Photomicrographs of the excised specimens. (A) Showing dysplastic cortex with disordered cortical lamination, malorientated nerve cells, and focal clustering (haematoxylin and eosin, originally $\times 160$, bar represents $25\ \mu\text{m}$). (B) Showing large dysplastic nerve cells (haematoxylin and eosin, originally $\times 400$, bar represents $62.5\ \mu\text{m}$).

of partial seizures.^{1,2} There have recently been major advances in structural brain imaging that allow detection of cortical abnormalities with high resolution MRI,²⁻⁴ although a considerable proportion of cases are not identifiable.^{3,4}

The histological diagnosis, complications, and failure of the patient to respond to pharmacological measures led us to perform multiple subpial transections with dramatic effect. The results of surgical treatment for cortical dysplasia depend on the site and grade of the lesion and type of procedure performed.⁴ The most established techniques are lesionectomy, resection of epileptogenic tissue as assessed by electrocorticography, or a combination of the two procedures. The use of multiple subpial transections has also been described⁵ and used in an attempt to avoid any deficit resulting from excision of primary sensorimotor cortex.^{5,6} This technique has been successfully used to end EPC due to Rasmussen's syndrome,⁹ but this is the first reported case we are aware of in which it has been used as a treatment for intractable epilepsy partialis continua due to cortical dysplasia. The outcome in terms of seizure control was good and our patient was spared the hemiparesis that would have resulted from resection of the motor cortex. We conclude that multiple subpial transections should therefore be considered in patients with medically refractory EPC secondary to

cortical dysplasia, even when structural imaging is unremarkable.

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6 Sawhney IM, Robertson IJA, Polkey CE, *et al.* Multiple subpial transection: a review of 21 cases. *J Neurol Neurosurg Psychiatry* 1995;58: 344-9.

CORRESPONDENCE

Sjögren's syndrome in patients with chronic idiopathic axonal polyneuropathy

We appreciate the results of the study by van Dijk *et al*¹ and add some results of our own regarding peripheral neuropathy and primary Sjögren's syndrome.

The proportion of patients who fulfil the diagnostic criteria for Sjögren's syndrome certainly depends on disease classification. Grant *et al* propose a distinct syndrome which includes patients with peripheral neuropathy and sicca complex.² In their study minor salivary gland biopsy was positive in 73% of patients, which is in accordance with our studies.^{3,4} In our studies and those of Grant *et al*² labial gland biopsy was not performed until sicca symptoms were established. This supports the desirability of searching for a

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- 4 Kuzniecky R, Murro A, King D, *et al.* MRI in childhood intractable partial epilepsies: pathological correlations. *Neurology* 1993;43: 681-7.
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non-invasive test which should replace salivary gland biopsy.

In our study of 44 patients with chronic axonal neuropathy of small nerve fibres primary Sjögren's syndrome was confirmed in 23%. The smaller proportion (4.6%) found by van Dijk *et al.*¹ can be explained by the strict criteria used by inclusion of patients in our study. In other papers we stress the use of psychophysical methods besides routine electrophysiological tests in primary Sjögren's syndrome.³⁻⁴ It seems that Dijk *et al.*¹ as well as Grant *et al.*² ignored a group of patients with small nerve fibre involvement lacking the psychophysical assessment of thermal specific and thermal pain sensitivity as well as electrophysiological evaluation of the autonomic nervous system.

We agree with the authors that in patients with peripheral neuropathy sicca symptoms are often unrecognised. All of our patients were referred to the outpatient's clinic because of positive sensory symptoms. Mainly they had burning pain. In most patients routine electrophysiological findings were within normal limits whereas thermal specific and thermal pain sensitivity assessment as well as electrophysiological determination of autonomic nervous system function disclosed distinct abnormalities.

