PostScript

CORRESPONDENCE

Deep brain stimulation for cervical dystonia

I read with interest the recent case report by Chang and colleagues on unilateral deep brain stimulation (DBS) of the globus pallidus internus (GPi) in a patient with delayed-onset posttraumatic cervical dystonia.[1] I congratulate the authors reporting another patient with cervical dystonia responding to GPi DBS. The unique feature in their case is that unilateral stimulation only was used. They report on a 23 year old man who developed cervical dystonia with head turning to the left three years after he sustained a severe closed head injury. Magnetic resonance (MR) studies five days after the injury demonstrated focal lesions of the left pallidum, but also of the right thalamus. Six years later only the left pallidal lesion could be appreciated by MR studies. The authors chose to implant a quadripolar DBS electrode in the left GPi for chronic stimulation. They further report that during chronic stimulation the patient's cervical dystonia improved, and that he could turn his head to the midline easier than preoperatively. The improvement was not assessed by standard rating scales for cervical dystonia, and it is said that the dystonia was stable three months after electrode implantation. The authors conclude that the cervical dystonia in their patient was secondary to the GPi lesion, and that unilateral DBS of the GPi contralateral to the dystonic sternocleidomastoid muscle is the treatment option of choice. I wonder whether the thalamic lesion shown in the early MR scans could also have been relevant in the development of this patient's dystonia. It has been demonstrated previously that posttraumatic cervical dystonia may be associated with subthalamic and upper brainstem lesions.[2]

Interestingly, Chang and colleagues conclusions on the side to be choosen for unilateral DBS are at odds with another recent case report. Escamilla-Sevilla and colleagues observed improvement of segmental cervical and truncal dystonia in a 24 year old man with idiopathic dystonia during unilateral stimulation of the GPi ipsilateral to the dystonic sternocleidomastoid muscle.[3] In that case no notable change of cervical dystonia was observed with bilateral stimulation for six months. When it then was decided to switch to unilateral stimulation of the right GPi there was progressive improvement over the next three months. Unfortunately, chronic stimulation of the left GPi was not performed in that case. These authors concluded that stimulation should be started on the side ipsilateral to the dystonic sternocleidomastoid muscle.

The discrepancy between these two reports reveals the problems inherent in conclusions made from single case reports. It also reminds of the historic discussions decades ago, when Cooper thought that thalamotomies should be performed on the side contralateral to the dystonic sternocleidomastoid while Hassler stated that ipsilateral lesioning would be more beneficial.[4] When we introduced the concept of GPi DBS for cervical dystonia in 1997 we discussed several alternatives regarding the choice of the target and also whether unilateral or bilateral DBS should be used.[5] We then decided to go ahead with bilateral stimulation for several reasons, based on contemporary imaging studies and also accumulating knowledge on the innervation of neck muscles. Magyar-Lehmann and colleagues, for example, showed that patients with cervical dystonia had higher glucose metabolism bilaterally in the lentiform nucleus in a PET study without significant differences regarding the laterality, the specific pattern, or the severity of cervical dystonia in individual cases.[6] Naumann and colleagues also demonstrated bilateral basal ganglia involvement in cervical dystonia patients by striatal D2-receptor binding studies.[7] In that study, there was no significant difference by intraindividual comparison of contralateral versus ipsilateral striatal epipride binding with regard to the direction of head rotation. In a recent transcranial magnetic stimulation study in normal subjects, ipsilateral as well as contralateral sternocleidomastoid responses were evoked by stimulation of an area of cortex near the representation of the trunk.[8] With that regard, however, it is also important to consider that head rotation in patients with cervical dystonia is not only due to contraction of the sternocleidomastoid, but also of the posterior neck muscles. In our series of patients who underwent bilateral pallidal DBS for treatment of cervical dystonia we have repeatedly observed clinical deterioration with dysfunction of stimulation on one side or when the battery on one side was depleted. It is unclear, therefore, whether or not additional benefit would have been achieved with stimulation also of the right GPi in the patient reported by Chang *et al.*

