

LETTERS TO THE EDITOR

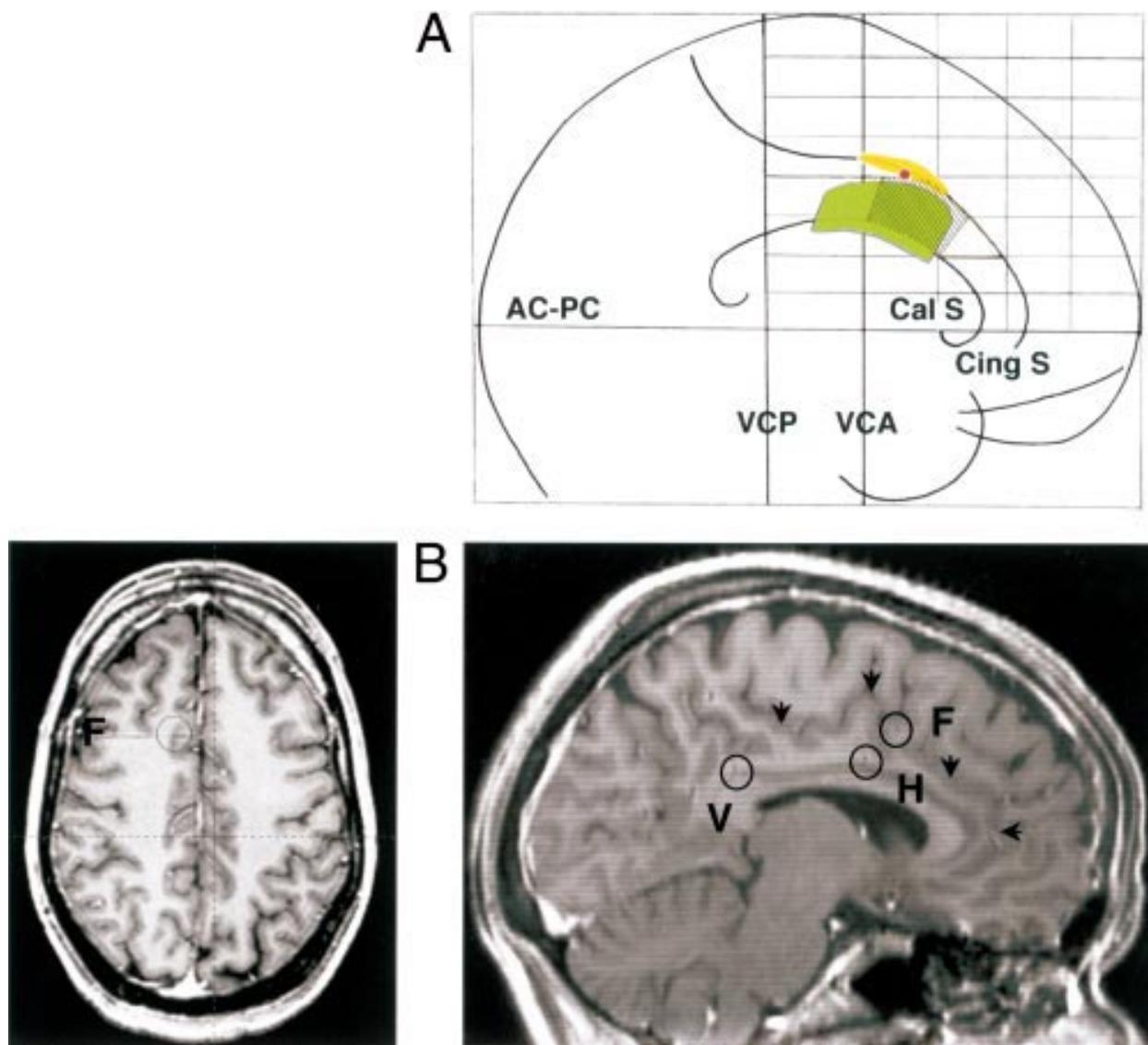
The cingulate hidden hand

Studies on primates, and increasing evidences in humans, support the notion that the anterior cingulate cortex, which subserves various executive functions, is involved in the preparation and execution of motor operations.¹ Recently, the specific role of the caudal part of the anterior cingulate cortex in manual control has been demonstrated in a

patient with a focal anterior cingulate cortex lesion,² thus providing additional arguments for the functional specialisation of cingulate motor areas in the human brain. Interestingly, the damaged area widely overlapped a more anterior part of Brodmann's area 24 from where complex coordinated movements adapted to environmental constraints have been electrically induced in epileptic patients (figure A).³ Both areas encompass dorsally the ventral bank of the cingulate sulcus, at a level that seems to correspond to the simian rostral cingulate motor area (CMAR),¹ but whether this region is specifically involved in voluntary movements in humans is not known. We report a finding which shows for the first time that compulsive goal directed motor behaviour can be electrically induced

in humans by stimulating the anterior cingulate sulcus.

