and 10 years (group B) and those surviving for more than 10 years (group A). The longest period of follow up in Group A was 39 years with a mean follow up period of 16·7 years. In group A there were three deaths, of which two were due to unrelated causes. One year before the death of case 3, he presented with a high fever and turbid CSF was obtained by lumbar puncture. Encephalitis Japonica was diagnosed, he was given no specific treatment, and was discharged from hospital with unsteadiness of gait and frequent convulsive seizures. One year later he complained of sudden headaches and convulsive seizures. He lost consciousness and died and there was no necropsy.

In group B (14 cases who survived between five and 10 years) 10 had died, eight due to recurrent tumour and two due to unrelated causes. The average age at operation and male/female ratio was 27·2 years and 8/7 in group A and 27·1 years and 10/4 in group B. The same surgical technique were used in both groups with 10 of 15 cystic tumours in group A and nine of 14 in group B. Histologically, five cases were adamantinomatous, seven were squamous, and three were not determined in group A, and in group B six were adamantinomatous, six squamous, and two undetermined. The NOR number was determined in 19 cases from their paraffin embedded specimens. All cases had a low proliferative potential (NOR 1·76 (SD 0·21) with no difference between the groups (group A 1·73 (0·13), 10 cases; group B 1·80 (0·27), nine cases).

It is well known that optic or hypothalamic glioma, cerebellar astrocytoma, ganglioglioma, and some non-glial benign tumours display spontaneous cessation of their growth after an active proliferative growth period but such a phenomenon has not been documented for craniopharyngioma. Among the 15 cases who survived more than 10 years in our series, there was no death due to tumour recurrence or regrowth. By contrast, among those 14 cases in group B with less than 10 years' survival, 10 patients died due to recurrence of the tumour. There are many statistical survival studies for craniopharyngioma but few dealing with long term survival.[3-6] Each of these series suggests that there is a low risk of recurrence once patients survive for 10 years. For example, in the series of Yasargil *et al*, patients surviving 11 to 22 years after primary surgery, no death occurred in 30 children and 33 adults—90% of these cases underwent total removal. Jose *et al* reported the follow up results of limited surgery and radiation therapy in 173 cases of craniopharyngioma with a median follow up of 12 years. The 10 and 20 year progression free survival rates were 83% and 79% and actuarial 10 and 20 year survival rates were 77% and 66%.

In conclusion, our study supports other evidence in the literature that there may be spontaneous reduction in the active proliferating potential of some craniopharyngiomas that is not predicted by the Ag-NOR technique. Of course, some craniopharyngiomas may always have a low proliferative potential and it is patients with these who survive for more than 10 years. Our data lend support to the policy of subtotal removal and subsequent adjuvant therapy in selected patients but a method for defining this subgroup remains unresolved.

AKIRA NISHIMOTO
TAKASHI MATSUHISA
KATSUZO KUNISHIO
TOMOHIDE MAESHIRO
TOMOHISA FURUTA
TAKASHI OHMOTO
Department of Neurological Surgery, Okayama University Medical School, 2-5-1 Shikata-cho, Okayama, Japan

Correspondence to: Dr Akira Nishimoto.

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Psychosocial factors in chronicity of epilepsy

Research has shown that after a recent diagnosis of epilepsy psychosocial effects are closely related to the severity of the medical condition and the recency of the last seizure,[1] suggesting that the stigmatising effect of the diagnosis cannot be viewed as the only or even the main predictor of psychosocial distress. Therefore, we must consider other factors. In this study we sought to explore the effect of chronicity. A comparative study was conducted, based on a validated psychosocial inventory,[2] of two groups of 62 subjects. These groups were matched for age (range between 17 and 57), sex, and employment type; duration of full time employment was similar as was tenure in current job. The chronic sample had a history of epilepsy spanning an average of 11 years and a minimum of five years. The chronic group was matched with respondents to the National General Practice Survey of Epilepsy (NGPSE)[3-5] drawn from a base of 594 patients registered by 275 primary care practices across the United Kingdom. The recently diagnosed sample had a history of epilepsy spanning a maximum of three years. The NGPSE is a population based national cohort study following up patients from the time of their first epileptic seizure. The importance of the NGPSE has been the ability to gather unbiased and comprehensive information on an unselected population based on prospective consecutive cases.