By the way, in the Discussion the authors cite data on the frequency of posttraumatic movement disorders secondary to severe head injury. I was surprised to see that these data were attributed to the study on posttraumatic hemidystonia by Lee and colleagues.[9] These data, however, were reported in a later study where we investigated the frequency of posttraumatic movement disorders in the survivors of head injury who were admitted to a multidisciplinary trauma unit during a five year period.[10]

In conclusion, for the moment I think it is advisable to continue with bilateral DBS in the treatment of cervical dystonia until solid evidence should become available that unilateral stimulation is sufficient. It would be most interesting to evaluate the different profiles of bilateral and alternating unilateral stimulation in patients who have bilateral electrodes. Whether such a study is feasible and practical, however, is open to debate.

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Failure of regular external ventricular drain exchange to reduce CSF infection

Dr Wong and colleagues undertook quite a careful prospective randomised trial aiming to determine whether routine changing of external ventricular drainage catheters reduces the risk of CSF infection.[1] Patients were randomised into two groups: group 1 (n = 51) had routine changes of the external ventricular drain at five day intervals; in group 2 (n = 52) the ventricular drain was not changed. There was no difference with respect to the basic demographic data and the incidence of CSF infection. The authors observed four CSF infections in group 1 (7.8%) and two in group 2 (3.8%). Despite the higher CSF infection rate in group 1, this difference was not statistically significant. Based on their results, the authors concluded that ''routinely changing external ventricular drainage catheters at five day intervals did not reduce the risk of CSF infection''.

The topic of ventricular catheters and the risk of CSF infection has been dealt with in numerous reports. The continuing interest for neurosurgeons is largely based on the fact that quite controversial recommendations

have been published regarding the use of external ventricular catheters.

In general, our experience with CSF infections is similar to that of Wong. We investigated which factors increase the incidence of CSF infections in a prospective study including 133 patients who underwent 152 surgical procedures for external CSF drainage.[2] Assessed variables included basic demographic data, with special reference to the duration of surgery, diameter of the catheter used (5 F *v* 10 F), distance of the subcutaneous tunnel between the burr hole and the cutaneous exit point, additional surgical procedures, and duration of CSF drainage.

In our study group we had a CSF infection rate of 4.5% per patient and 3.9% per surgical procedure. Whereas most of the variables assessed showed no statistically significant correlation with the incidence of CSF infection, interestingly we observed a close correlation between the length of the subcutaneous tunnel and the incidence of infection. In 83% of the patients with CSF infections the catheter was tunnelled subcutaneously for less than 5 cm, whereas in only 17% was the catheter tunnelled for more than 5 cm. This observation was associated with the fact that there was a higher incidence of CSF leakage through the cutaneous exit point with shorter tunnels despite correct operative management.

Taking into consideration that in the study by Wong *et al*, ''all the bacteria are common in the skin flora of patients in the intensive care unit'' and ''all infections occurred after day 10'' (mean 13 days), these findings strongly support our observation of increased CSF infections caused by secondary contamination rather than as by contamination during the catheter placement procedure.

In agreement with Dr Wong, we do not recommend routine replacement of the ventricular catheter, but based on our data we strongly recommend a sufficient length of subcutaneous tunnelling (5 cm or more) to reduce the risk of CSF infection, because despite efficient antibiotic treatment a CSF infection is still a serious complication and must be avoided.

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Authors' reply

We were pleased to see the above letter about the importance of tunnelling. Subgaleal or subcutaneous tunnelling of ventricular drains has been accepted since the late 1970s as a way of reducing ventriculostomy related CSF infections. In accordance with this concept, our protocol is to use a tunnel of 4 cm or more as necessary. It is gratifying to see recent confirmation of this in the correspondents' own series. Our own low CSF infection rate in the ''no change'' group (3.8%) in such a high risk group of patients further supports this concept.

There is still much debate on what constitutes the most favourable tunnel length. Some would advocate a short tunnel of 4.5 cm, whereas others prefer the tunnel to reach the lower chest or upper abdomen.[1] All of these documented series, including our own, had a low CSF infection rate of 3–4%, giving a long average duration of catheter placement of 11 to 18 days. In Khanna's series[1] the change to a long tunnel appeared to contain the infection rate, giving an average of 18.3 days for an indwelling catheter. In cases where a long duration of catheter placement is likely, conversion to a long tunnel may be advisable, both to reduce the infection rate and for convenience in mobilisation.