The patient, a 30 year old right handed woman, had medically intractable epileptic seizures which proved to arise from the right parietal cortex. Before surgery, she underwent intracerebral EEG recordings and stimulation to locate the epileptogenic zone, using 13 stereotactically implanted multilead intracerebral electrodes, three of which investigated the right cingulate cortex. Electrical stimulation at low intensity of the anterior and posterior cingulate gyrus (figure B, electrodes H and V) did not result in any motor reaction. Conversely, using the same parameters, stimulation of the ventral bank of the anterior cingulate sulcus (figure A red dot, and B electrode F) incited the patient to act.



Anatomical representation of the electrical stimulation site. (A) Normalised proportional grid system of the atlas of Talairach and Tournoux, sagittal view. The anterior cingulate cortex lesion of the case of Turken and Swick² (in green) widely covers the cingulate area from where electrical stimulation can elicit complex coordinated movements (cross hatching).³ The proposed location of the CMAR is represented in yellow,⁴ and includes the stimulation site from where an "incitement to act" has been induced in the present case (red dot). AC/PC=anterior commissure/posterior commissure; VCA/VCP=coronal plane passing through the anterior/posterior commissure; Cal S/Cing S=callosal/cingulate sulcus. (B) MRI of the patient performed after removing the electrodes. Right side: right parasagittal view showing the three sites of stimulation in the cingulate cortex. Bipolar electrical stimulation (1 ms, 50 Hz, 5 s) was delivered from a conventional rectangular pulse generator and applied between 2 mesial contiguous contacts (0.8 mm diameter, 2 mm length, 1.5 mm apart) of the electrodes V, H, and F which were respectively located in the posterior cingulate gyrus, in the anterior cingulate gyrus, and in the ventral bank of the anterior cingulate sulcus. Bipolar recordings between these contiguous contacts were previously shown to exhibit cortical electrical activity. Stimulation was performed at low intensity (V: 1.2 mA, H: 1.2 mA, F: 1.4 mA) under the threshold of afterdischarge. Incitement to act was elicited on F. Arrows=cingulate sulcus. Left side: horizontal view passing through the plane of electrode F showing that the site of stimulation where an "incitement to act" was induced was clearly located in the ventral bank of the cingulate sulcus (empty circle). The stimulated contacts were located laterally at 9.5 mm and 13 mm from the medial line.

It consisted in an irresistible urge to grasp something, resulting in exploratory eye movements scanning both sides of the visual field, and accompanied by a wandering arm movement contralateral to the stimulation side. Then, after the patient had visually localised a potential target, her left hand moved towards the object to grasp it, as if mimicking a spontaneous movement. This irrepressible need started and ended with stimulation, and the patient was unable to control it. Yet, the patient was aware of both her inability to resist and of the movement she thus performed and could describe very precisely. Interestingly, the arm movement seemed visually guided, as when the patient was asked to keep her eyes closed while stimulated, her arm executed a wandering movement which did not result in grasping an object. This was not true if the object location had been memorised before closing her eyes; in that case, the arm moved blindly towards the place where the object was set.

Our finding demonstrates that a cingulate motor area buried in the ventral bank of the cingulate sulcus, just below the presupplementary motor area, is engaged in motor intention in humans, involving the contralateral upper limb. We can assume that the stimulated zone was restricted to the cingulate sulcus, as the stimulation was performed here with a highly localising technique using low intensity in bipolar mode through adjacent contacts distant only by 3.5 mm. This area could be homologous to the simian CMAR, which contains many neurons firing in relation to the intention to move,⁴ and has essentially an arm representation. In addition, the CMAR lies within the cingulate sulcus anterior to the VCA plane, and then seems embedded in foci of activation seen in humans during relatively simple movements, as well as during more complex manual tasks. It remains that the behavioural response we found involved a high level of motor integration, as previously reported in epileptic patients when stimulating the anterior cingulate gyrus proper,⁵ but it was preceded in the present case by an “urge to grasp”, which gave rise to a compulsive and adapted manual reaction only when there was visual guidance. The anterior cingulate sulcus has been recently proved in humans to be responsive to visual stimuli,⁵ and together with our finding, this may suggest that this area plays a part in integrating visual information in execution for movement.

