Downloaded from jnnp.bmj.com on November 22, 2012 - Published by group.bmj.com

PostScript

CORRESPONDENCE

Neuropsychological and psychiatric complications in endoscopic third ventriculostomy

We read with interest the recent paper by Benabarre *et al*[1] of the first reported case of endoscopic third ventriculostomy followed by severe psychiatric complications.

In our department, we also had a patient who developed severe psychiatric symptoms after an endoscopic third ventriculostomy (ETV).

A 45 year old woman with an aqueductal stenosis underwent an EVT because of progressive gait and visual disturbances. In November 1997 she underwent an ETV through a right side precoronal burr hole using a rigid neuroendoscope. The third ventricular floor was perforated with a 4 French Fogarty catheter, the perforation being enlarged with the inflatable balloon. No problems were encountered during the procedure, although we noted an incomplete septum pellucidum. After ETV her gait and visual disturbances gradually resolved. However, after the procedure the patient was nervous and agonised, and she complained of a crepitating sound in her head and behaved aggressively towards her spouse. Because her complaints and behaviour worsened a psychiatric evaluation was performed. Psychotic depression was diagnosed and three weeks after the EVT she was admitted to the department of psychiatry. For several months she was treated with antipsychotic and antidepressant drugs and her psychotic depression partially resolved. She is still being treated for mild depression. Postoperative magnetic resonance imaging six weeks, three months, and one year after the EVT showed no normalisation of the ventricular system, but no other abnormalities were seen.

Signs and symptoms were abrupt and probably organic because of the apparently strong relation between the procedure and the start of the psychotic depressive episode. Previously, the patient had no psychiatric complaints and had undergone other invasive procedures under general anaesthesia and admittance to the hospital. It is not clear how this psychotic depression after EVT can be explained. Sometimes when ETV is performed, injury of the fornix is seen. The fornix constitutes the sole efferent system from the hippocampus and both are involved in the limbic system. The limbic system has an important role in mood and emotional behaviour. We hypothesize that in this patient a combination of incomplete septum pellucidum and an injury of the fornix may have caused an organic personality syndrome after EVT. We agree with Benabarre *et al* that clinicians should be aware of and take into account this potential serious complication of EVT in this so called minimally invasive procedure.

J van Aalst, E A M Beuls
Department of Neurosurgery, University Hospital Maastricht, P Debyelaan 25, Postbus 5800, 6202 AZ Maastricht, The Netherlands

G J Luijckx
Department of Neurology, University Hospital Groningen, Hanzeplein 1, PO Box 30001, 9700 RB Groningen, The Netherlands

Correspondence to: Dr J van Aalst; jaspervanaalst@hotmail.com

Reference

1 **Benabarre A**, Ibanez J, Boget T, *et al*. Neuropsychological and psychiatric complications in endoscopic third ventriculostomy: a clinical case report. *J Neurol Neurosurg Psychiatry* 2001;**71**:268–71.

Authors' reply

We are satisfied with the clinical interest generated by our case report where we explained the first case of endoscopic third ventriculostomy followed by severe psychiatric complications.

Van Aalst *et al* have reported a patient who developed severe psychiatric symptoms after an endoscopic third ventriculostomy. Their patient underwent an endoscopic third ventriculostomy through a right sided precoronal burr hole using a rigid neuroendoscope. In our case the patient underwent surgery under general anaesthesia and a 6.5 mm rigid neuroendoscope (Gaab Endoscope, Storz, Tutlingen, Germany) was inserted through a right sided precoronal burr hole in the frontal horn of the right lateral ventricle, following the technique described by Vries[1] and Viñas *et al*.[2] Fenestration of the floor of the third ventricle to the basal cisterns was performed with bipolar coagulation and enlarged with a 3 French Fogarty catheter. The surgical technique was similar in both cases.

Both patients behaved aggressively but ours developed a severe complication consisting of an organic personality disorder characterised by impulsiveness, physical heteroaggressiveness, binge eating, hypersomnia, and impaired memory and frontal executive functions. In our discussion we postulated that a frontal lobe lesion may explain some of the symptoms presented such as the uncontrolled impulses, the aggressive behaviour, and even the binge eating. However, a longitudinal neuropsychological evaluation showed a severe deficit in immediate memory and difficulties in planning and consolidation of newly learned information, which may be best related to damage in the frontal-basal structures of the brain: the fornix and its connection to the hippocampus and the mamillary bodies. Postoperative magnetic resonance images confirmed the clinical hypothesis.