We are aware that there is no specific test to evaluate Sjögren's syndrome or peripheral neuropathy with sicca complex as defined by Kennett and Harding.⁵ However, there is no reason to perform labial gland biopsy in each patient with chronic idiopathic axonal neuropathy as described in the study of van Dijk *et al.*¹ Finally, we want to stress the need for a non-invasive method for diagnosing Sjögren's syndrome.⁵

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- 1 van Dijk GW, Notermans NC, Kater L, *et al.* Sjögren's syndrome in patients with chronic idiopathic axonal polyneuropathy. *J Neurol Neurosurg Psychiatry* 1997;63:376-8.
- 2 Grant JA, Hunder GG, Homburger HA, *et al.* Peripheral neuropathy associated with sicca complex. *Neurology* 1997;48:855-62.
- 3 Denišlić M, Meh D. Neurophysiological assessment of peripheral neuropathy in primary Sjögren's syndrome. *Clin Invest* 1994;72:822-9.
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Authors' reply:

We thank Denišlić and Meh for their comments on our study regarding Sjögren's syndrome in patients with chronic idiopathic axonal polyneuropathy (CIAP).¹ As another example of the wide range of peripheral nerve disorders in Sjögren's syndrome, they stress the importance of recognising patients with small nerve fibre disease. We agree that these patients were excluded from our study, as axonal nerve fibre dysfunction on electrophysiological examination was required to confirm the clinical diagnosis of polyneuropathy in our study, and the routine neurophysiological investigations often do not show abnormalities in patients with small nerve fibre disease. When small nerve fibre dysfunction of unknown cause is present, we also perform an investigation for Sjögren's syndrome as we described.

We do not recommend performing a labial salivary gland biopsy in every patient with CIAP. As in the figure, we suggest performing further tests for Sjögren's syndrome in men with CIAP only when ocular or oral sicca symptoms are present and only a labial salivary gland biopsy when objective tests for tear gland and salivary gland dysfunction or serological abnormalities are also present.

Diagnostic approach to Sjögren's syndrome in patients with chronic idiopathic axonal polyneuropathy (CIAP). SSG = sublabial salivary gland biopsy.

However, in women with CIAP we suggest a full investigation for Sjögren's syndrome that includes a questionnaire for ocular and oral symptoms, a Schirmer's test or Rose bengal staining score, and determination of serological abnormalities; when an abnormal result is found in any of these tests, a sublabial salivary gland biopsy should be performed to see if a diagnosis of Sjögren's syndrome can be made.

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- 1 Van Dijk GW, Notermans NC, Kater L, *et al.* Sjögren's syndrome in patients with chronic idiopathic axonal polyneuropathy. *J Neurol Neurosurg Psychiatry* 1997;63:376-8.

BOOK REVIEWS

Neurology Practice Guidelines. Edited by RICHARD LECHTENBERG, HENRY S SCHUTTA. (Pp 544; US\$150). New York: Marcel Dekker, 1998. ISBN 0-8247-0104-6.

With an ever increasing number of neurology texts concentrating on diagnosis and medical science, a book with an emphasis on therapeutics is a welcome addition. The key problem is that whereas older treatments may change little, up to date ones change in the time it takes such a book to come to fruition. Nevertheless this text does cover a broad range of therapeutic issues with access to key publications in a way that would be difficult to find in another single volume.

It is divided into sections, for example headache, degenerative diseases, and seizure disorders. Each starts with a brief overview and goes on to chapters dedicated to specific topics. Common diseases are given substantial coverage but the choice of more minor topics is curious. Whole chapters are devoted to botulism, diphtheritic neuropathy, and organophosphate poisoning and a few lines only to tuberculosis or AIDS and its complications. The emphasis varies throughout the book. A very valuable chapter on Parkinson's disease devotes three pages to diagnosis and 40 pages to topical issues in therapeutics, whereas the chapter on myasthenia gravis devotes 14 pages to diagnosis and eight to treatment. In some chapters the discussion of treatment is little more than a list of drugs and their side effects. The more successful chapters give an analysis of relevant publications and try to give up to date treatment options for specific clinical situations. Others are more didactic, drawing on the authors' personal experiences. The authors are Ameri-