

PostScript

LETTERS

Leech therapy in the treatment of median nerve compression due to forearm haematoma

A 67 year old patient underwent interventional cardiac catheterisation for cardiac angina. An 80% stenosis of the left anterior descending artery and a 90% stenosis of the posterior descending artery were stented. A distal stenosis of the left anterior descending artery was dilated. Catheterisation was performed via the right radial artery. The patient received a combination of clopidogrel, acetylsalicylic acid, and intravenous heparin. One hour after treatment, the patient noticed diffuse pain in the right forearm as well as numbness in the thumb and index and middle fingers, with subsequent weakness of the thenar muscles and difficulty in flexion of the distal phalanges of the thumb and index finger. In addition, the patient noticed increasing swelling and subsequent hardening of the right forearm together with a blue-red discoloration.

Neurological examination at this time revealed a sensory deficit of the right median nerve with decreased pinprick and light touch sensation as well as impaired two point discrimination. The motor function of the small thenar muscles and flexor pollicis longus, and flexor digitorum profundus of the index and middle fingers was slightly impaired. The pronator teres function was unremarkable. On examination, there was massive swelling of the forearm and blue-red discoloration. The circumference of the right forearm was increased by 5 cm compared with the left. However, the right radial artery pulse could be palpated without difficulty. Clinically, the patient was diagnosed as having compartment syndrome of the right forearm with affection of the median nerve due to a postpuncture haematoma following cardiac interventional therapy. Because of the close relation of the syndrome with the local puncture for cardiac catheterisation, and in order not to delay treatment, we did not perform neurophysiological studies or imaging studies such as sonography.

A hand surgeon was consulted and surgical incision and removal of the haematoma were considered. However, as the functional impairment of the nerves and muscles was only moderate at this time we initiated therapy with medicinal leeches. A total of 13 leeches were applied to the volar surface of the right forearm. All leeches bit into the skin and sucked about 145 ml of blood. The patient's symptoms markedly improved within 24 hours. On the next day, only slight sensory disturbances persisted on the skin of the right thumb. No further therapy was needed.

At follow up three months later, the patient was noted to have undergone cardiac bypass surgery. Neurologically, the median nerve function was normal. At this time, motor and sensory nerve conduction velocities and amplitudes, as well as distal motor latency of the median nerve, were normal.

Discussion

The transradial approach is increasingly being used for cardiac catheterisation and also for cerebral angiography. In general, this approach is considered to be associated with fewer major complications, requires a shorter observation period, and there is no need for bed rest.¹ Despite a generally low complication rate, a variety of adverse events, although rare, can occur such as transient radial artery spasm, failure to access the brachial artery, radial artery occlusion, radial artery perforation, pseudoaneurysm, skin desquamation, severe pain and forearm haematoma.¹ The haematoma can lead to compartment syndrome, which can compromise the viability and function of the nerves and muscles.²⁻³ The treatment depends on the severity of the symptoms, current neurological status, and the integrity of the radial artery. In our patient, we initiated treatment with medicinal leeches shortly after onset of the symptoms and establishment of the diagnosis.

In the past years, the use of medicinal leeches (*Hirudo medicinalis*, fig 1) has been rediscovered as an effective method to relieve venous congestion.⁴ The treatment aims to counteract tissue ischaemia, hypoxia, acidosis, necrosis, and gangrene. The possible mechanism of action of leech therapy is based on the anticoagulant properties of hirudin (contained in leech saliva) and the capacity of the leech to suck the blood thereby relieving the pressure in the affected compartment.⁵ In particular, medicinal leeches have been very effective in regions with diffusely spreading haematomas such as in the tongue or scrotum.^{6,7}

Medicinal leeches are commercially available in pharmacies. Before use, the skin has



Figure 1 *Hirudo medicinalis* (medicinal leech). The leeches in action on the forearm of a patient with forearm haematoma.

to be cleaned with warm water. In case of a haematoma as in our patient, up to 15 leeches per session are positioned over the skin using a wooden stick or by hand. During 30–60 minutes, each leech sucks 8–10 ml of blood. After one to two hours, the leeches fall off spontaneously. If needed, the leeches can be animated by pricking and scraping the surface of the patient's skin or by brushing a small amount of butter on the skin. After removal of the leeches the local bleeding should not be suppressed. The bleeding can persist up to 12 hours. When carried out correctly, the risk of bacterial infection is negligible.²

Leech therapy in treatment of median nerve dysfunction due to forearm compartment syndrome following transradial catheterisation has not hitherto been reported in the literature. Recently, Avci *et al* reported successful leech therapy in the treatment of digital neurovascular compression due to a forgotten digital tourniquet.⁸ We suggest leech therapy as a treatment option in similar conditions, although a hand surgeon should be available in case a surgical procedure becomes necessary.