We do not totally agree with the diagnosis postulated by Aalst *et al*. Is true that signs and symptoms were abrupt and probably organic because of the apparently strong relation between the procedure and the start of the psychotic depressive episode. However, we think that an organic personality syndrome cannot be conceptually diagnosed after EVT because the correct *Diagnostic and statistical manual of mental disorders* category may be a mental disorder not otherwise specified due to a general medical condition.[3] This residual category should be used for situations in which it has been established that the disturbance is caused by the direct physiological effects of a general medical condition but where the criteria are not met for a specific mental disorder due to a general medical condition (such as dissociative symptoms due to complex partial seizures). The diagnosis of an organic personality syndrome requires that the patient suffer a persistent personality disturbance that is a change from the person's previous characteristic personality pattern. Eight specific types are described: labile type, disinhibited type, aggressive type, apathetic type, paranoid type, other type, combined type, and unspecified type. We think that the patient described by Van Aalst *et al* did not meet all of these diagnostic criteria.

We think it would be very interesting to know more of the clinical aspects of the patient. For example, had this patient suffered from other depressed states during her life? The patient's problem may have been a severe recurrence of a depressive disorder.

We agree that these clinical cases should help clinicians to take these potential complications of endoscopic third ventriculostomy into account before indicating this so called minimally invasive procedure.

A Benabarre, T Boget, E Vieta, A Martínez-Aràn
Institut Clínic de Psiquiatria I Psicologia, Hospital Clínic de Barcelona, Facultat de Medicina de la Universitat de Barcelona, C/ Villarroel 170, 08036 Barcelona, Spain

Correspondence to: Dr A Benabarre; 31555abh@comb.es

References

1 **Vries JK**. An endoscopic technique for third ventriculostomy. *Surg Neurol* 1978;**9**:165–8.
2 **Viñas FC**, Dujovny N, Dujovny M. Microanatomical basis for the third ventriculostomy. *Minim Invasive Neurosurg* 1996;**39**:116–21.
3 **American Psychiatric Association**. *Diagnostic and statistical manual of mental disorders*, fourth edn. Washington: The Association, 1994.

Neuropathological findings in multiple system atrophy with dystonia

Boesch *et al*,[1] reporting their experience with dystonia in multiple system atrophy (MSA), observed in 40% of untreated patients with anterocollis and unilateral limb dystonia representing the most frequent forms. All were MSA-P cases. Discussing the pathophysiology of dystonia, they quoted previous neuropathological studies attributing anterocollis to neuronal loss in the ventral putamen.[2] In five of their 24 patients, four of whom were listed as levodopa responsive MSA-P, neuropathology confirmed the diagnosis of MSA, but the degree of degeneration varied considerably. As the neuropathology findings could not be included into this paper, these and other morphological data will be briefly discussed. In case 14 (woman aged 57 with four years duration) with unilateral end of dose focal dyskinesias, there were moderate fibrillary gliosis with minimal neuronal loss in the dorsolateal putamen and widespread glial cytoplasmic inclusions, considerable focal cell loss in the the caudal ventrolateral parts of the substantia nigra pars compacta (SNC) and locus coeruleus without Lewy bodies or

Downloaded from jnnp.bmj.com on November 22, 2012 - Published by group.bmj.com

pontine involvement, corresponding to MSA-P grade II.[3 4] Case 18 with five years duration of illness and craniocervical peak dose dystonia, showed similar mild degeneration of the dorsolateral putmen and SNC (grade II), while cases 2 and 16 (disease duration four and five years, respectively) with generalised peak dose dystonia, histopathologically revealed subtotal neuronal loss and gliosis in the dorsolateral and, much less, in the anterior putamen and caudate nucleus associated with mild gliosis in the globus pallidus and degeneration of the SNC, more severe in the dorsolateral middle and caudal than in the medioventral and rostral parts, corresponding to MSA-P grade III.[3 4] A review of necropsy cases of MSA with dystonia in the literature and eight personal cases gave the following results: among eight patients with anterocollis, five were MSA-P, and three MSA-C, all of the former with severe degeneration of the putamen, more severe in dorsolateral than ventral parts, and the SNC were all grade III. Among 28 cases with spastic and generalised dystonia, 21 were MSA-P and eight MSA-C. Twenty six brains revealed severe degeneration of the putamen, 12 of the caudate nucleus, seven of the globus pallidus, and all except for one had severe, rather diffuse involvement of the SNC. Only in single cases, the thalamus, corpus subthalamicum and other brain stem nuclei, for example, locus coeruleus, reticular SN, arcuate nucleus, etc, were involved. These data indicate that dystonia is more frequent in MSA-P than in MSA-C, in most but not all cases related to severe degeneration of the dorsolateral putamen and SNC with less involvement of the ventral putamen. While in MSA with spastic dystonia both the putamen and SNC appear equally involved, in MSA with dystonic contractures, the putamen appears more often affected than the SNC, whereas other subcortical and brain stem nuclei and the pontocerebellar system are only affected in single cases. This confirms previous studies on focal dystonia emphasising the role of the putamen as a major site of dysfunction[5 6] with impairment of the basal ganglia circuitry.[7] However, in view of the variability in the intensity and distribution of the neuropathological lesions in MSA with dystonia, further studies are needed for the elucidation of their pathophysiological basis.

