sup> Noseworthy JH, Miller J, Murray TJ, Regan D. Auditory brainstem responses in postconcussion syndrome. Arch Neurol 1981;38:275-8.
- <sup>31</sup> Starr A. Auditory brain-stem responses in brain death. Brain 1976;99: 543-54.
- <sup>32</sup> Jones SJ. Investigation of brachial plexus traction lesions by peripheral and spinal somatosensory evoked potentials. *J Neurol Neurosurg Psychiatry* 1979;**42**:107-16.
- <sup>33</sup> Hume AL, Cant BR. Central somatosensory conduction after head injury. Ann Neurol 1981;**10**:411-9.
- <sup>34</sup> 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.
- <sup>35</sup> Mastaglia FL, Black JL, Collins DWK, Gutteridge GH, Yuen RWM. Slowing of conduction in visual pathway in hypothyroidism. Lancet 1978;i:387-8.
- <sup>36</sup> 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.
- <sup>37</sup> Hume AL, Cant BR, Shaw NA. Central somatosensory conduction time in comatose patients. *Ann Neurol* 1979;5:379-84.
- <sup>3\*</sup> Halliday AM, Barrett G, Carroll WM, Kriss A. Problems in defining the normal limits of the visual evoked potential. In: Courjon J, Mauguière F, Revol M, eds. *Clinical applications of evoked potentials in neurology*. New York: Raven Press, 1982:1-9. (Advances in neurology. Vol 32.)
- <sup>39</sup> Goodin DS, Squires KC, Starr A. Long latency event-related components of the auditory evoked potential in dementia. *Brain* 1978;**101**:635-48.
- <sup>10</sup> Deccke L, Grözinger B, Kornhuber HH. Voluntary finger movement in man: cerebral potentials and theory. *Biol Cybern* 1976;23:99-119.
- <sup>11</sup> 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 scars<sup>1</sup> 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%.<sup>2 3</sup>

Several factors influence the risk of scar formation and the final appearance.<sup>4</sup> 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.<sup>5</sup> 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 the site of injection. This lesion reaches its maximum size at about six weeks, when the skin overlying it becomes thin and shiny and frequently ulcerates. The ulcer is typically about 5 mm diameter and usually heals by the 13th week.<sup>6</sup>

A persistent infected ulcer in an area with poor scarring properties is a good recipe for an ugly scar. One survey carried out in Africa found that the shoulder region was the most frequent site of keloid formation; out of a total of 286 keloids, vaccination (almost a fifth) was second only to unspecified trauma as a predisposing factor.<sup>7</sup>

Hypertrophic or keloid scars may be treated by pressure, surgery, radiotherapy, or steroids, either singly or in combination. The application of pressure sustained over several months using tailored elastic garments may limit or reduce the formation of hypertrophic or keloid scars.8 9 Careful excision of the scar or granulating area and subcuticular suture of the defect may effect good healing and decrease the chance of formation of a bad scar. Only too commonly, however, the incision is longer than the scar or lesion and is closed under tension with sutures placed remote from the margin of the wound. The result is a larger scar flanked by ugly suture marks. Occasionally, such an error is compounded by a further, wider excision and similar closure leading to a disastrous result. Excisional surgery is rarely effective in producing an improvement.10

The scar, especially if circular and raised, may best be treated surgically by shaving flat and covering with a thin split-skin graft taken from a donor site which must be unobtrusive, lest an ugly scar develops there. Low-dose superficial irradiation within a few days of surgery may reduce the risk of keloid formation after surgery but carries the risk in coloured skin of altered pigmentation within the field and around the scar.

Corticosteroids such as triamcinolone may be injected into the lesion either by needle or dermojet. Three or four infections at monthly intervals may cause progressive atrophy of the scar.<sup>11 12</sup> Care must be taken not to inject subdermally or resorption of fat may cause a contour defect.

Prevention is the most effective approach. No matter how carefully BCG is given, the resulting chronic infection often causes an ugly scar. Even in the hands of the most experienced surgeon the results of treatment of these scars are often disappointing. The final scar may be flatter but may still be unsightly owing to its size, abnormal colour, and texture.

When other sites are used for vaccination the problem of obtrusive scarring can be overcome-or at least minimised. Some authorities recommend a site low down the arm, at the level of, or below, the insertion of the deltoid<sup>13 14</sup>; use of this lower site reduces the incidence of hypertrophic or keloid scars.<sup>2</sup> Alternatively, the inner aspect of the arm may be used; this has better scarring properties than the outer aspect and is also more concealed. Other possible sites are the thighs, buttocks, chest, and abdominal wall. The advantages that the concealed nature of those sites offer are somewhat offset by the difficulties of access, which makes them inconvenient for use in mass immunisation. In these circumstances the arm remains the easiest target, but the upper deltoid region should not be used for BCG inoculation.