The concept used in our paper of relating the number of ventricular catheter insertions to the CSF infection rate concurs with earlier series[2] as well as that of the correspondent. It is important to investigate the possible pathogenesis and to consider viable means of achieving improved results. Results from our own data indicate that the source of infection is bacteria found in the patients' own skin flora. Regular changing of the catheter (which in theory should reduce the opportunity for colonisation leading to infection) has not only failed to reduce infection but may even have increased it. Tunnelling may be helpful in preventing colonisation from progressing to infection. Most infections appear to be caused by resistant skin flora introduced at the time of the procedure, despite the use of standard aseptic technique and prophylactic antibiotic cover. Regular audits to ascertain the MRSA status of both the intensive care unit and operating environment are therefore of great importance.

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Non-convulsive status epilepticus

In a recent article, Husain *et al* argue that severely impaired mental state, ocular movement abnormalities, and the patient's history could suggest a diagnosis of non-convulsive status epilepticus (NCSE), and then be a selection criterion for patients with impaired consciousness to undergo an urgent EEG recording.[1] However, our experience with 50 adult patients (12 men and 38 women, mean age 65.9 years) meeting the criteria for the diagnosis of NCSE[2] suggests that there are no peculiar clinical features characteristic of this condition. Twenty eight patients had absence status, 16 had complex partial status, and six had aphasic status; 11 had tonic-clonic seizures just before the onset of status, and eight had a history of chronic epilepsy. The main clinical presentation of NCSE was aphasia (six patients), mutacism (two patients), psychiatric disturbances (four patients), delirium (34 patients), and stupor or coma (four patients). We feel therefore that all acute alterations of mental state or changes in behaviour from baseline for which no alternative explanation is available may raise a suspicion of NCSE and call for urgent EEG. In patients with known epilepsy presenting with prolonged confusion after convulsions, an urgent EEG is warranted to distinguish between postictal encephalopathy and generalised NCSE.

Kaplan[3] reported that the diagnosis of NCSE was initially missed in the emergency room when the behavioural or cognitive changes from baseline were ascribed to other causes, including intoxication, postictal encephalopathy, pre-existing psychiatric conditions, or mental retardation. In our study, the delay in diagnosis of NCSE ranged from three hours to 28 days, with a mean of three days. In two patients the diagnosis was delayed despite an emergency EEG, because of the lack of clear epileptiform features in the EEG abnormalities. A suspicion of NCSE is the most important clinical indication for performing an emergency EEG.[4] Because of the different EEG patterns and the pleomorphic clinical features, a diagnosis of NCSE is only possible with an expert integration of EEG findings and clinical data, as emphasised by Niedermeyer and Ribeiro.[5]

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Author's reply

The interest and comments of Audenino *et al* are greatly appreciated. In our paper, 48 patients who were suspected of being in NCSE were evaluated prospectively by neurology residents; the diagnosis of NCSE was later confirmed or ruled out on the basis of the patient's EEG. Remote risk factors for seizures (such as previous stroke, neurosurgery, significant head trauma), impaired mental status, and ocular movement abnormalities (sustained eye deviation, nystagmus, hippus) were found significantly more often in the NCSE group. The combined sensitivity of remote risk factors for seizures and ocular movement abnormalities was 100%; there was no patient in the NCSE group who did not have either of these findings.

Audenino *et al* present their series of 50 patients, all of whom met their criteria for NCSE. In their series, like ours, women outnumbered men. They note that there are ''no peculiar clinical features of NCSE.'' They also suggest that all alterations of behaviour for which there is no alternative explanation should be evaluated with an urgent EEG.

