Neurologist-in-training

The aim of this section is to prepare the neurologist-in-training for the FMH examination, to confront her or him with specific problems of everyday neurological practice and to give him or her updates on recent controversies in clinical neurology.

Contributions and correspondence to Andrea O. Rossetti who is responsible for this section:
Andrea O. Rossetti, Neurologie, CHUV, 1011 Lausanne, andrea.rossetti@chuv.ch

Neurological MCQ

J. Novy, P. Michel

Service de Neurologie, CHUV, Lausanne

Select the correct answer(s).

1 Which one among these diseases is not a cause of a stiff-legged gait?

A cervical myelopathy
B cerebral palsy
C stiff-person syndrome
D demyelinating disease
E SCA-1 and SCA-2
F familiar dystonia

2 All but one is true for freezing gait:

A Difficulty with the initiation and maintenance of locomotion.
B Movements are often or near normal once the patient is walking.
C Freezing may return as the patient changes direction.
D Freezing is often overcome with sensory cues.
E There may be stepping movements with side-to-side shuffling without forward engagement.
F It is only present in Parkinson's disease.

3 Which one among these diseases is not a cause of freezing gait?

A Parkinson's disease
B normal pressure hydrocephalus
C Lewy body disease
D progressive supranuclear palsy
E multiple sclerosis
F subcortical vascular dementia

4 Two of the following diseases are not causes of sensory ataxia:

A SCA-1
B chemotherapy
C paraproteinaemia
D paraneoplastic subacute sensory neuropathy
E normal pressure hydrocephalus

5 Which assumption concerning psychogenic gait is false?

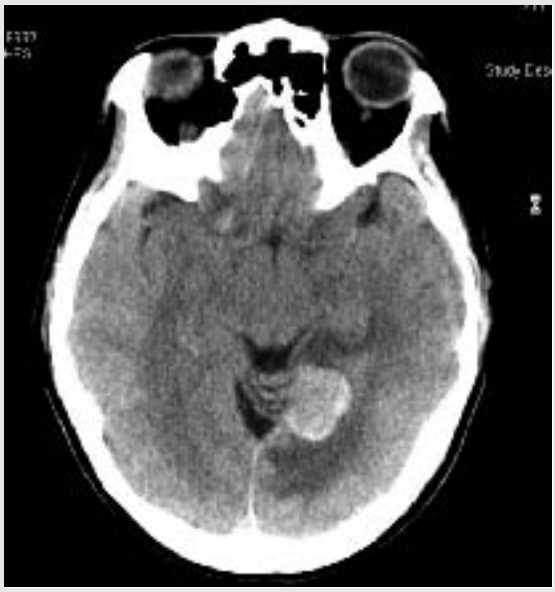
A Prognosis is worse if history is recent.
B Prognosis is worse if the disorder has been persisting for longer than six months.
C Dramatic fluctuation may occur over minutes.
D Uneconomical postures with wastage of muscular effort are common.
E Psychiatric history is sometimes revealing.

(For correct answers, see page 181)

Neuroimaging

■ **A. O. Rossetti**

Service de Neurologie, CHUV, Lausanne



1

A 54-year-old woman is admitted to the emergency department following her first generalised convulsive seizure during the day, which was preceded by the vision of white flashes on her right. She does not have any provoking factors (sleep disturbances, alcohol intake, medications).

What is your differential diagnosis on the CT scan performed w/o contrast injection?