All patients in the chronic group had experienced more than 20 seizures. The recently diagnosed group also included some patients who had experienced large numbers of seizures but over 50% of the group had experienced three or fewer seizures at the time of data collection. The chronic patients were more likely to be registered disabled ($p < 0·01$), to have informed their employer of their epilepsy ($p < 0·05$), and to have had a seizure in public ($p < 0·01$).

Moderate or severe problems in adjustment of patients with a diagnosis of epilepsy

		Recent diagnosis (%)	*Chronic epilepsy* (%)
A1	Acceptance of the diagnosis	26	65*
A2	Fear of having seizures	75	69
A3	Fear of stigma affecting employment	48	53
A4	Lack of confidence about the future	24	68*
A5	Lack of confidence about travelling	15	39*
A6	Adverse effect on social life	29	62*
A7	Adverse effect on leisure pursuits	27	66*
A8	Change of outlook on life/self	29	67*
A9	Difficulty with the family	21	32
A10	Attitudes to taking medication	35	37
A11	Attitude to the medical profession	26	41
A12	Depression or emotional reactions	26	43*
A13	Feeling of increased social isolation	27	58*
A14	Lack of energy/lethargy	29	53*

*$p < 0·05$.

The table shows the percentage of patients who indicated a moderate or severe problem of adjustment in each of the 14 areas comprising the psychosocial inventory.

Variable means were compared with *t*-tests. A significance level of $p < 0·05$ was taken to indicate an important difference between groups.

Clearly, epilepsy, or at least having seizures, can be very distressing for the patient; the inventory not surprisingly showed that most patients with epilepsy have problems of psychosocial adjustment. Only in one area, however, did significant difficulties occur in more than 50% of the newly diagnosed group, this was the fear of seizures; in the chronic group, difficulties occurred in more than 50% of patients in nine of the 14 areas. The chronic group were more worried about their future, had a more negative outlook on life, had problems in relation to social life and leisure activities, and were more likely to feel socially isolated.

Although these findings clearly show that chronicity has an adverse effect on adjustment to epilepsy overall, the following areas were not affected: fear of stigma in employment, attitudes towards the medical profession, attitudes towards taking medication, and difficulties with the family. In these areas, seemingly unaffected by the experience of having epilepsy over a period of time or by the experience of a larger number of seizures, other dynamics must be involved in causing problems with adjustment, for instance the effect of the stigma of diagnosis. In different patients, however, factors vary widely and responses to the condition vary in a complex manner. Interventions, therefore, intended to improve psychosocial adjustment, require a high degree of complexity and flexibility.

The findings from this study are important for two reasons: firstly, they illustrate that a potent factor affecting psychosocial adjustment is chronicity; secondly, they emphasise that in most areas stigma is not "inherent" in the condition, and furthermore that there are wide individual variations. We have focused on chronicity but there are of course other factors involved in the process of adjustment. The problems of psychosocial adjustment are clearly relevant

for the person with epilepsy, but these findings also show that poor adjustment is not inevitable.

J E CHAPLIN
Rehabilitation Resource Centre, Department of Systems Science, City University, Northampton Square, London EC1V 0HB, UK
SD SHORVON
INSEG (Institute of Neurology and National Society for Epilepsy Research Group), Institute of Neurology, London and Chalfont Centre for Epilepsy
M FLOYD
Rehabilitation Resource Centre
R YEPEZ LASSO
INSEG, Institute of Neurology

