because of its focus on memory impairments, the concept of MCI should be used with caution outside the field of Alzheimer's disease research. The concept of CIND appears to be applicable more widely, but its current broad and non-specific definition remains a limitation. Thus the nature and magnitude of the cognitive impairments should also be taken into account when the classification of CIND is used. Future prospective studies, using clear criteria, should resolve whether the concept of CIND could serve to identify DM2 patients who are at increased risk of developing dementia.

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References

- Stewart R, Liolitsa D. Type 2 diabetes mellitus, cognitive impairment and dementia. *Diabet Med* 1999;16:93–112.
- 2 Peila R, Rodriguez BL, Launer LJ. Type 2 diabetes, APOE gene, and the risk for dementia and related pathologies: the Honolulu-Asia aging study. *Diabetes* 2002;51:1256–62.
- Petersen RC, Stevens JC, Ganguli M, et al. Practice parameter: early detection of dementia: mild cognitive impairment (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2001;56:1133-42.
 Tuekko H, Frerichs R, Graham J, et al. Five-year
- 4 Tuokko H, Frerichs R, Graham J, et al. Five-yea follow-up of cognitive impairment with no dementia. Arch Neurol 2003;60:577–82.
- 5 Ritchie K, Artero S, Touchon J. Classification criteria for mild cognitive impairment: a population-based validation study. *Neurology* 2001;56:37–42.

Excessive daytime sleepiness in migraine patients

Headache and sleep disorders are related in several ways. Sleep disorders occur in headache patients, headache is a common manifestation of sleep disorders, and secondary disorders may cause headache and sleep complaints. Excessive daytime sleepiness (EDS) or excessive somnolence is a common symptom, with a prevalence of 10-20% in the general population.¹

EDS is a subjective feeling of a compelling need for sleep at unusual times and in abnormal environmental conditions. Sleep deprivation, sleep fragmentation, and hypoxia are believed to be the main mechanisms leading to EDS. EDS increases the risk of car accidents, causes health status and quality of life to deteriorate, and may increase mortality. EDS is associated with obstructive sleep apnoea syndrome, brain tumours, epilepsy, stroke, degenerative diseases, trauma, multiple sclerosis, and neuromuscular disorders.1 The prevalence, mechanisms, impact, diagnosis, and treatment of EDS have never been assessed in migraine patients

We studied 200 consecutive patients with chronic or episodic migraine diagnosed according to the second edition of the International Headache Society diagnostic criteria for migraine² from the Jefferson Headache Center, Philadelphia, USA . The Epworth sleepiness scale (ESS)³ was applied to all patients and correlated with the diagnosis of chronic/episodic migraine, age, sex, body mass index (BMI), and headache frequency. Questions on mental and physical fatigue, concentration, and memory problems were rated using a 1 to 5 scale. The local ethics committee approved the study. EDS was defined as an ESS score of 10 or more.

Statistical analysis was done using the χ^2 and Fisher exact tests for proportions, and Spearman and Pearson's correlation tests. The level of significance was set at p<0.05.

Demographic data are given in table 1. Headache after dozing off was reported in 35% of all migraine patients (29% episodic, 40% chronic), and in 70% of patients with EDS. The chance of dozing off in a car was high in 1% of patients, moderate in 2%, and slight in 15%. The ESS correlated with mental fatigue, physical fatigue, concentration, and memory complaints (p<0.05), but did not correlate with BMI, age, or sex (NS). The mean (SD) ESS was 8.4 (4.3). An ESS score of 10 or more was present in 37% of all people with migraine, in 32.4% of those with episodic migraine, and in 39.8% of those with chronic migraine. A score of 15 or more was present in 10% of all migraine sufferers, in 15.3% of those with chronic migraine, and in 4.3% of those with episodic migraine (p<0.05; table 1).

Comment

EDS is increasingly recognised as a significant public health problem.¹ It is common in migraine compared with the general population, with around a twofold increased prevalence in our migraineurs.

The risk of car accidents is assessed in other medical disorders based upon daytime sleepiness severity. Little attention has been paid to the risk of car accidents in migraine sufferers. EDS should be evaluated in this population because of the risk of accidents in those who report severe EDS.