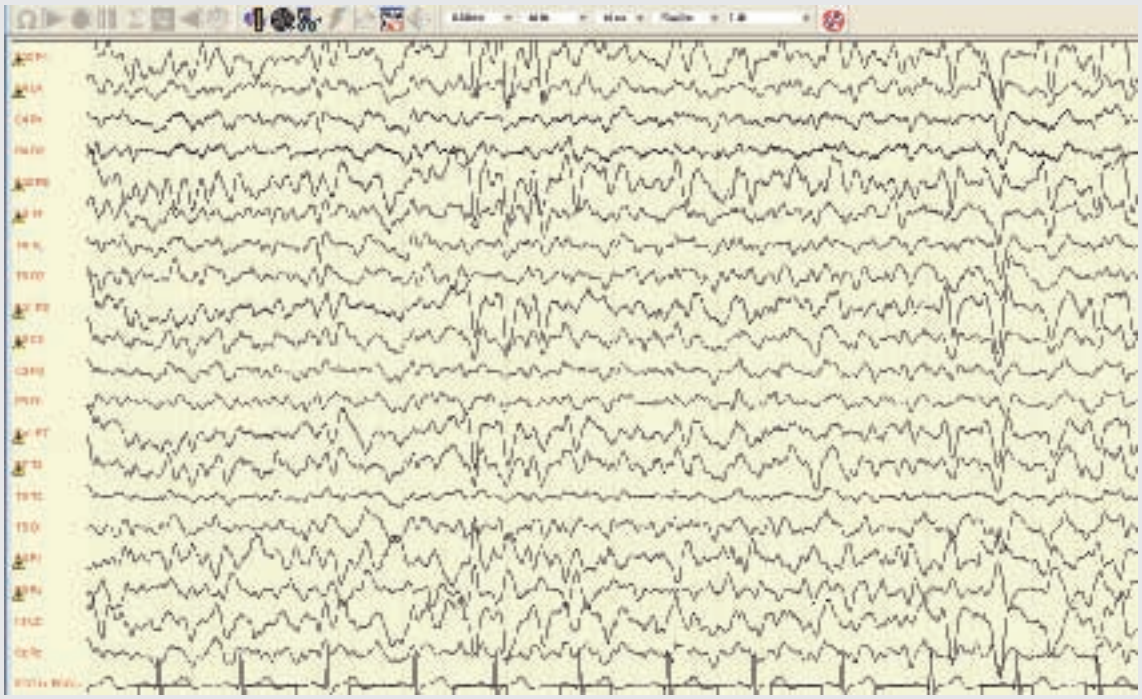
(For correct answers, see page 181)

2

A 61-year-old woman presents in a subacute confusional state 3 days after the beginning of a flu. On examination, she has verbal stereotypes with a markedly reduced digit span, moderate meningeal irritation signs and a bilateral grasping. Her consciousness fluctuates from one minute to the other between light somnolence and stupor. She is on no medication, but her sodium is 130 mmol/ and her CSF shows 45 lymphocytes/ml.

What are your thoughts regarding her EEG (bipolar sagittal "double banana" montage; 10 mm/sec, 7 µV/mm)?

(For correct answers, see page 181)



Read for you

A. O. Rossetti

Service de Neurologie, CHUV, Lausanne

Carotid stenosis: to cut or to stent?

Endarterectomy has been shown to be an effective treatment for symptomatic carotid stenosis, with a marginal benefit for patients with 50–69% stenosis and a clear benefit for those with >70% stenosis [1]; especially if the procedure is performed within 2 weeks after stroke [2]. Stenting carries fewer risks of local complications, but a systematic review of existing trials failed to show its superiority as compared to endarterectomy [3]. The French randomised trial was initiated in 2000 and interrupted in 2005 [4].

Patients with symptomatic carotid stenosis 60–99% (NASCET criteria) and low or moderate operative risk were enrolled; the aim was to show non-inferiority of stenting. Surgeons had to have performed at least 25 endarterectomies and interventional physicians at least 12 stentings; after 2003, all stentings were performed using cerebral protection. The primary endpoint was occurrence of any stroke or death within 30 days after treatment.

Among 520 patients completing the procedures, this endpoint had an incidence of 3.9% after endarterectomy and 9.6% after stenting, with a relative risk of 2.5 (95% CI, 1.2–5.1). The incidence was lower for subjects stented with protection devices (7.9 vs 25%, $p = 0.03$). Cranial nerve injury was more frequent after endarterectomy (7.7 vs 1.1%, $p < 0.001$), and the hospital stay was shorter after stenting ($p = 0.01$).