S KREMER
S CHASSAGNON
D HOFFMANN
A L BENABID
P KAHANE

Neurophysiopathologie de l'Épilepsie,
Department of Neurosurgery and INSERM U318
Research Unit, CHU de Grenoble,
BP217 X - 38043 Grenoble Cedex, France

Correspondence to: Dr P Kahane
philippe.kahane@ujf-grenoble.fr

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Localised myelitis caused by visceral larva migrans due to *Ascaris suum* masquerading as an isolated spinal cord tumour

Eosinophilic meningitis is caused by various parasites, a representative of which is *Angiostrongylus cantonensis*. The disease has also occasionally been reported in visceral larva migrans due to *Toxocara canis*, although the parenchymatous involvement of the CNS is extremely rare in *T canis* visceral larva migrans.¹ Recently an outbreak of visceral larva migrans due to *Ascaris suum* infection has been reported in Kyushu, Japan, where chemical fertiliser has been replaced in part with pig manure.² We report a case of myelopathy probably due to *A suum* infection.

A 22 year old man, living in the Tokyo metropolitan area, noticed that his right hand was swollen and warm in mid-August, 1999. The oedema subsided spontaneously within a week. In early October, he felt thermoaesthesia in his right leg while he was taking a shower. Because he had sometimes felt numbness in one or both axillae from the beginning of November, he was admitted to a hospital on 7 December 1999. Physical examination on admission showed hypalgesia and thermoaesthesia below the Th9 level on the right and a positive Lhermitte's sign. Peripheral blood eosinophil count was raised at 610/μl (10.5% leucocytes). Serum IgE concentration was 155 IU/ml (normal <240 IU/ml). Thoracic MRI disclosed an isolated high signal intensity lesion at the Th1 spine level on T2 weighted images. His symptoms improved gradually without any treatment. He was transferred to our hospital for diagnostic evaluation on 31 January 2000. He had a habit of eating raw beef liver and chicken liver. He had been to Fukuoka City, which is located at the northern part of Kyushu island, and ate raw beef liver in early August, 1999.

Physical examination on 31 January was normal except for the positive Lhermitte's sign. The peripheral blood eosinophil counts remained increased at 470/μl (7.0% leucocytes). Serum IgE, IgM, IgG, IgA, glutamic oxaloacetic transaminase, and glutamic pyruvic transaminase were normal. Antinuclear antibody, anti-ds-DNA antibody, c-ANCA, p-ANCA, and all other autoantibodies tested for were negative. Radioallergosorbent tests for *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae* were positive. Chest radiography and CT of the liver were normal.

His CSF showed 10 cells/μl, 10% of which were eosinophils, a protein concentration of 65 mg/dl, normal glucose concentration, and a normal IgG index. Oligoclonal IgG bands were negative. Spinal cord MRI showed a high signal intensity lesion at the Th1 spine level on T2 weighted images, which was enhanced by gadolinium-DTPA (figure). Brain MRI, somatosensory evoked potentials, motor evoked potentials, visual evoked potentials, and peripheral nerve conduction studies were all normal. On multiple dot ELISA for 12 parasite antigens, *A suum*, *T*

canis, *Dirofilaria immitis*, *Anisakis simplex*, *Gnathostoma doloresi*, *Strongyloides ratti*, *Paragonimus westermanii*, *Paragonimus miyazakii*, *Fasciola hepatica*, *Clonorchis sinensis*, *Spirometra erinacei* and *Cysticercus cellulosae*, both serum and CSF bound strongly to *A suum* and weakly to *T canis* but not to the others. On Ouchterlony's double diffusion test in agarose gel, the patient's serum but not CSF formed a sharp precipitin band against *A suum* antigen, but not against either *T canis* or *Angiostrongylus cantonensis* antigens. Parasite eggs were not found in repeated stool examinations.