I agree that for all patients with an altered sensorium, NCSE should be considered as a possible diagnosis. Furthermore, there is general agreement that an EEG is required for the diagnosis of NCSE. In our study it was also noted that the presence of a metabolic or other type of encephalopathy did not necessarily imply exclusion of NCSE. Thus should urgent EEG be requested for all patients with altered mental status, regardless of comorbidities? No; this is impractical not only after working hours, but also during working hours in most hospitals. Therefore, we attempted to triage patients who should be getting an urgent EEG. This can be accomplished on the basis of the high sensitivity of the above findings. This high sensitivity should not be mistaken for high specificity; in fact the specificity was low. A valid criticism would be the low specificity, but our objective was not to find an alternative to EEG for the diagnosis of NCSE, but rather to triage those in need of one.

Another important difference between Audenino's series and our own is that ours was obtained prospectively. A prospectively obtained neurological history and examination is likely to be more detailed than information obtained retrospectively from a review of the clinical records. A history of remote risk factors for seizures and the presence of ocular abnormalities can be missed during an urgent neurological evaluation unless specifically sought.

I would like to emphasise again that the objective of this study was not to find alternatives to EEG in the diagnosis of NCSE, but rather to help select those who should have an urgent EEG. Performing an urgent EEG on every patient with alterations in mental status is not practical or possible in most institutions.

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Hemicraniectomy for large middle cerebral artery territory infarction: do these patients really benefit from this procedure?

Pranesh *et al* presented a series of 19 patients undergoing decompressive hemicraniectomy for large middle cerebral artery infarction with clinical and radiological signs of transtentorial herniation.[1] Among these, 10 patients (53%) suffered from a dominant hemisphere stroke. Neurological state was assessed according to the National Institutes of Health Stroke scale (NIHSS) initially and one week after surgery, and functional outcome at three months' follow up using the Barthel index (BI) and Rankin scale (RS). The mean NIHSS score improved from 20.5 before surgery to 10.5 postoperatively. At last follow up mean BI was significantly better in younger patients (60.7) than in older patients (41.3). The authors conclude that hemicraniectomy may be a useful procedure on patients with large middle cerebral artery infarction.

Recently we undertook a prospective non-randomised study in 26 patients with decompressive hemicraniectomy for right sided middle cerebral artery infarction, analysing functional outcome (NIHSS, BI, RS) at one year of follow up.[2] In contrast to all previous reports, neuropsychological testing was also done, focusing on right hemisphere function (evaluation of visuospatial and visuoconstructive abilities, attention, spatial span, and self rated mood). In 18 surviving patients at the one year follow up the functional outcome was good or fair in nine (BI >75, RS 2–3), moderate in six (BI 30–70, RS 4), and poor in three (BI 0–25, RS 5). Thus only nine of 26 patients (35%) were functionally independent and needed no or only minimal assistance for daily life activities. As was shown previously,[3] age was identified as a significant and independent predictive factor on outcome, with better functional results in younger patients. Neuropsychological testing was possible in 14 patients, while four were too disabled to be evaluated. All patients showed profound attention deficits, and visuospatial and visuoconstructive deficits was observed in those with less formal education. These disturbances led to a substantial handicap for professional activities.

On the basis of our functional and particularly neuropsychological results in patients with isolated non-dominant middle cerebral artery infarction, we would strongly discourage hemicraniectomy in patients with left sided, dominant hemisphere or multiterritory infarction, as there is a significantly higher risk of dependency, hopelessness, and more severe neuropsychological deficits in such cases. In our opinion decompressive hemicraniectomy should be restricted to younger patients with non-dominant hemisphere infarction. The goal of the procedure is to operate on these patients in an early stage of the disease, before additional infarction had occurred as a result of local mass effect and herniation. Up to now, we have operated on 39 patients with middle cerebral artery infarction in our institution, but our experiences do not encourage to us to act with great enthusiasm.

Pranesh *et al* stated correctly that this surgical procedure can be undertaken safely, however, the main difficulty is in deciding to *not* operate on such patients, despite the simplicity of the surgical procedure.

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Authors' reply

The points raised by Sandalcioglu *et al* are well taken. It was considered justified to undertake decompression even on the dominant side because, if such patients were left with a severe disability, the excellent family support system in India would be available. We do agree that the quality of life is poor after such a decompression. However, the recovery of speech function in our patients has been remarkable, apart from saving their lives which was the patients' relatives' wish.