K A Jellinger
Institute of Clinical Neurobiology, 7 Kenyougasse, 1070 Vienna, Austria

Correspondence to: Dr K Jellinger; jellinger@univie.ac.at

References

1 **Boesch SM**, Wenning GK, Ransmayr G, *et al*. Dystonia in multiple system atrophy. *J Neurol Neurosurg Psychiatry* 2002;**72**:300–3.
2 **Fearnley JM**, Lees AJ. Striatonigral degeneration: a clinicopathological study. *Brain* 1990;**113**:1823–42.
3 **Jellinger KA**, Wenning GK, Seppi K, *et al*. A novel grading scale for striatonigral degeneration. *Ann Neurol* 2001;**56** (suppl 1):S49.
4 **Wenning GK**, Seppi K, Tison F, *et al*. A novel gradig scale for striatonigral degeneration (multple system atrophy). *J Neural Transm* 2002;**104**:307–20.
5 **Perlmutter JS**, Stambuck MK, Markham J, *et al*. Decreased [18F] spiperone binding in putamen in idiopathic focal dystonia. *J Neurosci* 1997;**17**:843–50.
6 **Levy LM**, Hallet M. Impaired brain GABA in focal dystonia. *Ann Neurol* 2002;**51**:93–101.
7 **Berardelli A**, Rothwell JC, Hallett M, *et al*. The pathophysiology of primary dystonia. *Brain* 1998;**121**:1195–212.

Social phobia in spasmodic torticollis: some conceptual issues

Gundel *et al*[1] tackle an important issue of social phobia (SP) in patients with spasmodic torticollis (ST). This is because social avoidance itself contributes to a significant degree of distress and dysfunction in patients with chronic medical conditions.[2] Also, potentially beneficial pharmacological and psychosocial treatments remain undelivered because of under recognition of this problem. Keeping these aspects in mind, we would like to highlight certain conceptual and methodological issues.

Firstly, the sample was restricted to patients with ST who required treatment with botulinum toxin. Also, the severity (Tsui) score for the sample has not been mentioned. Hence the sample may not be representative of all patients with ST.

Secondly, life events were assessed for the one year period before the initial manifestation of ST. As per the authors the mean (SD) duration of illness, at time of assessment, was 11.9 (11.3) years. Although life events were reported in 50% of patients, yet caution should be exercised in relation to this result as elicitation of life events is associated with problems in recall, especially in illnesses of long duration.

Thirdly, 80% of patients with SP were classified as reactive. Though the authors mention that social anxiety had occurred after onset of ST, they do not provide data on the duration of SP, and hence fail to demonstrate a temporal relation between onset of symptoms of SP and ST. Related to this is the issue of primary/secondary/tertiary psychiatric comorbidity. Comorbidity as a concept has different dimensions.[3] As this classification is not followed in DSM- IV,[4] it would be helpful if the authors provide essential details for a better understanding of the same. The authors postulate a subgroup of patients with SP who have symptoms of social anxiety secondary to ST—that is, presence of a probable cause-effect relation. But, this brief report fails to mention as to how this subgroup was identified. Indeed, as is known, to be classified as "organic psychiatric disorders", certain diagnostic criteria need to be fulfilled in both ICD-10 [5] and DSM-IV.[4] Although SP may not be taken as "organic" in origin, yet labelling it as secondary to (or arising out of) ST requires further evidence.

Fourthly, the authors make an important assertion that DSM-IV[4] excludes patients with social anxiety secondary to medical conditions, and this is without empirical basis. In fact, there seems to be no place for such patients with social anxiety/phobia in DSM-IV. On the other hand, ICD-10[5] diagnostic criteria do not preclude against the diagnosis of SP in patients with physical conditions. It seems that ICD-10 is more broad based and less conservative than DSM-IV in terms of identifying various types of psychiatric morbidity in the physically ill. Hence, it may be more appropriate to use ICD-10 for such patients until the DSM classificatory system resolves this issue in greater detail with availability of the current empirical evidence.[1]

Lastly, though the authors adopted a rigorous methodology for determination of diagnosis, yet it seems surprising that no patient was assessed for Axis II diagnosis; anxious–avoidant personality disorder (AAPD). AAPD is the most common differential diagnosis for SP and it has been shown that differentiating it from SP is difficult.[4 6] Additionally, patients with SP can have premorbid anxious traits (or AAPD) making personality an important comorbid issue.[6 7] This conceptual and diagnostic overlap needs to be kept in mind before giving a definitive diagnosis of SP as the treatment is influenced to a great degree by the diagnosis. Hence, there is a need to evaluate for an additional or alternative diagnosis of AAPD in patients with SP.