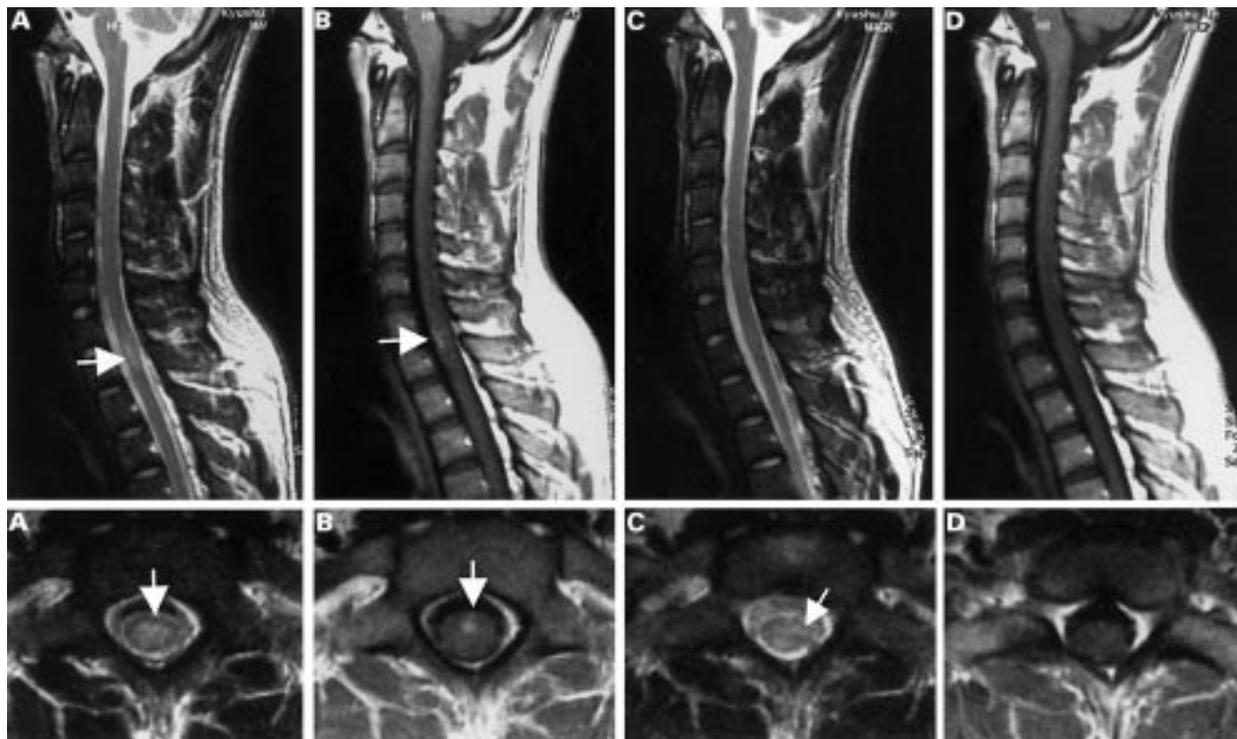
The diagnosis of *A suum* infection was made, and albendazole (600 mg/day) was given for 2 weeks with a 1 week interval. Thereafter, Lhermitte's sign as well as the MRI lesion were almost resolved (figure). His CSF after therapy showed normal cell counts (0/μl) and a normal protein concentration (41 mg/dl).

This is the first case report of myelopathy caused by visceral larva migrans probably due to *A suum*. The presence of a specific antibody against *A suum* in CSF as well as in serum, together with the presence of eosinophils in CSF, suggest that the spinal cord lesion is attributable to the presence of parasite larvae in the lesion. The resolution of the lesion after albendazole treatment strongly supports this hypothesis.

Among *A suum* visceral larva migrans cases, only one probable case of encephalopathy in which multiple enhanced lesions on brain MRI were markedly attenuated by corticosteroids has been reported.³ In that case, an immunological mechanism was suggested for the CNS involvement. In our patient, albendazole, which efficiently passes through the blood-brain barrier, showed beneficial effects clinically as well as on MRI. Ascarid larvae have been reported to survive longer in the parenchymatous tissue and secrete antigens that cause allergic reactions in hosts.⁴ Albendazole was therefore considered to have directly killed the larvae and effectively suppressed the host's immunological reactions in the present patient.