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BOOK REVIEWS

Psychiatric genetics and genomics

Edited by Peter McGuffin Michael J Owen, and Irving J Gottesman. Published by Oxford University Press, 2002, pp 472, £65.00. ISBN 0-19-263148-9

At all turns we can less and less speak of medicine without qualifying it with the term molecular. Our genetic underpinnings and their consequences have assumed their rightful place as extremely important factors in the pathophysiology of most disease—in fact it sometimes seems nearly all disease. In (arguably) the current bible of molecular medicine,[1] Barton Childs argues, to my mind convincingly, that the future general textbook of medicine will move even further away from the traditions of Osler and firmly towards Garrod. Disease becomes incongruence between variable homeostatic mechanisms and the internal and external environments. At the centre is biochemical individuality and its molecular counterpart mutation. Proteins (and the genes that drive their production) are ubiquitous parts of our homeostatic mechanisms at all levels, the molecular and subsequent biochemical variation determines how we interact with environmental experiences, including social, and how these feedback on the system.

That psychiatric illness is not exempt from such genetic considerations has been clear for some time. What this new volume edited by Peter McGuffin and his colleagues shows, however, is how widely permeating this has become. There are chapters here that range from personality and cognition (an excellent one from Plomin, Happe, and Caspi) through to personality disorders, anxiety, and eating disorders, through to the more mainline genetics of schizophrenia and affective psychoses. In general they are well written and surprisingly up to date. As a source book of references alone this is worth having and those to very recent publications including 2002 are numerous. The traditional tripos of family, twin, and adoption studies is covered for most disorders before moving into linkage, association, and, where relevant, other molecular analyses such as cytogenetics. The chapter on dementia naturally moves further into the field of molecular pathology and biology, and covers the transmissible encephalopathies and CJD. Contentious areas are not omitted and the chapter on ethical issues is thoughtful and avoids the tokenism (or complete omission) that was the hallmark of some previous works.

Are there any drawbacks? There are always some to be found and as usual these may simply reflect bias on the part of the reviewer. The chapter on mental retardation is exceptionally short given its huge clinical importance (mental retardation and epilepsy together are the most common of all neurological conditions) and the recent explosion of interest in the genetic (and epigenetic) phenomena involved. However, this is a relatively small quibble; it is a well produced and worthwhile volume. On the reviewer's copy the edges of many chapters are already very well thumbed and grubby, which is as good a recommendation as any.

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Psychiatric and cognitive disorders in Parkinson's disease

Edited by Sergio E Starkstein and Marcelo Merello. Published by Cambridge University Press, Cambridge, 2002, pp 160, £47.50. ISBN 0-521-66305-9

This comprehensive account of the common (but frequently overlooked and under treated) emotional and cognitive aspects of Parkinson's disease is thoughtfully organised and well written. The two authors have presented their material in a consistent manner, free from the difficulties (for example, redundancy) often associated with multi-authored texts. Tables and illustrative clinical vignettes are helpful. References are up to date and thorough. In general, the book is well edited (although the two figures demonstrating the cortico-subcortical connections need revision). The text itself is less than 200 pages and is relatively easy to read in its entirety, but each chapter can stand alone.

The first few pages briefly highlight the content of and rationale for each of the chapters. The next 50 pages provide a useful background for the non-movement disorder specialist. Chapter two reviews motor features and their treatment. Interestingly, the discussion on surgical approaches is as long as the discussion of pharmacotherapy. This probably reflects the fact that deep brain stimulation is becoming more widely available. The third chapter provides a concise but thorough and clearly presented overview of the differential diagnosis of Parkinson's disease, with a very relevant discussion of dementia with Lewy bodies. The discussion of Alzheimer's disease might have been enhanced by a note that these patients can sometimes have ''pseudoparkinsonism'', characterised by paratonia and gait apraxia (rather than true rigidity and a parkinsonian gait). Chapter four (the one chapter devoted solely to cognition) effectively conveys the notion that dementia in Parkinson's disease may be not be a homogeneous phenomenon. The authors make the interesting point that bradyphrenia may be accounted for solely by depression and/or incipient cognitive decline. Chapter five (Depression in Parkinson's disease) highlights how common depression is in this illness and provides evidence that untreated depression may result in cognitive decline, making a strong case for early recognition and treatment of depression. Chapter six includes a discussion of anxiety, apathy, and the debatable concept of a distinct premorbid personality type. Chapter seven mainly focuses on dopaminergic drug induced psychosis. Chapter eight deals with the treatment of depression and psychosis. The appendix consists of several Parkinson's disease specific scales but does not include other scales commonly used to evaluate depression and anxiety in Parkinson's disease.