Overall, although, this study provides a database on psychiatric morbidity, especially SP, in patients with ST but the conceptual issues related to diagnosis of SP need to be critically considered.

P Sharma, N Gupta
Department of Psychiatry, PGIMER, Chandigarh-160012, India

Correspondence to: Professor N Gupta; tinysam@glide.net.in

References

1 **Gundel H**, Wolf A, Xidora V, *et al*. Social phobia in spasmodic torticollis. *J Neurol Neurosurg Psychiatry* 2001;**71**:499–504.
2 **Oberlander EI**, Schneir FR, Liebowitz MR. Physical disability and social phobia. *J Clin Psychopharmacol* 1994;**14**:136–43.
3 **Wittchen HU**, Essau CA. Comorbidity of anxiety disorders and depression: Does it affect course and outcome? *J Psychobiol* 1989;**4**:315–23.
4 **American Psychiatric Association**. *Diagnostic and statistical manual of mental disorders*. 4th edn. Washington, DC: American Psychiatric Press, 1994.
5 **World Health Organisation**. *The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organisation, 1992.
6 **Lampe LA**. Social phobia: a review of recent research trends. *Curr Opin Psychiatry* 2000;**13**:149–55.
7 **Cloninger CR**. Svrakie DN. Personality disorders. In: Sadock BJ, Sadock VA, eds. *Comprehensive text book of psychiatry*. 7th edn. Philadelphia: Lippincott Williams and Wilkins, 2000:1723–64.

Authors' reply

(1) We agree with Sharma and Gupta in that our results are based on spasmodic torticollis (ST) patients treated with botulinum toxin, although in untreated patients social phobia may be even more severe. Cultural and other biases could also play a part. The mean Tsui Score of our sample was 7.58, SD 3.77, range 2–26.

(2) There is no perfect method for retrospectively measuring stressful life events. Data should therefore be interpreted with the appropriate caution. Because of the relatively small point prevalence ration of 5.4 per 100 000 in cervical dystonia,[1] it would be methodologically virtually impossible to investigate this issue in a longitudinal study.

(3) We used the questions F41–F50 for the diagnosis of social phobia when conducting the SCID interview.[2 3] If criteria A-G or A-H were met by a participant, she/he was additionally asked if these symptoms had initially occurred before or after the manifestation of ST and what had been the main reason for developing social phobia from the patients' view. Based on the patient's answer and the clinical judgment of the interviewer, social phobia was classified as reactive or not reactive to ST.

We did include the data about the duration of social phobia in our data file when social phobia was diagnosed as the clinically most important psychiatric diagnosis. Of these n=48 patients (41.3%), the mean duration of

Downloaded from jnnp.bmj.com on November 22, 2012 - Published by group.bmj.com

ST was 12.4 (SD 10.8) years and the mean duration of social phobia was 11.2 years (SD 11.3). Within this sample of 48 patients, three patients reported an onset of social phobia before the onset of ST. Mean duration of social phobia in these three patients was 3, 33, and 38 years.

(4) In accordance with studies on social phobia in adult stutterers[4 5] and essential tremor,[6] we used DSM-IV criteria.[3] ICD-10 does not preclude against the diagnosis of social phobia in patients with chronic somatic disease and thus may conveniently be used to classify this condition in patients with disfiguring or disabling physical conditions.

(5) The issue on anxious-avoidant personality disorder (AAPD) as differential diagnosis to social phobia is very interesting and should be explored in future studies. AAPD before the diagnosis of ST may indicate increased vulnerability of ST patients to develop social phobia or psychiatric disease.

The points raised by Sharma and Gupta are valuable and should be taken into account in further research on the issue of social phobia in ST. Disturbed body image and negative self referent cognitions with the consequence of developing social phobia and/or a feeling of stigma are clearly a main psychological problem in a large subgroup of ST patients (see, for example, Papathanasiou *et al*[7]).

