forearm in radial nerve palsy but did not specifically note damage to its important branch, the PIN. During the first world war Tinel described the anatomy without mentioning the nerve by name and without discussion of causation. By the end of the second world war the PIN had been so named.¹

The PIN consists of one bundle of motor nerve fibres at the arcade of Fröhse (the supinator arch) but divides into two bundles near its point of exit from the supinator muscle. The recurrent superficial motor branch innervates the extensor digitorum, extensor digiti minimi, and extensor carpi ulnaris; the descending or deep motor branch innervates the abductor pollicis longus, extensor pollicis brevis, extensor pollicis longus, and extensor indicis.²

The syndrome is also known as the supinator syndrome. It is distinguished from a seventh cervical root syndrome in which the triceps is involved but the wrist extensors usually are spared.

Palsy of the PIN produces the characteristic syndrome of weakness of extension of the digits and thumb with preservation of extension of the wrist which, nevertheless, may deviate radially. Sensory function, supination, and brachioradialis are not affected. Pain is often felt lateral to the elbow.

Palsy of the PIN may be caused by acute trauma in gunshot wound or pressure from a space occupying lesion. Repetitive use of the forearm has caused PIN palsy in a bartender, violinist, swimmer, conductor, waiter, factory worker, and an embroiderer. It has also been reported with strenuous arm exercises, snow shovelling, in frisbee games and tennis playing, and after the prolonged carrying of an M60 machine gun. It has also been reported after cannulation of a forearm vein.³

We report a case of PIN palsy after repetitive movements of the forearm and we have named this variant "upholsterer's PIN".

A 47 year old right handed woman, an enthusiastic pianist, at an upholstery class one evening used both hands to compress springs and apply clips to the springs. During that evening she noted aching around the lower part of the right arm and on the next day found she could not extend the right thumb and fingers. She had not slept heavily on that side and had not noted trauma to the

elbow. She did not have cervical pain or numbness.

The region of the lateral epicondyle of the elbow was tender. Finger flexion and volar flexion at the wrist were strong as were flexion and extension of the elbow, supination, and pronation. Examination failed to show movement in the extensor digitorum and extensor pollicis longus.

An EMG showed changes typical of a PIN lesion—namely, active denervation in the extensor carpi radialis brevis, extensor digitorum communis, and extensor carpi ulnaris. The brachioradialis, biceps, and triceps brachii were normal as was sensory conduction in the superficial branch of the radial nerve. The active denervation noted in the extensor carpi radialis longus is an anomaly and suggests aberrant innervation or anatomical anomaly.

At exploration the PIN was degenerate from the origin of the superficial radial nerve to the branch of the brachioradialis although it did not seem compressed. An 8 to 10 mm segment of obvious abnormality within the PIN itself was resected and the nerve ends sutured together. The patient continued to recover function and did not require tendon transfer as her functional deficit substantially improved.

The histology of the excised segment showed very severe demyelination, presumed to be the primary pathology. Demyelination secondary to underlying axonal disease could not be excluded (figure 1).

The clinical features were typical of a PIN palsy. However, EMG studies showed active denervation in extensor carpi radialis longus and extensor carpi radialis brevis. As the extensor carpi radialis longus is innervated by the radial nerve before the origin of the PIN, it is possible to distinguish a PIN palsy from a more proximal radial nerve injury, which typically produces wrist drop and has also been reported after repetitive muscle contraction.⁴

We suggest that focal demyelination in the PIN and conduction block resulted from ischaemia due to direct pressure on the nerve at an area of anatomical constriction as a result of the repetitive action of the surrounding muscles.

Patients with PIN palsy sometimes report a history of forceful, repetitive rotation of the

forearm preceding the onset of symptoms. Because we do not know of a report of PIN palsy in association with the compressing of springs, we have named this palsy "Upholsterer's PIN".

We are extremely grateful to Dr Elias Ragi for the nerve conduction studies and to Dr R Simpson for supplying the photographs illustrating nerve demyelination.

S M WILSON

V DEVARAJ

C GARDNER-THORPE

Plastic Surgery Department and the Department of Neurology, Peninsular Medical School, Royal Devon and Exeter Hospital, Barrack Road, Exeter EX2 5DW, UK

Correspondence to: Dr C Gardner-Thorpe
cgardnerthorpe@doctors.org.uk

- 1 Spinner M. The arcade of Fröhse and its relationship to posterior interosseous nerve paralysis. *J Bone Joint Surg Br* 1968;50:809-12.
- 2 Suematsu N, Hirayama T. Posterior interosseous nerve palsy. *J Hand Surg (Br)* 1998;23:104-6.
- 3 Staal A, et al. *Mononeuropathies: examination, diagnosis and treatment*. London: W B Saunders, 1999:46.
- 4 Streibe E. Upper arm radial nerve palsy after muscular effort: report of three cases. *Neurology* 1992;42:1632-4.

CORRESPONDENCE

Neurocysticercosis and epilepsy in developing countries

I have several comments to make about the article by Pal *et al*¹ on neurocysticercosis and epilepsy in developing countries.