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Mild impairments in cognition in patients with type 2 diabetes mellitus: the use of the concepts MCI and CIND

Type 2 diabetes mellitus (DM2) is associated with moderate cognitive impairment in verbal memory, mental flexibility, and information processing speed, while other cognitive functions remain relatively unaffected.¹ Moreover, epidemiological studies have shown that DM2 patients have a twofold increased risk of developing either vascular dementia or Alzheimer's disease.^{1,2} In the present study we examined whether mild cognitive impairment (MCI) and "cognitive impairment, no dementia" (CIND)—two concepts that are used to describe cognitive impairment in the transitional state between normal aging and early dementia—can be applied to the cognitive impairments encountered in a population based sample of DM2 patients. Recently, these concepts have attracted considerable attention, as individuals who meet the criteria for either MCI or CIND are known to have a substantially increased risk of developing dementia.^{3,4} MCI is defined as a memory deficit without impairments in other cognitive domains.³ Patients with MCI develop Alzheimer's disease at an annual incidence of between 6% and 25%, compared with 0.2–3.9% in the general population of the same age. The broader concept of CIND is used to describe more general cognitive impairments, often encountered in relation to vascular risk factors. The diagnosis requires impairment in one or more cognitive domains and no dementia.⁵ A fivefold increased risk of developing Alzheimer's disease, vascular dementia, or mixed Alzheimer/vascular dementia has been reported in patients with CIND.⁴ If either of the concepts MCI or CIND is found to be useful in describing the cognitive problems in DM2, it may help identify DM2 patients who are at increased risk of developing dementia.

Participants were recruited for the Utrecht diabetic encephalopathy study, which assesses the impact of macrovascular and microvascular disease on cognition in DM2. This research was approved by the medical ethics committee of the University Medical Centre Utrecht. Patients (n = 90) were aged between 60 and 75 years, were known to have had DM2 for at least one year, and were recruited through their general practitioner. Age and education matched control participants (n = 40) were recruited through the patient (mostly spouses) (table 1). Exclusion criteria were a psychiatric or neurological disorder (unrelated to DM2) that could influence cognitive functioning, a history of alcohol or substance abuse, and dementia. All participants were functioning independently at home and had intact comprehension of the Dutch language. The participants had an extensive neuropsychological examination (11 tasks) addressing the following cognitive domains in both a verbal and a non-verbal form: abstract reasoning, memory, working memory, information processing speed, visuo-construction, attention, and mental flexibility.

Previous studies used variable case definitions of CIND and MCI, often based on the diagnostic opinion of experienced clinicians. We preferred a numerical approach, comparing test scores with available age and education adjusted normative data. This procedure

results in an objective classification and facilitates comparison of different studies. Performance of the participants on each test was rated as either within the normal range (0), below average (1), or impaired (2). "Normal performance" was defined as performance between -1 SD and $+1$ SD from the normative mean, "below average" as between -1 SD and -1.65 SD from the normative mean (the lowest 16% of the normal population), and "impaired" as below -1.65 SD from the normative mean (the lowest 5% of the normal population). Performance on a cognitive domain as a whole was classified as impaired when the average rating of tests in that domain was >1 .

Participants were classified as having CIND if they were impaired in one or more of the cognitive domains.⁵ When memory was the only affected domain, the participant was also classified as having MCI, applying the Petersen criteria.³ As a decrease in information processing speed is common in an elderly population, impairment in this domain had to be accompanied by a rating of 1 or 2 in more than half the tasks in another domain (average rating of tests in that domain >0.5) for the individual to be classified as CIND.