H Gündel
Department of Psychosomatic Medicine, Klinikum rechts der Isar, TU München, Langerstr 3, 81675 München, Germany

A O Ceballos-Baumann
Department of Neurology, Klinikum rechts der Isar

Correspondence to: Dr H Gündel; H.Guendel@lrz.tu-muenchen.de

References

1 **Castelon Konkiewitz E**, Trender-Gerhard I, Kamm C, *et al*. Service-based survey of dystonia in munich. *Neuroepidemiology* 2002;**21**:202–6.
2 **Wittchen HU**, Wunderlich U, Gruschwitz S, *et al*. *Strukturiertes klinisches Interview für DSM-IV, Achse I (SKID)*. Göttingen: Hogrefe, 1997.
3 **American Psychiatric Association**. *Diagnostic and statistical manual of mental disorders*. 4th edn. Washington, DC: American Psychiatric Association, 1994.
4 **Oberlander EI**, Schneier FR, Liebowitz MR. Physical disability and social phobia. *J Clin Psychopharmacol* 1994;**14**:136–43.
5 **Stein MB**, Baird A, Walker JR. Social phobia in adults with stuttering. *Am J Psychiatry* 1996;**153**:278–80.
6 **George MS**, Lydiard RB. Social phobia secondary to physical disability. A review of benign essential tremor (BET) and stuttering. *Psychosomatics* 1994;**35**:520–3.
7 **Papathanasiou I**, MacDonald L, Whurr R, *et al*. Perceived stigma in spasmodic torticollis. *Mov Disord* 2001;**16**:280–5.

Executive dysfunction and depressive symptoms in cerebrovascular disease

The article by Kramer *et al*[1] suggests that subcortical ischaemic vascular disease is associated with subtle declines in executive functioning and visual memory, even in non-demented patients. The authors compared 27 control subjects and 12 non-demented patients, who were selected after exclusion of major depression, bipolar affective disorder, and other DSM-IV I axis disorders. We wish to contribute with personal data to this topic suggesting that, even in absence of a clinical diagnosis of depression, depressive symptoms may modulate executive dysfunctions in non-demented subjects with cerebrovascular disease.

We examined 34 consecutive patients with cognitive impairment-no dementia (CI-ND) (mean (SD), age: 78.1 (6.3), range 65–90; years of education: 4.9 (1.7), range 3–10; Mini Mental State Examination score: 24.0 (2.4), range 18–27). The diagnosis of CI-ND was made on the basis of a standardised multidimensional protocol including history, clinical examination, detailed neuropsychological testing, and computed tomography. The presence and severity of cortical, white matter, and deep subcortical lesions and of leukoaraiosis were assessed on computed tomographic film with a standardised visual rating scale.[2] With this method, the patients were quantitatively divided in two groups (50th centile) according to the severity of cerebrovascular disease: 17 patients had none or mild, and 17 had moderate or severe cerebrovascular disease. The two groups had similar age (mean (SD), age: 79.1 (6.5) and 76.9 (6.0); p=0.31, *t* test), educational level (mean (SD), years of schooling: 4.7 (1.5) and 5.3 (1.9); p=0.27 *t* test), cognitive impairment (mean (SD), Mini Mental State Examination score: 24.2 (2.7) and 23.8 (2.0); p=0.63 *t* test), functional status (mean (SD), Barthel Index: 85.3 (18.9) and 80.8 (21.4); p=0.57, *t* test), and comorbidity (mean (SD), Charlson Index: 2.1 (2.2) and 2.2 (1.6); p=0.95, *t* test). Comparing the neuropsychological tests, we found that the patients in the group with none or mild cerebrovascular disease performed better on Babcock (mean (SD), score: 11.3 (2.3) and 8.6 (3.0); p=0.01, *t* test), on digit symbol (mean (SD), associations in 90 seconds: 14.3 (6.8) and 11.7 (4.9); p=0.24, *t* test), on trail making A (mean (SD), seconds: 143.0 (82.6) and 228.2 (157.7); p=0.05, *t* test), but were significantly less depressed (mean (SD) number of symptoms on Center of Epidemiological Studies depression scale: CES-D, 10.8 (7.2) and 18.8 (6.6); p=0.002, *t* test). However, when the effect of cerebrovascular severity on all significant variables was weighted in a multivariate linear regression model, only depressive symptoms maintained the statistical significance (CES-D: β 0.82; 95% confidence intervals, 1.2 to 4.5; p=0.02).

Several studies have shown that depressed patients have a lower performance than non-depressed ones on executive functions.[3] Furthermore, the relation between cerebrovascular disease and depression has recently been established.[4] In their study, Kramer and colleagues performed an extensive neuropsychological evaluation, but failed to take into consideration that depressive symptoms may be detected in elderly population even after exclusion of major depression. On the contrary, we suggest that depressive symptoms need to be considered in the interpretation of even subtle executive dysfunctions in cerebrovascular disease patients.