The title should refer to "neurocysticercosis in India" because almost all examples deal with that country, despite the fact that, as the authors correctly state, it is a disease found in many developing countries. Furthermore, several statements made by the authors do not reflect the knowledge gathered in Mexico, which is summarised in this letter.

The authors indicate in the text that "Population control measures have not shown long term effectiveness" . . . "There is little epidemiological data on risk factors" . . . "Migration from the countryside and the risk of urban slums obviously influence the changing epidemiology" . . . "mass treatment with praziquantel may produce early benefit but larger term evaluation shows no lasting impact". Sarti *et al* have studied exhaustively risk factors² and evaluated control measures in Mexico; the results indicate that the main risk factor is the tapeworm carrier in the close environment, and that treatment of tapeworm carriers or health education of the community are useful control measures as evaluated at 6 and 42 months after their implementation in rural populations.^{3,4} Moreover, vaccines for swine cysticercosis are being successfully developed and evaluated.⁵

Multiple studies performed in various countries clearly show the usefulness of serological methods for diagnosis, including antigen detection, which Pal *et al*¹ indicate was recently developed, but there are several prior references including that of 1989 by Correa *et*

Figure 1 Nerve biopsy stained by osmium tetroxide shows demyelination with some residual myelin in globules.

al with the same monoclonal antibodies and in the same *Journal* (see Flisser² for review). Similarly, the authors lack knowledge on the initial Mexican publications regarding the association of HLA and cysticercosis.²

Many Mexican authors have published long ago the clinical and imaging descriptions of neurocysticercosis as well as its classification.² Furthermore the authors criticise cestocidal treatment in neurocysticercosis and state that "there is no evidence that cysticidal treatment does more good than harm". Publications by Mexican authors²⁻⁶ clearly demonstrate the usefulness of drug treatment, as does the welfare of patients (children and adults) after such treatment seen in routine work by many neurologists in Mexico. The fact that seizures and inflammation are exacerbated during treatment is the main manifestation of a cestocidal effect, as was experimentally described in cysticercotic pigs that received treatment and had an important enhancement of the inflammatory response surrounding the parasites.²⁻⁶ Due to this, anti-inflammatory and anticonvulsive drugs are recommended in an individual based scheme.

The second part of the article—"management of epilepsy in the developing world"—misleads on the problem of cysticercosis, as it deals almost exclusively with epilepsy in children, when it is well known that the association of this syndrome with neurocysticercosis is mainly with late onset epilepsy.²⁻⁶

From these considerations, different conclusions are drawn:

- Epidemiology of neurocysticercosis is quite well known (not only in Mexico but in several countries where it has been studied)
- Imaging and serological diagnosis are useful tools
- Cysticidal treatment is usually beneficial to patients with neurocysticercosis
- Neurocysticercosis is a major cause of acquired epilepsy
- Prevention measures, based on health education, targeted treatment, and swine vaccination, will probably be sufficient to control neurocysticercosis, and even to eradicate the parasite.

A FLISSER

Departamento de Microbiología y Parasitología,
Facultad de Medicina, Universidad Nacional
Autónoma de México, México DF 04510, México

Correspondence to: flisser@servidor.unam.mx

- 1 Pal KD, Carpio A, Sander JWAS. Neurocysticercosis and epilepsy in developing countries. *J Neurol Neurosurg Psychiatry* 2000;68:137-43.
- 2 Flisser A. Taeniasis and cysticercosis due to *Taenia solium*. In: T Sun, ed. *Progress in clinical parasitology*. Florida, USA: CRC Press, 1994: 77-116. (Review article.)
- 3 Sarti E, Schantz PM, Avila G, et al. Mass treatment against human taeniasis for the control of cysticercosis: population-based intervention study. *Trans R Soc Trop Med Hyg* 2000;94:85-9.
- 4 Sarti E, Flisser A, Schantz PM, et al. Development and evaluation of a health education intervention against *Taenia solium* in a rural community in Mexico. *Am J Trop Med Hyg* 1997;56:127-32.
- 5 Plancarte A, Flisser A, Gauci CG, et al. Vaccination against *Taenia solium* cysticercosis in pigs using native and recombinant oncosphere antigens. *Int J Parasitol* 1999;29:643-7.
- 6 Sotelo J, Flisser A. Neurocysticercosis. Practical treatment guidelines. *CNS Drug* 1997;7:17-25. (Review article.)

High psychiatric morbidity in patients with medically unexplained symptoms

Carson *et al*¹ reported that one third of the patients referred by general practitioners to general neurology outpatient clinics had

symptoms that were poorly explained by identifiable organic disease. Similar trends have been reported from a developing country—namely, India. Bagadia *et al*,² for example, have reported that 57% of non-acutely ill patients attending a general hospital medical outpatient department scored high on the general health questionnaire, indicating a likelihood of psychiatric morbidity. The same authors in another study³ have reported that in a general medical outpatient department, 36% of the patients had psychiatric morbidity, of which 24% had pure psychiatric illness and 12% had psychiatric disorders with associated physical illnesses. These figures have an important bearing on the health care delivery system. These patients take up considerable time of a medical specialist for reasons other than those he specialises in and they also account for higher medical expenses.

