Letters

Neurological competences in the graduating psychiatrist

Sir: Graduates presenting for specialist examinations in psychiatry are expected to manifest competency in clinical neurology. For those concerned, certain pertinent questions arise: how important is this requirement, in practical terms; what should this competency comprise; and how best can such competency be attained? In an attempt to address these issues, the views of specialist psychiatrists were solicited by questionnaire.

The questionnaire comprised three parts and was mailed to 184 of the 196 psychiatrists

listed in the 1981 South African Medical and Dental Council register. Part 1 included items relating to professional background. In Part 2, specialists were asked to rate 38 items of neurological competence as either "essential", "desirable" or "not required" in the graduating psychiatrist (table). These categories were assigned values of 2, 1 and 0, respectively. Part 3 sought comments on the design of neurology training programmes for psychiatrists. Of the 120 (65%) respondents, 85 were hospital affiliated, 51 practised privately and 16 resided abroad. Aggregated ratings for the 38 items of competence are shown in the table. The overall mean rating score was 69.5% (SD15; range, 19-100) with 110 (90%)

respondents recording a score of 50% or more. There was no significant difference ($p > 0.05$, Student *t* test) between mean rating score of psychiatrists whose neurology training had been more (71.5%, $n = 88$) or less (65.5%, $n = 24$) than 3 months, or who had (70.5%, $n = 40$) or had not (69%, $n = 80$) assumed clinical responsibility. Replies to Part 3 of the questionnaire were recorded by 60 respondents and are reported in detail elsewhere.²

The results of this study affirm that a certain proficiency in clinical neurology is integral to a training in psychiatry. That this opinion is shared by psychiatrists whether or not clinical neurology had featured prominently in their own training adds to its validity. The study has also identified those clinical neurological competences most relevant to professional psychiatric practice. For the neurologist, as teacher and examiner, such data offer a sound basis for defining educational objectives, thereby allowing programme contents to be rationally determined, teaching methods to be selected and pertinent assessment procedures to be devised.³

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Table: Part 2 of questionnaire, with aggregated ratings of the 120 psychiatrists expressed as percentages*

Item	Neurological competence	Essential	Desirable	Not required
1	Elicit history, perform neurologic examination	85	15	0
2	Interpret abnormal physical signs	67.5	31.5	1
3	Evaluate apraxia, agnosia, aphasia, disorders of memory	70	27.5	2.5
4	Differentiate physical disability from simulation	91	9	0
<i>Recognise characteristic presentations</i>				
5	Intrinsic, extrinsic myelopathies	20.5	64	15.5
6	Motor neuron disease	33	53	14
7	Cranial, peripheral neuropathies	34.5	58	7.5
8	Nerve root syndromes	29	54	17
9	Myasthenia gravis	41.5	51	7.5
10	Polymyositis	22.5	55.5	22
11	Muscular dystrophies	22.5	55.5	22
<i>Judgement, evaluation, management</i>				
12	Transient loss of consciousness, coma	85	14	1
13	Headache, facial pain	70.5	28	1.5
14	Dizziness, vertigo	63	33.5	3.5
15	Pain syndromes	56.5	40	3.5
16	Involuntary movements	67	32	1
<i>Specified areas of knowledge</i>				
17	Cranio-cerebral trauma	63	33.5	3.5
18	The epilepsies	89	11	0
19	The dementias	96	4	0
20	Cerebrovascular disorders	67.5	31.5	1
21	Parkinsonism	70	29	1
22	Multiple sclerosis	49.5	43.5	7
23	CNS infections	52.5	43.5	4
24	Metabolic encephalopathies	56	40.5	3.5
25	Sleep disorders, narcolepsy	75	24	1
26	Drugs in common use	82.5	17.5	0
27	Existence less common disorders	6	70	24
28	Awareness research trends	12.5	67	20.5
29	Inheritance pattern common hereditary disorders	27.5	56	16.5
<i>Special investigations</i>				
30	Skull X-ray: basic principles interpretation	57	29	14
31	EEG: features normal record	50.5	43	6.5
32	Isotope encephalogram: basic features normal patterns	21.5	55	23.5
33	CT brain scan: basic features normal appearance	29	48	23
34	Lumbar puncture: indications contraindications	75	21.5	3.5
35	Rationale for ordering: Electromyography	15	45	40
36	Caloric responses	16	41.5	42.5
37	Visual evoked potentials	16	49	35
38	Auditory evoked potentials	11.5	53.5	35

*To nearest 0.5%

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Benign unilateral seizures or epilepsy?