The striking difference in the primary endpoint certainly justified the premature interruption of the trial. As the authors note, the incidence of the primary endpoint among endarterctomised patients was lower in this study than in previous ones, even if the majority received general anaesthesia, probably owing to the development of better perioperative risk management over time. Therefore, it cannot be excluded that stenting, as a relatively new procedure, is "suffering" from being at the beginning of the "learning curve". Other potential problems affecting the quality of the results in stented subjects are the inhomogeneous use of protection devices and the "generous" requirement of only 12 previous procedures (as compared to 25 operations) for a participating endovascular physician. Furthermore, the study was not blinded, this being less a problem for death recording as for assessment of minor stroke.

Practically, this study prompts these considerations: firstly, at present time it is probably reasonable, outside study protocols, to prefer endarterectomy in patients with low or moderate operative risk; secondly, further studies designed more carefully, especially regarding training of participating physicians, uniform protocol of stenting procedure and blinded assessment, are needed (several are indeed ongoing).

References

1 Rothwell PM, Eliasziw M, Gutnikov SA, Fox AJ, Taylor DW, Mayberg MR, et al. Analysis of pooled data from the randomised controlled trials of endarterectomy for symptomatic carotid stenosis. Lancet. 2003;361:107–16.

2 Rothwell PM, Eliasziw M, Gutnikov SA, Warlow CP, Barnett HJ; Carotid Endarterectomy Trialists Collaboration. Endarterectomy for symptomatic carotid stenosis in relation to clinical subgroups and timing of surgery. Lancet. 2004;363:915–24.

3 Coward LJ, Featherstone RL, Brown MM. Safety and efficacy of endovascular treatment of carotid stenosis compared with carotid endarterectomy: a Cochrane systematic review of the randomised evidence. Stroke. 2005;36:905–11.

4 Mas JL, Chatellier G, Beyssen B, Branchereau A, Moulin T, Becquemin JP, et al. Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. N Eng J Med. 2006;355:1660–771.

Neurologist-in-training

Answers to MCQ

1 E **2** F **3** E **4** A and E **5** A

Answers to Neuroimaging

1 There is a spontaneously hyperdense, homogenous mass in the posterior aspect of the left mesial temporal lobe, with a surrounding oedema. Theoretically, this may be due to 4 reasons: fresh blood, calcifications, accidental contrast injection or melanin.

Indeed, on targeted questioning, the patient reported a melanoma excision on her right arm 12 years before. She is therefore suffering from a "delayed" brain metastasis.

2 There is a disorganised background activity, dominated by a diffuse theta-delta slowing, with superimposed bi-frontal delta intermixed with transients having a "triphasic" appearance. One may find this image in toxic-metabolic encephalopathies or in nonconvulsive status epilepticus. This represents one of the most difficult challenges for electroencephalographists.

In the literature triphasic waves associated with encephalopathy are described as showing a second phase being greater than the first (as here), a phase lag (i.e., the nadir of a wave is not synchronous among the different leads, you may observe this on the Fp2-F8 F8-T4 derivations in the middle of the picture; note, however, the almost complete absence of triphasics in the posterior derivations, which is rather atypical for a diffuse encephalopathy) and a frequency lower than 2 Hz (here rather 3–4 Hz); furthermore, the recording should show some reactivity (which was doubtful in this case).

In consideration of the consciousness fluctuation, 0.5 mg clonazepam IV followed by 15 mg/kg phenytoin were administered; as a consequence the patient became very drowsy. On the following day she was alert and oriented, with the following EEG (see below):

A noticeable improvement, with a badly developed alpha background is seen, alternating with frontal intermittent rhythmic delta activity ("FIRDA") and triphasic waves (this complex is also called "Z waves"); the recording was reactive. The diagnosis of partial-complex nonconvulsive frontal status epilepticus on viral meningoencephalitis was therefore retained. This case illustrates the need for a clinical assessment paralleling the EEG interpretation, and the fact that the "benzodiazepine test" is surely diagnostic for nonconvulsive status if the subject awakens; however, often the patient does not become immediately alert, but only after some delay.