Overall, the number of tasks on which performance was impaired was higher in DM2 patients than in controls (patients: five tasks, interquartile range two to eight; controls: two tasks, interquartile range one to five; Mann-Whitney U test, $p < 0.05$). The domains that were affected most often in both groups were mental flexibility (patients

11%; controls 10%) and information processing speed (patients 17%; controls 11%). Memory was relatively spared (patients 4%; controls 3%). Significantly more patients than controls met the criteria for CIND (χ^2 test, $p < 0.05$; table 1). The proportion of participants classified as having MCI did not differ between the two groups. In addition, hypertension, major vascular events (excluding non-invalidating stroke), retinopathy, and neuropathy were more common in the DM2 patients (table 1). Within the DM2 group no significant differences were found between the patients with or without CIND for age, diabetes duration, HbA_{1c}, or any of the factors described above. The same applied for the controls with or without CIND.

Comment

The results show that DM2 patients overall had more cognitive impairments than control participants, predominantly affecting mental flexibility and information processing speed; these cognitive domains are known to be the most sensitive to cognitive decline associated with aging. The prevalence of MCI and CIND in the control group was comparable to previous population based studies.^{3,5} CIND, but not MCI, was significantly more common in DM2 patients than in controls. Memory impairment, which is the main feature of MCI, was not the most prominent impairment in the DM2 patients. Rather, a more general pattern of cognitive impairment, affecting multiple domains, was observed. This pattern fits better within the broader concept of CIND. These results illustrate that,

Table 1 Characteristics of the participants and the number and relative frequencies of participants classified as having CIND or MCI

	DM2 patients (n = 90)	Controls (n = 40)	Statistics*
Demographic characteristics			
Age (years)	66.8 (5.5)	65.2 (4.5)	NS
Educational level (median, IQR)	4 (3 to 5)	4 (3 to 5)	NS
Male sex	47 (52%)	18 (45%)	NS
Estimated premorbid IQ	98.3 (16.9)	103.2 (15.2)	NS
Impaired cognitive function			
CIND	34 (38%)	8 (20%)	$p < 0.05$
MCI	2 (2%)	0 (0)	NS
Medical characteristics			
DM2 duration (years)	9.6 (6.5)	–	NA
Treatment			
Diet	59 (65%)	–	NA
Oral medication	25 (28%)	–	NA
Insulin	6 (7%)	–	NA
HbA _{1c} (%)	7.0 (1.2)	5.5 (0.3)	$p < 0.05$
Hypertension†	67 (74%)	14 (35%)	$p < 0.05$
Non-invalidating stroke	5 (6%)	2 (5%)	NS
Other major vascular events‡	21 (23%)	3 (8%)	$p < 0.05$
Retinopathy§	29 (32%)	0 (0)	$p < 0.05$
Neuropathy¶	38 (42%)	7 (18%)	$p < 0.05$
Depression**	5 (6%)	0 (0)	NS

Values are mean (SD) or n (%) unless specified.

*Between group comparisons for parametric data were done with *t* tests, for non-parametric data with the Mann-Whitney U test, and for proportions with χ^2 tests.

†Systolic pressure >160 mm Hg, diastolic pressure >95 mm Hg, or use of antihypertensive drug treatment.

‡Myocardial infarction and/or vascular surgery or endovascular intervention.

§Diabetic retinopathy severity scale as used in the Wisconsin epidemiological study of diabetic retinopathy, cut off score 1.5.

¶Toronto clinical neuropathy scoring system, cut off score 5.

**Beck depression inventory, cut off score 16.

CIND, cognitive impairment, no dementia; DM2, type 2 diabetes; IQ, intelligence quotient; IQR, interquartile range; MCI, mild cognitive impairment; NA, not applicable.

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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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 1 Demographic data and Epworth sleepiness scale in 200 patients with episodic and chronic migraine

Demographic data	
Age (years)	45 (12.5)
Sex (F/M)	162/38 (81%/19%)
BMI	27.8 (6.0)
Episodic migraine	72 (36%)
Chronic migraine	128 (64%)
Epworth sleepiness scale	
Mean (SD) score	8.4 (4.3)
Score 10 or more	
All migraine	37%
Episodic migraine	32.4%
Chronic migraine	39.8%*
Score 15 or more	
All migraine	10%
Episodic migraine	4.3%
Chronic migraine	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 disorders—such 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.¹

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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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 discipline-specific 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 visuospatial 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.

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