G Bellelli, E Lucchi
"Ancelle della Carità" Hospital, Cremona, and Geriatric Research Group, Brescia, Italy

G Cipriani
Geriatric Research Group, Brescia, Italy

G B Frisoni
Laboratory of Epidemiology and Neuroimaging IRCCS "San Giovanni di Dio Fatebenefratelli", Brescia, and Geriatric Research Group, Brescia, Italy

M Trabucchi
Geriatric Research Group, Brescia, and University "Tor Vergata", Roma, Italy

Correspondence to: Dr G Bellelli, "Ancelle della Carità" Hospital, via Aselli 14, 26100 Cremona, Italy; bellelli-giuseppe@poliambulanza.it

References

1 **Kramer JH**, Reed BR, Mungas D, *et al*. Executive dysfunction in subcortical ischaemic vascular disease. *J Neurol Neurosurg Psychiatry* 2002;**72**:217–20.
2 **Frisoni GB**, Beltramello A, Binetti G, *et al*. Computed tomography in the detection of the vascular component in dementia. *Gerontology* 1995;**41**:121–8.
3 **Channon S**, Green PS. Executive function in depression: the role of performance strategies in aiding depressed and non-depressed participants. *J Neurol Neurosurg Psychiatry* 1999;**66**:162–71.
4 **Alexopoulos GS**, Meyers BS, Young RC, *et al*. 'Vascular depression' hypothesis. *Arch Gen Psychiatry* 1997;**10**:915–22.

Author's reply

We completely agree with Dr Bellelli and his colleagues about the importance of considering depressive symptoms when evaluating executive and other cognitive abilities in subjects with cerebrovascular disease. It is increasingly clear that subcortical-frontal ischaemic vascular disease, symptoms of depression, and executive impairment are related, although the nature of these relations is complex and not yet fully understood. Their data on patients with mild cognitive impairment amply illustrate the need to consider depression and other moderator variables when evaluating cognitive dysfunction.

We would like to emphasise two points. Firstly, data on depression related symptoms (for example, depressed mood, anhedonia, guilt feelings, etc) were collected on all but two cases in our sample. Although we did not report these data in our paper, there were no correlations between any of our executive measures and depressive symptoms. In addition, when we used linear regression to simultaneously consider depression and subcortical lacunes, only the presence of subcortical lacunes predicted executive functioning.

Our second point is to emphasise that while linear regression is a powerful technique for considering multivariate models, researchers must not confuse correlation with causation. For example, in the analyses by Bellelli *et al*, depression but not cerebrovascular severity remained in the model predicting performance on executive tasks. This cannot be interpreted to imply that the executive impairment in cerebrovascular patients is caused by their depression and not by their cerebrovascular disease. Cerebrovascular disease might cause both depression and executive impairment. Because cerebrovascular disease, depression, and executive impairment are all related, however, it would not be unusual for one of these variables to be excluded from a regression model.

A causal relation between subcortical-frontal neuropathology and deficits on frontally mediated executive tasks makes sense neuroanatomically. Elucidation of the underlying mechanisms (for example, white matter

Downloaded from jnnp.bmj.com on November 22, 2012 - Published by group.bmj.com

disease versus lacunar volume versus strategic lacunes versus cortical microvascular change) and the precise behaviour consequences (for example, processing speed; working memory; set maintenance; inhibition) remains a high research priority.

J Kramer
University of California San Francisco, Department of Psychiatry and Langley Porter Psychiatric Institute, 401 Parnassus Avenue, Box PAC-0984, San Francisco, CA 94143–0984;
kramer@itsa.ucsf.edu

BOOK REVIEWS

Clinical neurology

By David Greenberg, Michael Aminof, and Roger Simon (Pp 390 $39.95). McGraw-Hill Professional, New York, 2002. ISBN 0-07-137543-0

Reviewing an undergraduate text is always quite a difficult task in that one has to approach one's speciality again through fresh and innocent eyes asking the simple question, would I recommend this? The book, in its fifth edition, claims to present a "problem oriented approach" that has been founded on the authors' experience in teaching both undergraduate students and junior medical staff at the University of California (San Francisco). The changeover in our own universities to this teaching approach made me believe that this would be a particularly enlightening and instructive read. I was especially interested to see how the presentation of basic essential knowledge (neuroanatomy, physiology, molecular pathogenesis) fitted into such a schema. I am afraid, in the end, that I was disappointed to find the book not dissimilar to the majority of recommended "standard" undergraduate texts. While each chapter follows the similar template of an initial approach to diagnosis, what follows is variable and not what I would regard as problem oriented. For example, while the section on headache does list modes of presentation (acute, subacute, and chronic), other chapters, such as "Disorders of somatic sensation" or "Motor deficits", present the usual long lists of conditions without balance (what is common and what is rare), encouraging a logical approach to symptom interpretation or developing an appropriate investigative pathway. The book does have its good points in that it is comprehensive with well defined key concepts at the beginning of each section and a comprehensive lists of references at the end but the therapeutics are patchy, the glossary of frequently used drugs unhelpful, and the account of neurological investigations inadequate. I would not recommend this as an addition to our current undergraduate reading list, as there are better texts available.