Other findings of the same study give us important clues for circumventing this problem. For instance, 66% of those patients whose symptoms were not fully explained by "organicity" had diagnosable anxiety and depressive disorders. Referring such patients directly to a psychiatrist would save time of both the consultant neurologists and the patients. One way of doing this would be to "sensitise" the general practitioners about identification of psychiatric problems. An Indian study showed that after a brief period of training in detection of psychiatric morbidity, the sensitivity of rural primary care physicians increased.⁴

We highlight some of the shortcomings of the report of Carson *et al*.¹ In a strict sense, the design does not fulfil the requirements for a "prospective cohort study".⁵ The follow up assessments were based only on the review of "neurological case notes", the details of which are not made explicit in the article.

The degree by which patients' symptoms were considered to be medically explained was rated by five neurologists using a four point Likert scale. This measure is central to the study. Development and standardisation of the scale are not mentioned in the report. For assessing the prevalence rates a reliable and valid scale is needed. The authors have attempted to validate the scale by reviewing the "neurological case notes" after 6 months in consultation with the treating consultant when necessary. Whether the authors reviewing the case notes were blind to the first rating is not clear. The lack of blindness can lead to serious biases. Further, provision of the components of the "initial consultation" (whether it was based only on history and physical examination or it included relevant investigation) would have helped in understanding the use of the Likert scale better.

N MURALI

JAGADISHA

N KAR

S REHMAN

P S V N SHARMA

Department of Psychiatry, Kasturba Medical College,
MANIPAL-576 119, India

Correspondence to: Dr N Murali
navankul@yahoo.com

- 1 Carson AJ, Ringbauer B, Stone J, et al. Do medically unexplained symptoms matter? A prospective cohort study of 300 new referrals to neurology outpatient clinics. *J Neurol Neurosurg Psychiatry* 2000;68:207-10.
- 2 Bagadia VN, Ayyar KS, Lakdawala PD, et al. Value of the general health questionnaire in detecting psychiatric morbidity in a general hospital outpatient population. *Indian Journal of Psychiatry* 1985;27:293-6.

- 3 Bagadia VN, Ayyar KS, Lakdawala PD, et al. Psychiatric morbidity among patients attending medical outpatient department. *Indian Journal of Psychiatry* 1986;28:139-44.
- 4 Isaac MK, Kapur RL, Chandrasekhar CR, et al. Mental health delivery through rural primary care: development and evaluation of a pilot training programme. *Indian Journal of Psychiatry* 1982;24:131-8.
- 5 Regier DA, Burke J. Quantitative and experimental methods in psychiatry. In: Sadock BJ, Sadock VA, eds. *Kaplan and Sadock's comprehensive textbook of psychiatry*, 7th ed. London: Lippincott Williams and Wilkins, 2000:500-45.

The psychoses of epilepsy

The recent editorial by Toone¹ serves to highlight the deficiencies in our understanding of the relation between epilepsy and psychosis. Although some aspects of this association are now better established than they were at the time of Slater's classic report,² insights into the pathophysiological processes underlying these psychoses seem to have advanced very little. A major reason for this, in my opinion, is that the greater focus of research has been on chronic schizophrenia-like psychoses rather than on the episodic psychoses, which are more likely to yield testable hypotheses.

It can be argued that postictal psychoses present the best opportunity to examine some of the pathophysiological mechanisms that underlie the relation. These psychoses occur in patients who have generally been examined in their prepsychotic states, are likely to develop in hospital while the patient is being monitored for epilepsy, and can be closely studied up to their termination and possible recurrence. That they usually occur in relation to partial complex epilepsy that is secondarily generalised suggests the importance of bilateral involvement of limbic structures in their genesis. The fact that postictal psychoses often follow a cluster of seizures must bring into focus the role of neural inhibitory processes in their development. Epileptic conditions are presumed to involve a chronic imbalance of excitatory and inhibitory influences,³ whereas increased inhibition plays an important part in the development and maintenance of the interictal state.⁴ Human temporal lobe epilepsy is associated with enhanced inhibition,⁵ which contrasts it from neocortical epilepsy in which the epileptogenic regions demonstrate reduced afterdischarge thresholds.⁶ The role these inhibitory processes have in the genesis of psychosis has not been adequately examined. Active inhibitory processes may produce focal reversible deficits—such as Todd's paralysis—which may persist for long periods. Is it possible that postictal psychoses are a manifestation of similar processes bilaterally in the limbic cortex? The question lends itself to examination using electrophysiological and neuroimaging techniques, and even in vivo neurotransmitter measurement using microdialysis in patients being monitored with depth electrodes.⁷

The inhibitory processes not only bring about the postictal state but are also necessary for maintaining the interictal state. Postictal and brief interictal psychoses may therefore not be so different from each other as is generally thought. As the development of seizures may be due to either disinhibition or hypersynchrony involving enhanced disinhibition,³ the occurrence of a seizure during psychosis may have different pathogenetic mechanisms and may indicate either disinhibition or increased inhibition. The effect on