One would not necessarily want to use this book as a reference for specific treatment guidelines and/or dosing of medications. Dosages are not always discussed (for example, for quetiapine) and a few statements are subject to disagreement. In their discussion about unpredictable levodopa responses, the authors appropriately suggest switching from controlled release levodopa to more frequent doses of an immediate release formulation but state that one should keep the same total daily levodopa dosage. Because controlled release tends to have lower bioavailability, many neurologists would reduce the total dose of levodopa when switching to immediate release preparations. The figure demonstrating the treatment of psychosis in Parkinson's disease suggests that one should check blood and urine for infection or metabolic problems, then check a CT scan before proceeding. Except in unusual circumstances, most Parkinson's disease specialists would not embark on such an extensive diagnostic investigation. The suggestion that severe psychosis warrants mandatory admission and that one should consider stopping all anti-Parkinson's disease medication does not reflect typical practice and could, in fact, be dangerous because of the risk of an NMS like syndrome.

In summary, this well written book will enable readers to have an up to date and well rounded knowledge base regarding the cognitive and psychiatric aspects of Parkinson's disease and would be quite helpful to all clinicians (including neurologists and non-neurologists) who deal with Parkinson's disease patients.

I Hegeman Richard

Surgical treatment of Parkinson's disease and other movement disorders

Edited by Daniel Tarsy, Jerrold L Vitek, and Andres M Lozano. Published by The Humana Press, Totowa, 2002, pp 353, US$165.00. ISBN 0-86903-921-8353

The editors have assembled a panel of leading experts to produce this book, which is well referenced and its black and white figures nicely produced. The book is predominantly concerned with the role of stereotactic surgery for movement disorders and this subject is examined in depth. The book is divided into four parts. The first section recounts, in three chapters, the rationale for surgical therapy. The circuitry and physiology of the basal ganglia are reviewed along with the historical development of surgery for Parkinson's disease.

The second and main part of the book describes the surgical management of Parkinson's disease and tremor patients, including patient selection and assessment, target selection and localisation, operative techniques, neuropsychological evaluation, and in situ programming of deep brain stimulators. This section also contains separate chapters on thalamotomy, pallidotomy, subthalamic nucleotomy, and deep brain stimulation of the thalamus, globus pallidus, and subthalamic nuclei. Within these chapters there is a rich diversity of opinion, which is one of the great strengths of this book and reflects this rapidly expanding field.

The third section reviews the surgical treatment of focal and generalised dystonia. This is presently a very exciting field and the relevant chapters detail experience with thalamotomy, pallidotomy, and pallidal stimulation as well as the roles of intrathecal baclofen pumps and peripheral denervation procedures for managing dystonic patients.

The final part of the book, labelled Miscellaneous, describes the use of PET for examining the changes in activity in the cerebral circuitry of movement disorder patients undergoing surgery. Finally, there is an account of the role of fetal transplantation and future surgical therapies for the treatment of Parkinson's disease.

This book provides the reader with considerable penetration into the rapidly expanding field of movement disorder surgery. I found it fascinating and informative. It has a place in the hospital or university neuroscience library and I particularly recommend it to neurologists, neuropsychologists, neurosurgeons, and research fellows who wish to have an overview and/or develop their interest in stereotactic surgery for movement disorders.