Sir: A benign form of "hemiconvulsive epilepsy with hemiplegia" has been recently reported in children.¹ We describe a patient who presented with a unilateral status epilepticus in childhood and an isolated relapse after a 17 year seizure- and drug-free interval.

A 21½-year-old left-handed man with no family history of major disease was born after a normal pregnancy with vertex delivery and normal neo-natal psychomotor development. At the age of 2½ months he developed a non-febrile unilateral left tonic-clonic status epilepticus (see fig A).

At this time, a right carotid arteriography showed indirect signs of cerebral oedema, particularly in the frontal lobe. CSF examination was normal, but blood tests indicated hypocalcaemia (1.65 mmol/l). Correction of the hypocalcaemia and treatment with phenobarbitone (8 mg/kg/day) stopped the unilateral status after twenty days. Anticonvulsant therapy was pursued for one year, then was discontinued. Several neuropsychological studies, blood tests and EEGs in the following two years were normal.

At the age of 17 years, the morning following a very stressful night, he presented with an isolated, brief (two-minute) left clonic seizure affecting the face and the upper and lower limbs. There was no loss of consciousness, but a post-ictal aphasia and a left hemiparesis were evident for twenty minutes. An EEG recording was performed one hour after the seizure (see fig B). Phenobarbitone treatment (3 mg/kg/day) was immediately initiated and discontinued after two years. Several clinical and EEG examinations in the four-years of follow-up after the relapse were normal, and no further seizures occurred. A CT scan carried out at the age of 21½ years also was normal.

The clinical evolution of children presenting with unilateral seizures, followed or not by residual hemiplegia, has been dis-

cussed by a few authors.²⁻⁵ It can be estimated that about 25% of children who have had unilateral seizures do not have further attacks, but about 14% will develop a benign epilepsy with rolandic spikes.⁵ The possibility of a later "epilepsy" characterised only by infrequent unilateral seizures, with or without post-ictal transient hemiplegia, appearing after a long free-interval, has been mentioned⁶ and two cases have also been described.¹

Our patient presented in early infancy

with a prolonged unilateral status epilepticus (perhaps facilitated by hypocalcaemia) which recovered without evident anatomical brain damage (normal CT scan). It is interesting to note the absence (see fig A) of the post-ictal "silent period" which reportedly has a bad prognosis.^{7,8} A second isolated seizure, affecting the same side of the body, appeared after a 17 year free interval. This relapse, probably related to stress in the preceding night, seems to attest to a low threshold or "predisposition to convulsion" of his right (dominant) hemisphere. The absence of further seizures during the four years of follow-up (despite no anticonvulsant treatment in the last two years) supports the benign nature of this condition, which seems to be closely related to the other "benign or functional" epilepsies.⁹ It must be emphasised, nevertheless, that "unilateral epilepsy" is not included in the International Classification of the Epilepsies,¹⁰ and that a recently proposed Classification of Epileptic Seizures¹¹ does not even consider "Unilateral Seizures", although this is not universally accepted.¹² We want to call attention to the existence of this kind of seizure which, as in our patient, may recur and have a benign evolution. Guided by such considerations, the question arises to whether a rare form of "Unilateral Benign Epilepsy" exists.