Visceral larva migrans due to ascarid parasites is characterised by hepatopulmonary lesions associated with marked eosinophilia,² which was also true for the previously reported patient with encephalopathy due to *A suum* visceral larva migrans.³ The present patient had neither pulmonary nor hepatic lesions, although he had had a transient hand oedema before the onset of neurological symptoms. Variations in clinical manifestations of visceral larva migrans may, at least in part, be attributed to the degree of infection. Even in a low grade infection without detectable hepatopulmonary lesions, ascarid larvae can migrate to an unexpected site to cause unusual clinical manifestations.

In areas where it is endemic, such as Kyushu, Japan, infection with *A suum* occurs primarily from ingesting vegetables contaminated with pig manure containing parasite eggs.^{2,3} In addition, some patients were assumed to be infected by eating raw beef or chicken liver contaminated with *A suum* larvae (Nawa Y, unpublished data). Infection of cattle with *A suum* has been reported when they were kept in the same field.⁵ Our patient developed myelopathy after ingesting raw beef liver, although he had never lived in the endemic areas. Because food borne parasitic zoonoses can be transported to areas distant from where they are endemic, neurologists should take a careful history of eating habits



Spinal cord MRI before (A, B) and after (C, D) albendazole therapy. T2 weighted images (A, C) and gadolinium enhanced T1 weighted images (B, D) are shown. The MRI lesion (arrow) seen before treatment was almost resolved after the treatment.

when eosinophilia that suggests visceral larva migrans is present in patients with neurological symptoms.

M OSOEGAWA
S MATSUMOTO
H OCHI
K YAMASAKI
I HORIUCHI
Y OHYAGI J-I KIRA

Department of Neurology, Neurological Institute,
Graduate School of Medical Sciences, Kyushu
University, Fukuoka 812–8582, Japan

K ISHIWATA
F NAKAMURA-UCHIYAMA
Y NAWA

Department of Parasitology, Miyazaki Medical
College, Miyazaki, 889–1692, Japan

Correspondence to: Professor Jun-ichi Kira
kira@neuro.med.kyushu-u.ac.jp

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Effect of lower limb position on ankle jerk assessment

The ankle jerk is one of the most commonly tested deep reflexes,¹ but the effect of the position of the lower limb on ratings of the reflex is unclear. Most commonly, the ankle jerk is assessed with the subject lying supine with the hip abducted and externally rotated, and the knee in moderate flexion before the

triceps surae tendon is tapped with the reflex hammer.^{2,3} Alternatively, the subject could sit with the legs dangling freely over the edge of the couch³ or with the knee on a chair with the ankles projecting freely over the edge of the chair.⁴ In a less commonly described position, the subject lies supine but the hip is adducted and the knee extended, and unlike the other positions, the triceps surae tendon is not struck by the reflex hammer, but the palmar surface of the examiner's hand placed against the sole of the foot.⁴ Anecdotal reports suggest that the ratings of ankle jerk are higher when the subject lies supine with the hip abducted and externally rotated,⁵ but one study suggested that the ankle jerk is best assessed with the hip adducted and the knee extended.⁴

It has been suggested that differences in the reported prevalence of absence of ankle jerk may be due to the position adopted for the lower limb to assess the ankle jerk rather than pathological absence of the reflex.^{4,6} To optimise the value of the ankle jerk in screening for polyneuropathy we compared the ankle jerk ratings of three examiners with subjects in three different lower limb positions to determine the lower limb position with the highest mean examiners' ratings.

Twenty one healthy young adults without neurological lesions were recruited into the study after informed consent. Examinations of three lower limb positions: subject supine with hip adducted and knees in extension; subject supine with hip abducted and externally rotated and knees in moderate flexion;

and subject kneeling with the ankles projecting freely over the edge of the chair, were performed as described.^{2–4} All subjects were examined in two replicate sessions by three examiners. The ankle jerk was rated on a scale of 0–3 as described in a standard text of neurological examination.³ The techniques of positioning the limbs, tapping the tendon and ratings of the reflex were reviewed and performed on three subjects not part of the study before examinations started. Examiners were blinded to the subjects, who were covered with only their feet exposed. The ratings of the ankle jerk by the three examiners for each lower limb position were treated as repeated measures for each subject, and averaged to produce the rating for the lower limb position. Differences in the mean examiners' ratings for the three lower limb positions were compared by non-parametric methods.

