Neuropsychological and psychiatric complications in endoscopic third ventriculostomy

We read with interest the recent paper by Benabarre et al. 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 ETV 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 agitated, 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 ETV 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 ETV 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 ETV 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 ETV. We agree with Benabarre et al. that clinicians should be aware of and take into account this potential serious complication of ETV in this so called minimally invasive procedure.

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 and Vifias et al.: 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 hoehnergusiveness, 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-based structures of the brain: the fornix and its connection to the hippocampus and the mammillary 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 ETV because the correct Diagnostic and statistical manual of mental disorder category may be a mental disorder not otherwise specified due to a general medical condition.

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.

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Neuropathological findings in multiple system atrophy with dystonia

Boesch et al. 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. 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 dorsolateral putamen and widespread glial cytoplasmic inclusions, considerable focal cell loss in the caudal ventrolateral parts of the substantia nigra pars compacta (SNC) and locus coeruleus without Lewy bodies or...
pontine involvement, corresponding to MSA-P grade II. Case 18 with five years duration of illness and craniooculopial peak dose dystonia, showed similar mild degeneration of the dorsolateral putamen and SNC (grade III), in 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 ventrolateral 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 ventromedial and rostral parts, corresponding to MSA-P grade III. A review of necropsy cases of MSA with dystonia in the literature and eight personal cases gave the following results: 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 dorso-lateral than ventral parts, and the SNC were all grade III. Among 28 cases with spas tic and generalised dystonia, 21 were MSA-P and eight MSA-C. Twenty six brains revealed severe degeneration of the putamen, 12 of the caudate, 14 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, reticulare SN, arcuate nuclei, 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 puta men 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. Focal dystonia and dystonia of the basal ganglia circuitry. However, there is 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.

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References

Social phobia in spasmodic torticollis: some conceptual issues
Gundel et al tackle an important issue of social phobia (SP) in patients with spasmodic torticollis (ST). The importance of this social avoidance itself contributes to a significant degree of distress and dysfunction in patients with chronic medical conditions. Also, potentially beneficial pharmacological and psychosocial treatments remain undervalued because of their under recognition of this problem. Keeping these aspects in mind, we would like to highlight several conceptual and methodological issues.

Firstly, the sample was restricted to patients with ST who required treatment with botul ium 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 manifesta tion of ST. As per the authors the mean (SD) duration of illness at the 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 with problem 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 comorbidities. Comorbidity as a concept has different dimensions.5 As this classification is not followed in DSM-IV 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 ST 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 and DSM-IV. 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 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 diagnostic criteria do not preclude 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 current ICD classification system resolves this issue in greater detail with availability of the current empirical evidence.

Lastly, though the authors adopted a rigorous methodology for determination of diagn osis, yet it seems surprising that no patient was assessed for Axis II diagnosis; anxietyavoiding personality disorder (AAPD). AAPD is the most common differential diagnosis for SP and it has been shown that differentiating it from SP is difficult.6 Additionally, patients with SP can have premorbid anxious traits (or AAPD), making personality an important comorbid issue.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.

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References

Authors’ reply
(1) We agree with Sharma and Gupta in that our results are based on spasmodic torticollis (ST) patients treated with botulimum toxin, although 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 2–6.

(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 ratio of 5.4 per 100,000 in cervical dystonia,8 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.9 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 patient’s view. Based on the patient’s answer and the clinical judgment of the interviewor, 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

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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 and essential tremor, we used DSM-IV criteria. 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 association between an 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 consideration that depressive symptoms need to be considered in elderly patients with 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), 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 for 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. Furthermore, the relation between cerebrovascular disease and depression has recently been established. 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.

Executive dysfunction and depressive symptoms in cerebrovascular disease

The article by Kramer et al suggests that subcortical-frontal ischaemic disease accounts for a significant proportion of depression cases. However, Kato 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.

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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 moderating 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 depression in cerebrovascular patients is caused by their depression and not by their cerebrovascular disease. Cerebrovascular disease might cause 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 moderating variables when evaluating cognitive dysfunction.
Clinical trials in neurology


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. As a trialist you need to ensure 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 prepared and conducted. I would recommend this book for neurologists who are involved in clinical trials, as well as for those involved in the design and analysis of clinical trials.

Michael Swash

Differential diagnosis in neuro-oncology


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Michael Swash

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 and endovascular therapy of the nervous system. 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 neurointerventionists, both those in training and

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BOOK REVIEWS

Clinical neurology


Reviewing an undergraduate text is always quite a difficult task in that one has to approach one’s specialty 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 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) fit 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 listing 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 from the fog around the Bay! A modern text

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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

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