Correspondence to: Dr Chaplin

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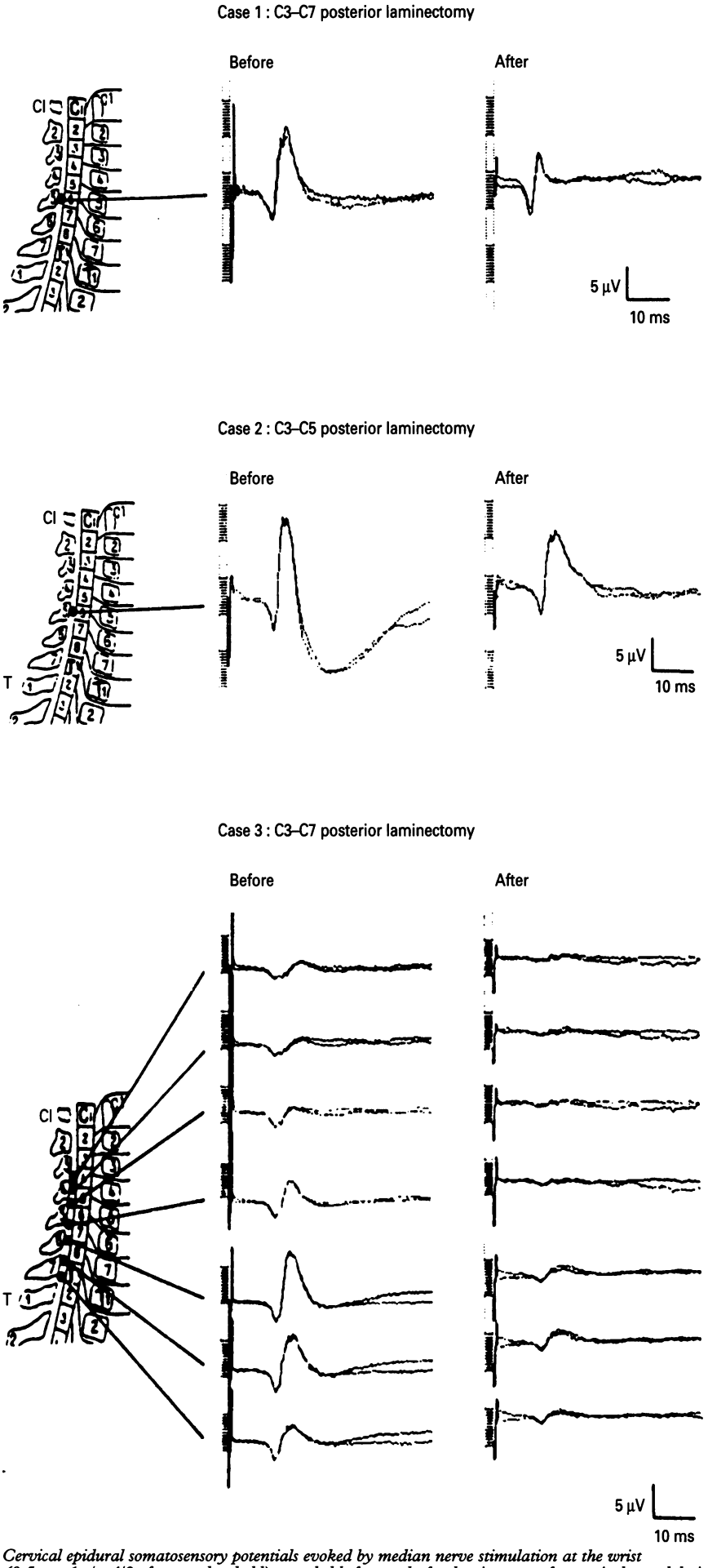
Spinal somatosensory potential monitoring in three cases of neurological deterioration after laminectomy for cervical spondylotic myelopathy

Posterior cervical laminectomy is a widely accepted treatment for multisegmental cervical spondylotic myelopathy, particularly when the condition is associated with a narrow spinal canal.[1] After laminectomy, some patients complain of increased weakness and paraesthesiae of the arms. This clinical finding is consistent with segmental damage either at the spinal cord or nerve root level. We report three patients of transient neurological deterioration in whom spinal somatosensory potential monitoring during operation gave useful information on the pathophysiology of the postoperative deficit.

Cervical spinal somatosensory potentials were recorded during operation, before and after laminectomy. The potentials were evoked by electrical stimulation of the median nerve at the wrist. Rectangular pulses (0·5 ms duration and amplitude 4/3 of the motor threshold) were delivered at 1 cycle/s. After exposure of the cervical laminae, the recording electrode (Medtronic Sigma 3483 or Quad 3487A) was placed in the epidural space, medially over the posterior columns. The reference electrode consisted of a 14 G needle inserted into the paraspinal muscles immediately caudal to the skin incision. A total of 50–150 stimuli were applied and averaged; analysis time was 50 ms with a horizontal resolution of 98 μs per point. An open bandpass (2–5000 Hz) was set and the negative upward convention was used. The potentials were analysed for latency, amplitude, duration, and waveform. The single components were labelled according to their polarity and to the expected latency.

Anaesthesia was induced with thiopentone sodium (5 mg/kg) and fentanyl (0·002



Cervical epidural somatosensory potentials evoked by median nerve stimulation at the wrist (0·5 ms, 1 c/s, 4/3 of motor threshold) recorded before and after laminectomy for cervical spondylotic myelopathy.