P Bain

Concise guide to neuropsychiatry and behavioural neurology, 2nd edition

Edited by Jeffrey L Cummings and Michael R Trimble. Published by American Psychiatric Publishing Inc, Washington DC, 2002, pp 246, US$29.95. ISBN 1-58562-078-5

Cognitive neurology is on the up. In Britain, at least, the numbers of trainee neurologists who aim to make this their focus of interest is—at last—increasing. And this is not only because of the attraction of the bright, kaleidoscopic lights of functional imaging! No, some neurologists in the making appreciate that perhaps there is a great deal still to be said for the careful assessment of patients with both focal and diffuse brain lesions. Not only does this offer an important insight into normal brain function, but it is critical for the development of therapies for cognitive impairments. So, is this handbook a helpful contribution to the renewed interest in cognitive function?

It certainly does have several features to recommend it. It is compact, to the point, and gives references to important papers in the literature. It covers a vast amount of neurology and neuropsychiatry in a breathtaking short format. However, although brevity is often to be admired, there is a danger that some of the points being made are going to be appreciated only by those who already know what you are talking about. This surely should not be the aim of a handbook that is aimed at trainees. Moreover, attempts to make things concise can sometimes lead to important omissions. In this text, for example, there is a small section on simultanag-

nosia that explains well the key feature of this condition, and goes on to mention that it may form part of Balint's syndrome. But missing is one line that explains that the latter is a rare syndrome observed usually when there is bilateral posterior damage, centred on the inferior parietal lobule. Corticobasal degeneration is confined to a single line in a table, although progressive supranuclear palsy does get a paragraph. Sometimes conciseness also leads to a blurring of distinctions: motor neglect is said to refer to a lack of motor response in the neglected hemispace and is noted also to affect the contralesional limb, whereas most experts use the term to refer to only a limb specific neglect. Finally, the figures could be improved upon. For example, the one showing key elements of the limbic system might look good in the original book from which it is taken, but it really is not very clear or helpful in the version scanned into this handbook.

These sorts of quibble apart, this is a useful guide that serves as a gateway to fuller descriptions and discussions in the primary or review literature. It is worth dipping into to see whether it suits you. My own preference would be for a slightly longer handbook that covers some of the syndromes, conditions, and treatments in a little more detail.

M Husain

Neural stem cells for brain and spinal cord repair

Edited by Tanja Zigova, Evan Y Snyder, and Paul R Sanberg. Published by The Humana Press, Totowa, 2002, pp 425, US$149.50. ISBN 0-58829-003-4

For scientists, clinicians, patients, and the biotech industry, transplantation of stem cells has become one of the major hopes for repair of what are currently incurable degenerative diseases and trauma to the brain and spinal cord. The 16 contributions contained in *Neural stem cells for brain and spinal cord repair* deal with these questions. They provide a needed, very handy, and comprehensive review, defining the current state of knowledge in this rapidly moving area.

The contributions cover the many possible sources of stem cells, embryo, and adult, including brain, neurospheres, neural crest, bone marrow, and already established human stem cell lines such as human neuroteratocarcinoma cells. Several chapters summarise the current methods for obtaining the various types of stem cells, the signalling pathways in differentiation, and the degree to which it is known that stem cells are able or can be induced by appropriate growth factors to adopt or turn into the differentiated cell types that would be needed for functional replacement in damaged tissues.

A number of contributions deal with practical applications, such as the possibility of glial cell precursors being used for treatment of demyelinating diseases, the use of stem cells to boost a failing host dopaminergic system in Parkinson's disease, and the concept of global replacement by genetically modified cells able to replace enzymes nonfunctional because of inherited genetic defects. Particularly interesting are the provocative observations (Magavi and Macklis) that transplanted stem cells are able to "detect" defects at a distance, and migrate through the host brain to repair damaged areas. A number of chapters consider the issue of stem cells for spinal cord injury and the questions of the relative contributions of local cell damage versus axonal disconnection.

Over the past few years stem cells have become something of a Holy Grail. The concept of a stem cell is one that can divide indefinitely, that will, like the genie of the lamp, become whatever the master requires, and that can be transplanted to repair virtually any affliction of the nervous system. It is, therefore, a sobering thought that although the haematopoietic stem cell has been identified and characterised for 40 years, and is readily available, many forms of leukaemia and radiation sickness can still be incurable. If we are still uncertain how to obtain beneficial effects with a tissue such as blood, which has no structural organisation, how much greater are the problems we must expect to encounter in the brain and spinal cord, the most complex tissue known in biology?