EDS was correlated with fatigue in the migraine patients in our study. Fatigue has been reported in 85% of chronic migraine sufferers, and was found to be very common as a premonitory symptom in migraine.⁴ Understanding the causes of EDS in migraine may shed light on the mechanisms of fatigue in these patients

Dozing off was recognised as a headache trigger in 35% of patients and in 70% of

Table 1Demographic data andEpworth sleepiness scale in 200patients with episodic and chronicmigraine

Demographic data Age (years) Sex (F/M) BMI Episodic migraine Chronic migraine	45 (12.5) 162/38 (81%/19%) 27.8 (6.0) 72 (36%) 128 (64%)
Epworth sleepiness scale Mean (SD) score	8.4 (4.3)
<i>Score 10 or more</i> All migraine Episodic migraine Chronic migraine	37% 32.4% 39.8%*
Score 15 or more All migraine Episodic migraine Chronic migraine	10% 4.3% 15.3%†
Values are mean (SD) or n (%). *NS; †p<0.05. BMI, body mass index.	

patients with EDS. EDS may aggravate migraine, and diagnosing and treating it may lead to better outcomes.

Sleep loss or inadequate sleeping time is the most common cause of EDS in the general population. Primary sleep disorderssuch as sleep disordered breathing, restless legs syndrome, and periodic leg movements in sleep-are prevalent, particularly among older people and may contribute to EDS. Other medical conditions, such as cardiovascular and pulmonary diseases, psychiatric illness, chronic pain syndromes, and several neurological and neurodegenerative disorders, can disrupt sleep and lead to EDS. Moreover, drugs including diuretics, antihypertensives, sympathomimetic agents, corticosteroids, sedative-hypnotics, analgesics, and certain antidepressants can cause EDS by interfering with sleep continuity or by having a direct sedating effect in the daytime.

Can migraine lead to EDS, is EDS the primary condition leading to migraine, or are migraine and EDS determined by different causes? All three possibilities may occur. First, EDS may be an accompanying symptom in migraine, and an increased EDS may be a result of having migraine; the frequency of migraine may also affect EDS, as our chronic migraineurs scored higher. Second, EDS may precipitate migraine attacks-in our study dozing off was reported to be a headache trigger in 35% of migraine patients and in 70% of those with EDS. Third, depression could be related to both migraine and EDS, because it is comorbid with migraine and can cause EDS. A control group and the evaluation of depression and anxiety symptoms would help to clarify the exact relation between EDS and migraine.

We previously hypothesised a hypothalamic involvement in chronic migraine.⁵ The hypothalamus is potentially the mediator of EDS in migraine patients. Orexin, a recently described neuropeptide, is thought to play a role in the regulation of food intake, sleepiness, autonomic nervous system activity, and energy balance. Orexin containing cells are located in the lateral hypothalamus, with widespread projections to the entire neuroaxis. Input from the suprachiasmatic nucleus to orexin containing neurones may explain the occurrence of clock dependent alertness. Orexin cells drive monoaminergic activity across the sleep cycle and this is related to pain modulation. A recent study showed that injection of orexin A in the posterior hypothalamic area decreased the A and C fibre responses to dural electrical stimulation and decreased spontaneous activity, while orexin B had the opposite effect, supporting a role of orexins A and B in the nociceptive processing of meningeal input.⁶

No specific treatment is available for EDS in migraine, though possibly physical exercise may play a role. Polysomnography, the multiple sleep latency test, and the ESS are useful tests for evaluating EDS in migraine patients; however, their clinical relevance has yet to be determined.

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References

- Carskadon MA. Evaluation of excessive daytime sleepiness. Neurophysiol Clin 1993;23:91–100.
- sleepiness. Neurophysiol Clin 1993;23:91-100.
 Headache Classification Subcommittee of the International Headache Society. In: The international classification of headache disorders, 2nd ed.Cephalalgia, 2004;24(suppl 1):1-160.
- Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep 1991-14-540-5
- 4 Giffin NJ, Ruggiero L, Lipton RB, et al. Premonitory symptoms in migraine: an electronic diary study. Neurology 2003;60:935–40.
- 5 **Peres MF**, Sanchez del Rio M, Seabra ML, *et al.* Hypothalamic involvement in chronic migraine.
- J Neurol Neurosurg Psychiatry 2001;71:747–51.
 Bartsch T, Levy MJ, Knight YE, et al. Differential modulation of nociceptive dural input to [hypocretin] Orexin A and B receptor activation in the posterior hypothalamic area. Pain 2004;109:367–78.

BOOK REVIEWS

Neurological rehabilitation of stroke

Edited by Nick Losseff. Published by Taylor & Francis Books Ltd, 2004, £29.95 (hardcover), pp 100. ISBN 1841843229

This is the second book in the Queen Square Neurological Rehabilitation Series and follows the same formula as the first, with chapters on the molecular basis of neural repair and recovery following stroke, the interdisciplinary team, the impact of stroke from both medical and patient viewpoints, outcome assessments, and models of service delivery. Successful neurological rehabilitation after stroke is achieved by working as an interdisciplinary team, and the book follows this principle with each chapter written by different members of the team, including the patient.