The mean examiners' ratings of the ankle jerk for the three lower limb positions are presented in the table. The mean rating was highest for the kneeling position and lowest for the hip adducted position. They were significantly different on non-parametric tests. The mean ranks on Kruskal-Wallis test were 33.3, 65.6, and 91.6 for the hip adducted position, hip abducted position, and kneeling position respectively in the first session; and 34.0, 64.6, and 92.0 for the hip adducted position, hip abducted position, and kneeling position respectively in the replicate session. χ^2 tests were significantly different for both sessions at $p < 0.0001$.

Ankle jerk ratings for three lower limb positions

Position	Ankle jerk ratings (first session)			Ankle jerk ratings (second session)		
	Mean	Median	Range	Mean	Median	Range
Hip adducted	0.8	0.7	0.0–3.0	0.8	0.7	0.0–3.0
Hip abducted	1.9	2.2	0.0–3.0	1.7	1.7	0.0–3.0
Kneeling	2.7	3.0	1.0–3.0	2.5	3.0	0.0–3.0

This study shows a strong effect of lower limb position on observer ratings of the ankle jerk in a sample of young healthy adults. This supports the notion that the position of the lower limb adopted to assess the ankle jerk may explain some of the differences in the prevalence of absent ankle jerk reported in several studies. The findings also agree with anecdotal reports³ that the sensitivity of the ankle jerk is higher when the hip is abducted than when the hip is adducted, but the kneeling position is the most sensitive of the three lower limb positions compared.

When screening for polyneuropathy, the kneeling position may be preferable to the other positions as it will reduce the number of false positives. However, the kneeling position will be unsuitable in settings where the patient is too ill or has impaired consciousness.¹

O S A OLUWOLE
T O ODEBODE
M O KOMOLAFE

College of Medicine, University of Ibadan, Nigeria

O S A OLUWOLE
Division of International Health, IHCAR,
Department of Public Health Sciences,
Karolinska Institute, SE-171 76,
Stockholm, Sweden

H LINK
Division of Neurology, Huddinge University Hospital,
Karolinska Institute

Correspondence to: Dr O S A Oluwole
osaoluwole@hotmail.com

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Randomised controlled trial of occupational therapy at home: results at 1 year

In the United Kingdom, about 25% of patients with stroke are not admitted to hospital. Unfortunately many of these patients remain in the community with little or no coordinated rehabilitation. We have recently published the results of a single blind randomised controlled trial of occupational therapy for patients with stroke remaining in the community.¹ We showed significant ben-

efits across a range of outcomes at 6 months after stroke; extended activities of daily living (EADL), personal ADL, gross motor function, handicap, and carer strain. Correspondence² suggested that these results were consistent with other similar smaller trials. In this report we present results at 1 year after stroke.

We identified patients from a community stroke register who had had a recent stroke (<1 month) and had not been admitted to hospital.¹ The stroke register covered a geographical area of Nottinghamshire and southern Derbyshire and incorporated 73 general practitioner practices, covering a population of 500 000 patients. Patients were included if they fulfilled the World Health Organisation (WHO) definition of stroke and had not been admitted to hospital. Patients were excluded if they lived in a nursing or residential home, could not speak or understand English before their stroke, or had a history of dementia. Patients were randomly allocated to up to 5 months of occupational therapy treatment at home (n=94) or to no intervention (n=91). The groups were well matched for baseline demographic characteristics.¹ The aim of occupational therapy was to encourage independence in personal and extended ADL. The number of visits given by the research occupational therapist to the intervention group ranged from 1 to 15, with a mean of six visits. Outcome measures were completed at 6 and 12 months after stroke. Outcome measures used at 1 year included the Barthel index, EADL scale, and the general health questionnaire (GHQ 28) for both patient and carer. All assessments were conducted in the patient's home by a blinded independent assessor.

Sixteen patients could not be assessed at 1 year: 10 had died during follow up (five in the occupational therapy group) and six withdrew from the study (four in the occupational therapy group). As the number of withdrawals from the study was small and evenly distributed between the groups, the analysis was conducted on the 147 patients who completed assessments. Demographic data at 1 year are illustrated in the table.

The primary outcome measure of the trial was the Nottingham EADL.³ Analysis of the EADL at 1 year after stroke, using the Mann-Whitney *U* test, showed that the group receiving occupational therapy were significantly less disabled (table).