While any text entering its fifth edition has proved its worth to many and clearly serves the students of San Francisco well, a true problem oriented text has failed to emerge from the fog around the Bay! A modern text to supplement our new and evolving approach to the undergraduate teaching of clinical neurology remains a challenging agenda.

Ian Bone

Clinical trials in neurology

Edited by Roberto J Guiloff (Pp 542, EUR178,00). Published by Springer, London, 2001. ISBN 1-85233-239-5

This much needed book is a gem. If you are involved in a clinical trial make sure you and all your collaborators have read it, before the trial commences. The application of the methodology of the randomised clinical trial has become the absolute test of all new medicines, not only in neurology but also generally in medicine and surgery. Once one is involved in a trial, however, it is difficult to keep a sense of independence from the desire that the trial should have a positive outcome—that, after all, is one of the several major reasons for using randomisation and double blinding. This book provides the information the clinician needs to understand how trials are organised, how data are collected, how they are analysed, and how their clinical significance should be assessed. Dr Guiloff has assembled an impressive team of contributors, all with expertise and experience in their subject. The book is organised in two parts. In the first part, general issues relating to ethics, regulatory matters, assessment, measurement, statistics, quality control, and important specific issues, such as the use of intention to treat analysis, handling drop outs in repeated measures analysis, and survival analysis, are presented. Well chosen examples enliven the text and tables. The role of the Cochrane Collaboration and of meta-analyses is considered in separate chapters. The second part of the book is concerned with the application of these general principles to neurological disorders. This section consists of groups of chapters related to ethical issues, trial design, measurement, sample size, data analysis, and critical discussion of the results of large trials in the major neurological disorders. The consistent high quality of these chapters is remarkable in a multiauthored text and a tribute to the hard work of the editor. The book concludes with a review by Michael Brooke that should be required reading for every neurologist. This book not only fills a gap on the neurologist's bookshelf but also will be opened again and again.

Michael Swash

Differential diagnosis in neuro-oncology

Edited by Jerzy Hildebrand and Michael Brada (Pp 298, £59.50). Published by Oxford University Press, Oxford, 2001. ISBN 0-19-263213-2

This book is written by two very experienced, eminent, and respected European neuro-oncologists. The aim of the book as described in the introduction is assisting in the correct diagnosis and therapeutic management of patients with neurological syndromes due to neoplastic conditions. The neurological syndromes in cancer patients cover the spectrum of neuro-oncological sites and each chapter is broken down into subheadings of introduction, clinical presentation, main aetiologies, investigations, treatment, and appropriate references. The neurological syndromes covered are altered consciousness, cognitive and behavioural disorders, epileptic seizures, cerebellar dysfunction, visual alterations, cranial nerve and brain stem lesions, spinal cord lesions, diffuse lesions of the peripheral nervous system, focal lesions of the peripheral nervous system, muscle disorders and fatigue, endocrine disorders, and treatment of the main neurological malignant diseases.

Overall this gives a very compact and user friendly description of the major clinical features and disorders encountered in the practice of clinical neuro-oncology. The strength of the book lies clearly in the widespread experience of the two authors. They have been able to include common and rarer neurological syndromes into an easily digestible and coherent clinical classification. In particular for neurosurgeons this book will offer a great deal since it provides major insights into non-surgical courses of neurological syndromes seen in patients with cancer. This applies particularly to problems related to chemotherapy and radiotherapy, as well as paraneoplastic syndromes, which are well covered.

While the book will be a useful reference tool for the surgeon, it is rather weak on neurosurgical aspects in neuro-oncology. For example, under neurosurgical therapy of glioma (p 227) it is stated that "optimal resection (of gliomas) should achieve tumour removal without morbidity." This is despite morbidities of 10–24% being described in most current series in the literature. There are also allusions to the use of neuronavigation tools in neurosurgery, which will make tumour removal "easier and more complete." However, at this stage there is no evidence for this. There is also an imbalance in the role of resection versus biopsy in gliomas. A recent Cochrane review found that there is no evidence in the literature to show that survival outcomes are better in patients who have resection rather than biopsy for malignant glioma. On page 223 it is also suggested that histologically glioblastomas are homogeneous—it is clearly not the case since they are heterogeneous.