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Fig (A) Two-minute period of awake EEG recording during the unilateral seizure state. Note the rhythmic discharge of spikes starting from the posterior right region and spreading to the whole hemisphere (A and B); deviation of the eyeballs and head to the left and tonic extension of the upper and lower limbs were noted. Left hemiconic jerks subsequently were seen in C (diffuse right-sided spike-and-wave discharges), whereas loss of consciousness, vomiting and left hemiplegia were evident in D (diffuse right-sided slowdown, more evident in the central region). (B) EEG recording one hour after the ictal relapse: slow background activity of the whole right hemisphere, more evident spikes in the fronto-central region.

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One-and-a-half syndrome due to polycythaemia

Sir: The "One-and-a-half" syndrome is a peculiar lateral gaze palsy. It combines two defects of horizontal eye movements: a lateral conjugate gaze palsy in one direction (one) and, in the opposite direction, a paralysis of adduction by one eye resulting from an internuclear ophthalmoplegia (and-a-half). Thus, one eye cannot deviate laterally from the midline and the other eye can only be abducted from the midline. The first reported clinicopathological case is that of Fisher.⁴ We have found 22 cases in the literature, with several causes: five have been proven and five were probable infarctions, two gliomas, two astrocytomas, one arteriovenous malformation, three haemorrhage, two probable cases of multiple sclerosis, one ependymoma and one metastatic tumour.¹⁻⁶ We report a case of the one-and-a-half syndrome secondary to polycythaemia.

A 63-year-old man presented with horizontal diplopia. Two years earlier he had a hemiparesis and sensory changes of the right side of the body which regressed in a few days. Examination did not reveal abnormalities other than oculomotor disturbances. With any type and any direction of lateral eye movements, the left eye remained fixed on the midline, while the right eye could only abduct from and return to the midline. In the vertical upward gaze, the right eye showed skew deviation with vertical divergence. Convergence was preserved. The pupillary reflexes were full. Laboratory studies showed: white-cell count was 11·840/mm³, red-cell count was 6·240·000/mm³, Hb 19·8 mg/dl., HCT 58·9% MCV 89%, MCH 30·7%, MCHC 33·7% the platelets count was 311·000/mm³. With the ⁵¹Cr method, the circulating blood volume was found to be increased. A computed tomography scan showed an area of low density in the left parietal region with normal ventricular size; with contrast there were no changes. Chest radiographs were normal. Both carotid and basilar angiograms and ECG were normal. The EEG showed slight slowing in the left temporal area. Intravenous pyelograms revealed two left renal cysts. Two phlebotomies of 400 ml were made. One week later there was complete recovery of the ocular disorder. Two weeks after admission the patient was discharged from hospital with dipyrindamole treatment.

The "one-and-a-half" syndrome usually results from a single, unilateral and relatively small lesion of the dorsal tegmentum in the lower pons.^{1,4} At first the abducens nucleus (VI) and/or the pontine reticular formation are damaged by this lesion and an ipsilateral conjugate gaze palsy is produced. Next damage by the same lesion to the ipsilateral medial longitudinal fasciculus, which lies at the same level or just above the abducens nucleus, accounts for unilateral internuclear ophthalmoplegia. The causes of the syndrome are varied.¹⁻⁶ In our patient, the "one-and-a-half" syndrome resulted from a probable cerebral thrombosis in the vertebral-basilar system secondary to polycythaemia, a condition in which the liability to cerebral thrombosis is increased.^{7,9} As far as we know, the present case is the first description of a "one-and-a-half" syndrome secondary to polycythaemia, a potentially reversible cause.

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Corynebacterium pyogenes meningitis

Sir: *Corynebacterium pyogenes* is a gram positive rod which is associated with suppurative or granulomatous lesions in various domestic animals with a few reported cases implicating human disease. We report what is believed to be the first case of *C. pyogenes* meningitis.

A 76-year-old man was admitted to hospital in a confused state. Over the previous twelve hours he had complained of severe headache, nausea and vomiting. His temperature was 38·0°C; pulse 98/min; blood pressure 180/110/mm Hg; respiration 24 per minute, and he responded slowly to commands. There was neck stiffness and photophobia with no other neurological signs. Acute meningitis was diagnosed and confirmed by lumbar puncture; (table). Chloramphenicol was prescribed as no organisms were identified on direct micro-