Although it is important to ascertain whether an intervention is effective at certain time points, the above analysis does not take into account variation over time or that outcome measures at different points in time were from the same patients. We therefore applied an analysis of serial measurements.

The area under the curve (AUC) was calculated as a summary measure⁴ for outcome assessments in both groups. There was a significant difference between the groups in favour of the treatment group on the AUC EADL scale (Mann-Whitney *p*=0.001, 95% confidence interval (95% CI) 7 to 28) and the AUC Barthel index (Mann-Whitney *p*=0.01, 95% CI 0 to 9). There were no significant differences in the AUC for the GHQ for either the patient (Mann-Whitney *p*=0.48, 95% CI -39 to 21) or the carer (Mann-Whitney *p*=0.66, 95% CI -44 to 29).

This study indicated that patients who received occupational therapy had a greater level of independence in activities of daily living over a period of 1 year than patients who did not. Differences between the groups in terms of extended ADL were still apparent at 1 year.

The persistence of the beneficial effect of domiciliary occupational therapy adds further support to its clinical usefulness. There are grounds for establishing community occupational therapy services for patients with stroke, but further evaluation is required to confirm the generalisability of these findings and examine their economic implications.

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M F WALKER
K HAWKINS
Division of Stroke Medicine, Clinical Sciences
Building, Nottingham City Hospital, Hucknall Road,
Nottingham NG5 1PB, UK

J R F GLADMAN
Ageing and Disability Research Unit, B Floor,
Queens Medical Centre, Nottingham, UK

N B LINCOLN
School of Psychology, Nottingham University,
Nottingham, UK

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CORRESPONDENCE

Relation between Glasgow outcome score extended (GOSE) and the EQ-5D health status questionnaire after head injury

In this *Journal* Wilson *et al* investigated aspects of the validity of the Glasgow outcome score (GOS) and the extended form (GOSE), particularly the relation between the GOS and subjective reports of health status.¹ A potentially useful relation between the GOSE, measured using a standard interview at 1 month after head injury,² and health status assessed using the Euroqol EQ-5D is now reported. The GOS is the most widely used method to describe overall outcome after head injury.³ The EQ-5D questionnaire is a validated tool that measures health status in

Comparison between the groups on demographic characteristics and outcome measures 1 year after stroke

	Occupational therapy n=73	No intervention n=74	
Demographic characteristics at 1 year:			
Mean (SD) age	73.3 (7.8)	74.7 (8)	
Sex (M/F)	41/32	31/43	
Side of hemiparesis			
Left	37	35	
Right	28	33	
Other	8	6	
Lives alone	21	29	
Outcome measure at 1 year	Median (IQR) scores	Median (IQR) scores	<i>p</i> Value
Barthel index	19 (16-20)	18 (15-20)	0.16
EADL	13 (13-18)	11 (4-17)	0.04
GHQ 28 Patient	20 (15-30)	18 (13-31)	0.62
GHQ 28 Carer	22 (11-28)	21 (14-25)	0.91

five quality of life dimensions,⁴ with the most appropriate of three statements in each of these selected by the respondent. These responses are coded to give a single score. A visual analogue score of current health state is also recorded.

Local research ethics committee approval was granted to measure the GOSE on, and to administer the EQ-5D questionnaire to, patients attending the emergency departments of four hospitals in Manchester (United Kingdom). All patients aged over 18 presenting with a head injury of any initial severity (Glasgow coma score 3-15) within 6 hours of injury were eligible for enrolment. Patients with extracranial injuries or preinjury morbidity were not excluded. Follow up occurred at 1 month. The GOSE was measured by telephone interview and the EQ-5D by postal survey. One hundred and twenty one patients were recruited (76 men), of whom 100 (83%) had outcome assigned by GOSE, and 67 (55%) returned the EQ-5D questionnaire. Four of the second group omitted the visual analogue score assessment. The EQ-5D questionnaire only was assessed in nine patients (7%). Fifty eight patients underwent outcome assignment by both methods, and these results form the basis of subsequent statistical analysis.

A predominance of apparently minor head injury (initial Glasgow coma score (GCS) 14-15) was found in the study respondents (56/58 (97%)). The proportion of those 56 patients with more minor injuries who had less than a good recovery (GOSE<7) at one month was 23%. The two patients with GCS less than 14 experienced a good recovery.