There is a gold rush feel to the stem cell area, and many of the claims currently being staked owe as much to hope as to practicality. Many basic questions remain to be solved. What range of cell types does the term stem cell include? How can we direct their development so that they become specific cell types? And, having done so, how can they be introduced into the nervous system in such a way that they will integrate themselves, detect deficiencies, and repair them? And to what extent do we have a clear concept of the dangers in using stem cells? But notwithstanding these unsolved issues, the concept that there exist, not only within the embryo, but also within the adult, cells with as yet uncharted reparative potential offers real hope for a new way to treat injuries and diseases for which there is currently no cure.

G Raisman

Perspectives in affective disorders, Vol 21

Edited by W P Kaschka. Published by Karger, Basel, 2002, pp 204, €134.50. ISBN 3-8055-7439-8

This book is a summary of an international symposium held in September 2001 to celebrate the 25th anniversary of the depression unit at the Weissenau Centre for Psychiatry in collaboration with the University of Ulm. This unit was founded as the first of its kind in Germany for the treatment of affective disorders and was the start of a development that has led to there being over 60 special depression units in that country. The symposium included a survey of past work and a summary of the present position and future prospects in basic research, diagnosis, therapy, and the care of affective disorders.

Perhaps the most interesting part of this book is the first section of three chapters, which details the development of services for depressed patients in Germany. An elective admission to a purpose built and managed unit for the assessment and treatment of severe depression is extremely unusual in the UK, with its "one size fits all" inpatient care strategy. Intuitively depression units are appealing, but it is disappointing that no evidence is presented in this volume to support the statement that they result in a great improvement in outcome, apart from a claim that 90% of patients would recommend the unit to friends or relatives.

The second section covers basic research in affective disorders, with contributions on genetics, functional imaging, and autonomic system control in depression. The third section of eight chapters is subtitled "Therapeutic perspectives in affective disorders". There are some genuinely novel and valuable contributions here, including chapters on pharmacogenetic aspects of antidepressants, molecular mechanisms of action of mood stabilisers, and mechanisms and management of weight gain.

This book is likely to have a limited readership outside Germany. The layout and presentation are rather dreary and although it will attract psychiatrists and psychologists with a specialist clinical or research interest in affective disorders, general clinicians are likely to pass it by.

C Bench

Obsessive compulsive disorder: a practical guide

Edited by Naomi Fineberg, Donnatella Marazziti, and Dan J Stein. Published by Martin Dunitz Publishers, London, 2001, pp 200, £24.95. ISBN 1-85317-919-1

The stated aim of this book is to provide a practical and accessible guide to the diagnosis, assessment, and management of obsessive compulsive disorder (OCD). The 14 chapters include contributions from an international panel of expert clinicians and a final chapter, The patient's perspective, from a psychologist who also has OCD.

The first four chapters present an overview of the nosology, epidemiology, psychopathology, and assessment of OCD. A chapter on quality of life is followed by three chapters that summarise the neurobiology of OCD in terms of genetic factors, neuroanatomy, and neurochemistry. The final section of the book provides chapters on pharmacological and psychological treatments for OCD, including treatment resistant cases and children and adolescents.

The strength of this book lies in the detail of the discussion of the subtleties of clinical assessment, pharmacotherapy, and psychotherapy. There are a number of clinical pearls contained in these chapters, which will help clinicians to ensure that their patients receive the most effective and appropriate treatments available. The chapter on integrated treatment approaches highlights the gap in evidence whereby it is still uncertain whether combining drug treatment with exposure therapy is any more effective than drug treatment given alone. Although busy general adult psychiatrists are unlikely to ever use the Yale-Brown Obsessive Compulsive Scale, included as an appendix, its inclusion helps to highlight the need for systematic assessment of target symptoms over the relatively prolonged timescale of response to treatment.

As a stand alone text this has many merits and can be recommended to anyone who is involved in the assessment and treatment of OCD. For those more interested in the neurobiology it provides a stimulating introduction with good references to the more detailed literature.

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Perspectives in affective disorders, Vol 21

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