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- <sup>1</sup> Montagu MW. Quoted in Halsband R, ed. The complete letters of Lady Mary Wortley Montagu. Vol 1. Oxford: Clarendon Press, 1965:339.
- <sup>2</sup> Gonzalez O. Prevalence of hypertrophic and keloid scars according to the site of administration of intradermal BCG vaccine. Bol Of Sanit Panam 1980;88:481-8.
- <sup>3</sup> Chao CW. Post-vaccination keloid. Bull Int Union Tuberc 1972; suppl 2: 178.
- <sup>4</sup> Ketchum LD, Cohen IK, Masters FW. Hypertrophic scars and keloids, a collective review. Plast Reconstr Surg 1974;53:140-54.
- <sup>5</sup> Crockett DJ. Regional keloid susceptibility. Br J Plast Surg 1964;17: 245-53
- <sup>6</sup> Irvine KN. BCG and vole vaccination. A practical handbook. London: National Association for the Prevention of Tuberculosis, 1957.
- Oluwasanmi JO. Keloids in the African. Clin Past Surg 1974;1:179-95. Kischer CW, Shetlar MR, Shetlar CL. Alteration of hypertrophic scars
- induced by mechanical pressure. Arch Dermatol 1975;iii:60-4. <sup>9</sup> Larson DL, Abston S, Evans EB, Dobrovsky M, Linares HA. Techniques
- arson DL, Abston S, Evans EB, Dobrovsky M, Linares HA. Techniques  $\int_{0}^{\infty}$  for decreasing scar formation and contractures in the burned patient. 7 Trauma 1971;11:807-23.
- <sup>10</sup> Musgrave RH. The pitfall of surgical excision of vaccination scars in the deltoid area. Plast Reconstr Surg 1973;51:198-9.
- <sup>11</sup> Griffith BH. The treatment of keloids with triamcinolone acetonide. Plast Reconstr Surg 1966;38:202-8.
- <sup>12</sup> Ketchum LD, Smith J, Robinson DW, Masters FW. The treatment of hypertrophic scar, keloid and scar contracture by triamcinolone aceto-

hypertrophic scar, keloid and scar contracture by transcription nide. Plast Reconstr Surg 1966;38:209-18. <sup>13</sup> Kempe CH, Benenson AS. Smallpox immunization in the United States. *JAMA* 1965;194:161-6. <sup>14</sup> Muliken JB, Gifford GH, Goldwyn RM. Vaccination caveat. The off-the-shoulder look. Am J Dis Child 1976;130:1094-5. **An absence of alcohol policy** Does the Government have a strategy for dealing with alcohol abuse, asked Lord Avebury in his opening address to the BMA symposium sponsored by the Medical Council on  $\overline{\varphi}$ Alcoholism and the Scotch Whisky Association to examine N that very question? His question was never answered D directly, and nor were many others, but in his finely written speech, Mr Geoffrey Finsberg, Joint Parliamentary Under Secretary of State for the Department of Health and Social Security, strove to give the impression that the Government did have a strategy. In our opinion he failed.

Mr Finsberg spoke only for the Department of Health and  $\exists$ Social Security, and, as the Central Policy Review Staff told us in its report,<sup>1</sup> no fewer than 16 different Government departments have an interest in alcohol. Many of these departments-for instance, the Treasury-are much higher in the departmental pecking order than the Department of Health and Social Security, and some of these departmentsfor instance, the Department of Employment and the Board of Trade-are more interested in jobs and profits than in the nation's health. The DHSS is left with few resources and little good will to cope with the problems that result from Britain drinking twice as much as it did two decades ago.

The Central Policy Review Staff's report on alcohol policies (which the Government refused to publish in 1979 but which was published this year in Sweden by Professor Kettil Bruun, who also spoke at the conference) concluded that "neither the existing machinery within Government, nor the bodies outside 2 it, provide the means for coherent formulation of policies...." It recommended that an advisory council on alcohol policies should be established with associated internal coordinating 2 arrangements. This recommendation, we suspect, is the one of that particularly upset the Government and caused it to suppress the report. The Government does not want a g coherent policy—it wants to have its cake and eat it. It wants of the £3597m brought in through tax on alcohol, and the £500m  $\frac{25}{250}$