There are also minor points in terms of preparation of the book, such as reference 38 in the treatment of gliomas—this is dated 1977 rather than 1997. Some of the plates are labelled incorrectly and the subheading in the myopathy section is under cognitive and behavioural disorders. These are, however, minor quibbles. The depth of coverage of medical and radiation neuro-oncology, the excellent core reference list, and numerous useful illustrations and algorithms make this a "must have" book. Do not be put off by the atrocious book cover—what on earth is it?!

Ian Whittle

Interventional and endovascular therapy of the nervous system. A practical guide

By Pearse Morris (Pp 318, £94.00). Published by Springer-Verlag, New York, 2002. ISBN 0-387-95193-8

As its title implies, this is a practical guide to interventional neuroradiology. All the conditions encountered by the interventionalist are covered in the 15 well chosen chapters. The book is remarkably up to date, given the rapidity of technical change in the specialty. This is a tribute to the author and to Springer, who have produced a high quality, superbly illustrated book of the type for which the publishers are rightly renowned.

The book is written primarily for neuro-interventionalists, both those in training and

Downloaded from jnnp.bmj.com on November 22, 2012 - Published by group.bmj.com

those who are "trained" (if there is such a state). There is also something in the book for clinicians—neurosurgeons, neurologists, and, one must not forget, anaesthetists and intensivists, all of whom may be involved in the management of these patients. The sections on pharmacology and haemostasis are particularly useful and it is worth noting that treatment methods in neurointervention are similar worldwide. Some minor differences exist, imposed by the various national regulatory bodies concerned with drugs and devices. The book can nevertheless be studied profitably by an international readership.

It would have been valuable to include something on the natural history of intracranial aneurysms, particularly the rather vexed question of what is to be done with the unruptured group of aneurysms. No more than a passing reference is made to Onyx, the non-adhesive liquid embolic agent, which is regularly used in the United Kingdom for cerebral arteriovenous malformations and less commonly for especially challenging aneurysms. This is a very recent development and should not detract from this being an excellent, up to date book, full of practical advice. I look forward to future editions. This book has already been discovered by our trainees. It is strongly recommended for their personal library and for inclusion in the neuroscience departmental library.

Paul Butler

Principles of neuroepidemiology

Edited by Tracy Batchelor and Merit E Cudkowicz (Pp 374, £65.00). Published by Butterworth Heinemann, Boston, 2001. ISBN 0750670428

This useful book gives an overview of the epidemiology of neurological disease. It is not fully comprehensive but this is to be expected in a relatively short book of just over 300 pages. It begins with four useful chapters on epidemiological methods, statistical principles, clinical trials, and measurement scales. These are fairly sketchy in parts but provide a worthwhile summary of the general principles.

The coverage of the epidemiology of neurological disease is good, with individual chapters on the 12 most common neurological syndromes and diseases, including back pain, headache, head injury, and sleep disorders, each of which is sometimes ignored in books of this kind. However, there are no chapters on muscle disease or peripheral neuropathy.

The book is rather US oriented. The editors admit in the preface that their "focus was limited to adult neurological diseases occurring in the US." However, in these days of international research collaboration and electronic communication, it is somewhat disappointing to find that only 2 of the 31 authors were from outside North America. The authorship was also dominated by Boston based clinicians and academics, who accounted for over 60% of the authors. Harvard certainly has a strong tradition of high quality epidemiology but a more varied authorship might have provided a broader perspective. There is also a tendency in several of the chapters for the research that is discussed and referenced to be US based.

Nevertheless, the book does provide a useful overview of the epidemiology of most of the neurological diseases that are common in the developed world. It would therefore be of use to the general neurologist or to the non-clinical neuroscientist with an interest in the clinical burden of neurological disease.

Peter Rothwell

Downloaded from jnnp.bmj.com on November 22, 2012 - Published by group.bmj.com

Neuropsychological and psychiatric complications in endoscopic third ventriculostomy

J van Aalst, E A M Beuls and G J Luijckx

J Neurol Neurosurg Psychiatry 2002 73: 460
doi: 10.1136/jnnp.73.4.460

Updated information and services can be found at:
http://jnnp.bmj.com/content/73/4/460.1.full.html

These include:

References This article cites 3 articles, 1 of which can be accessed free at:
http://jnnp.bmj.com/content/73/4/460.1.full.html#ref-list-1

Email alerting service Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/