A significant correlation between the EQ-5D score and GOSE was found (Spearman's r 0.427 $p=0.001$). The correlation between the visual analogue score reading and GOSE was also significant (Spearman's r 0.38 $p=0.005$). All 18 patients who replied "no problem" in each health state category of the EQ-5D questionnaire had a good recovery as measured by GOSE (>6) (Pearson's χ^2 7.54 $p=0.006$).

The follow up rate for the GOSE, assessed by telephone interview, was superior to the EQ-5D postal survey. A few patients, however, responded only by post. In an unselected group of patients with predominantly minor head injuries it seems that EQ-5D health status score can be used to infer a good recovery after head injury if no problems are identified in the five domains of health status.

WILL TOWNEND
Accident and Emergency Department,
Hope Hospital, Salford M6 8HD, UK
willtoen@globalnet.co.uk

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nerves and to neuropathologists, but the generalist in both specialties will find it useful. For such a heavily illustrated book it is reasonably priced at £125.

DAVID J DICK

Infectious diseases of the nervous system. Edited by LARRY E DAVIS and PETER G E KENNEDY. (Pp 544, £65.00.) Published by Butterworth Heinemann, Oxford, 2000. ISBN 0-7506-4213-0

Infections of the nervous system often present to and are managed by neurologists, rather than infectious disease physicians. A number of books on these infections have been published recently, but mainly written by infectious disease physicians, who often have a different perspective to neurological disease than neurologists. This book, edited by two neurologists and written mainly by neurologists, aims to fill this gap for practising neurologists and internists.

This book is not an exhaustive text on infections of the nervous system, but the editors have chosen infections in which they think there have been recent developments. Thus, there are chapters on prions, viruses (herpes simplex, varicella zoster, cytomegalovirus, Japanese B, HIV, and rabies), bacteria (*Borrelia*, syphilis, tuberculosis) and parasites (trypanosomiasis, malaria, *Ehrlichia* species). There are other chapters on postinfectious encephalomyelitis, vaccines against bacterial meningitis, and recurrent aseptic meningitis.

Most of the chapters are written well, structured, and with up to date and extensive references. There are some excellent clinical descriptions (for example, herpes encephalitis, prion disease, trypanosomiasis) with good neuroradiological illustrations. I found the chapters on postinfectious encephalomyelitis (although they required more illustrative images to demonstrate the range) and recurrent aseptic meningitis particularly useful. The chapter on trypanosomiasis is one of the best reviews I have read on the subject, although the length may not be justified in a book of this nature. The chapter on tuberculosis meningitis is scanty for the increasing importance of this infection. The lack of neurological details, discussion about the differential diagnosis, and management of sequelae in some chapters is disappointing, as the neurologists are often consulted about these issues.

I think that this book will be useful to neurologists with a specific interest in infectious diseases (particularly tropical diseases), but will not replace one of the more standard texts on nervous system infections.

CRJC NEWTON

BOOK REVIEWS

An Atlas of Peripheral Nerve Pathology.

By ROSALIND KING. (Pp 217, £125). Published by Arnold, London, 1999. ISBN 0-340-586664.

Whether it is because peripheral nerve has a limited repertoire of responses to pathological insults or whether it is because advances in other areas of neuropathology have sidelined interest, for many years peripheral nerve disease has been the Cinderella of that specialty. Indeed there have been very few monographs on this subject since Roy Weller first published his *Pathology of Peripheral Nerve* in 1977. Now, with an increase in knowledge of peripheral nerve disease, and advances in electron microscopy and the genetics of inherited neuropathies, the publication of this *Atlas of Peripheral Nerve Pathology* is timely.

Broadly the book is divided into two sections. In the first the author takes us through the normal light and ultrastructural appearance of peripheral nerve. There then follow chapters on biopsy techniques and importantly a section devoted to artefact. After chapters on morphometry and abnormal structure of peripheral nerve the reader is led into the second section which is devoted to pathological changes. In summary, there are sections on the pathology of the axon, myelin sheath, Schwann cell, interstitium, and vasa nervorum.

This is a handsome well illustrated book with good quality and well annotated photomicrographs, many in colour. The electron micrographs are clear and crisp. The text is well referenced and indexed. This book is primarily an atlas so it would be unfair to criticise deficiencies in the text. However a discussion on the indications for nerve biopsy with some suggested guidelines would have been useful and I think some mention should have been made of the procedure's potential complications.

This book has obvious appeal to clinicians with an interest in disease of the peripheral