The first chapter is a summary of recovery after stroke from the basic science level and how this applies to current stroke management. It describes animal and human models of brain plasticity, the potential for functional recovery, the biochemical changes around the time of a stroke, and studies of pharmacological management in stroke recovery. It is fairly detailed and includes a comprehensive reference section.

The theme of the second chapter is the interdisciplinary team. It explains the roles of team members in the acute rehabilitation of stroke, identified as the first 6 weeks after the event. Emphasis is made on the change from the multidisciplinary team, where individuals work alongside each other on disciplinespecific goals; to an interdisciplinary team approach with team members working together on mutually set goals with high levels of communication. The principle of using the World Health Organization (WHO) International Classification of Function (ICF) system rather than the International Classification of Impairment, Disability, and Handicap to plan goal-orientated rehabilitation is explained. This chapter gives quite a general overview of the interdisciplinary team, but specific points are illustrated in a case summary at the end.

The medical view of stroke is the subject of the third chapter. It is both concise and comprehensive in the description of the pathogenesis, mortality, incidence, and prevalence of stroke. It discusses outcome in terms of impact at the patient and population level using the ICF system and considers the implications on resources of the National Sentinel Stroke Audit 2001/2 and the Royal College of Physicians Stroke Guidelines.

The next chapter describes the impact of stroke from a personal view and despite it being the shortest chapter it is perhaps the most essential for those involved in stroke management. It is written by Robert McCrum, author of "My Year Off", and gives a brief summary of his own experience of a stroke. He includes a particularly constructive Do's and Don'ts list.

The fifth chapter looks at evaluating rehabilitation as an intervention following stroke, using outcomes at the patient level and more broadly at the provider and population level. It includes the use of the ICF system of impairment, activity and participation, health related quality of life from the patient perspective, and also the evidence for stroke rehabilitation and cost-effectiveness of stroke care.

The topic for the final chapter is service delivery and it details the evidence for the need for specialist stroke management and describes the models of care currently used to achieve this.

The writers emphasize that this book only covers the first 6 weeks of rehabilitation after a stroke and it would be a mistake to come away from reading it with the impression that the rehabilitation process ends there. Further rehabilitation usually continues in an inpatient neurological rehabilitation unit or in the community. A patient's functional level may continue to improve over many months, and in some cases years after the acute event. In summary this handy textbook provides a concise and accessible overview of the acute rehabilitation of stroke. It follows an evidence-based approach and includes a comprehensive reference list. I would recommend it to anyone involved in stroke rehabilitation and those new to neurological rehabilitation would find it particularly useful.

R Botell

Cognitive neuropsychology of Alzheimer's disease

Edited by Robin Morris, James Becker. Published by Oxford University Press, Oxford, 2004, £60.00 (hardcover), pp 422. ISBN 0-19-850830-1

This book tackles issues relating to the neuropsychology of Alzheimer's disease (AD) at a number of levels, ranging from detailed accounts of specific cognitive deficits, to the global patterns of impairment seen with different presentations of the disease, to the broad theoretical and clinical approaches that should be adopted when examining cognitive dysfunction across groups, subgroups, and individuals.

The early chapters of the book provide a detailed introduction to Alzheimer's disease, which is not restricted merely to discussion of the history and general characteristics of the condition, but also tackle a number of much broader questions that weigh upon how our approaches to understanding, investigating, and managing the disease should evolve and develop in the coming years. A particularly excellent chapter by Gray and Della Sala on measuring impairment and charting decline will be of special interest to young researchers wishing to avoid the pitfalls and understand the complexities of tracking objectively a heterogeneous disease across different time points, age bands, and subject groups.

Latterly, a number of specific cognitive neuropsychological disorders are discussed in detail, with clear reference to both their clinical and diagnostic value and also their contribution to our theoretical understanding of brain function and organisation. Among the topics covered in depth, it was somewhat surprising not to find a chapter dealing with visuoperceptual and visuospatial abilities. Given their importance not only for distinguishing between AD and other forms of dementia-for example, frontotemporal lobar degeneration, but also for understanding certain atypical presentations of the disease-for example, biparietal AD, these cognitive skills perhaps merit more than the cursory mention they receive elsewhere in the textbook.

Although the title of the volume might suggest a rather narrow focus upon cognitive aspects of AD, the editors have been more ambitious, and indeed successful, in placing the neuropsychology of AD within the context of the neurobiology, pharmacology, and treatment of the disease. As a result, this textbook represents an important contribution to both the specific and general education of cognitive neuropsychologists, and that of individuals approaching the topic of Alzheimer's disease from a variety of alternative clinical and scientific backgrounds.