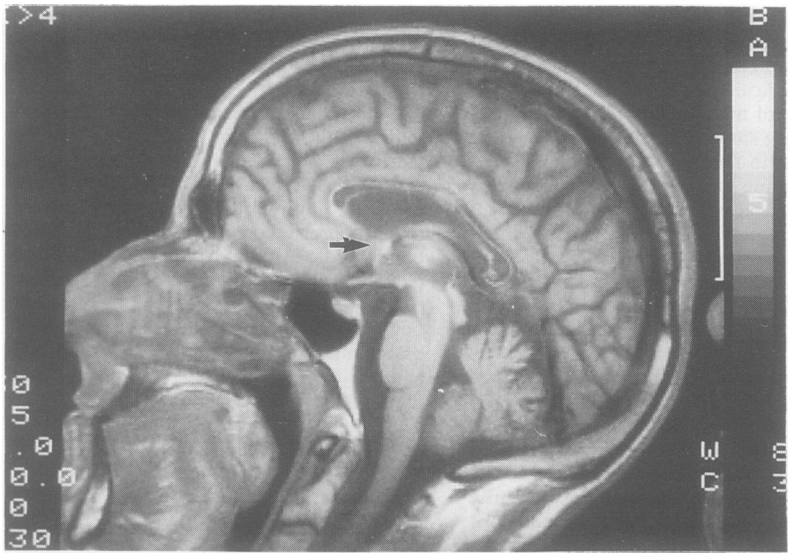

Figure Sagittal MRI of the brain, T1 weighted image (TR 600, TE 15, 1·0 Tesla) shows an extensive lesion (hyposignal) involving the whole corpus callosum. The anterior commissure was spared (arrow).

placed in his right hand. Similarly, left pseudo-hemianopia was present: the patient correctly named 2 objects out of 15 presented tachistoscopically in his left visual field, but 15 out of 15 when they were presented in his right visual field. The dichotic listening test also revealed total extinction on the left side. The patient obviously experienced difficulty in reaching objects located in the hemispace contralateral to his hand. While the patient was looking at a central visual fixation point, a lateral target (pencil) was presented in his peripheral visual field, either right or left. The patient was instructed to reach out and take this lateral target (right or left) with one hand (right or left), without moving his eyes from the central fixation point. Twenty trials were made for each of the four combinations. When using the hand ipsilateral to the lateral target, the patient easily and accurately reached this target: 20/20 correct responses were obtained for the right hand in the right hemispace, and 19/20 for the left hand in the left hemispace. However, when he had to reach a target located contralaterally to the hand used, he experienced marked difficulty: the direction of the arm movement was grossly inaccurate and the target was missed. Only 5/20 correct responses were obtained with the right hand in the left hemisphere, and 9/20 with the left hand in the right hemispace. MRI showed a lesion involving the whole extent of the corpus callosum (figure). The anterior commissure and the cerebellar peduncles were spared, and there were no visible lesions in the region of the floor of the third ventricle. There were no abnormalities in the cerebral cortex, in particular in the parietal lobes or the corona radiata.

The presence of a disconnection syndrome, associated with a large lesion affecting the corpus callosum was, in the context of severe alcoholism, compatible with Marchiafava-Bignami disease. The most interesting finding was the impairment of visually-guided reaching movements, in the absence of motor weakness and somatosensory or visual field defects. This bilateral crossed visuo-motor impairment was consistent with bilateral crossed optic ataxia, that is, a specific impairment of visuo-motor coordination.[1] Left ideomotor apraxia is a different entity from bilateral crossed optic ataxia as: 1) They are different types of movements (imitative gestures vs visually guided reaching movements); 2) The movement disorders are not observed in the same movement fields (the whole movement field vs the contralateral movement field), and 3) With the same arm (left arm vs both arms).

The crucial role played by the posterior parietal cortex in this function has been established,[1,2] and a number of cases of optic ataxia following posterior parietal lesions have been published. However, optic ataxia may be observed in the absence of a parietal lesion. The posterior parietal cortex is connected to the motor areas of the frontal lobes, ipsilaterally through parieto-frontal association fibres and contralaterally through the corpus callosum.[3] Thus a lesion affecting one of these fascicles could theoretically result in optic ataxia. A lesion affecting the intrahemispheric association fibres could result in ipsilateral optic ataxia, but as such a lesion probably also partly involves the primary motor cortex region, the ensuing motor deficit interferes with the demonstration of optic ataxia. A corpus callosum lesion could result in bilateral crossed optic ataxia. This syndrome was reported in one case of a split brain.[4] To our knowledge, no other case of bilateral crossed optic ataxia following a lesion restricted to the corpus callosum has been reported. Our case confirms that bilateral crossed optic ataxia should be included in the classic signs of the disconnection syndrome.

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MATTERS ARISING

False facilitation to repetitive stimulation

We have read with interest a letter from Drs Pullicino and Beck on incremental response to repetitive nerve stimulation in Guillain-Barré syndrome (GBS).[1] They suggested that acetylcholine (ACh) release from motor nerve terminals might be impaired in acute GBS. In motor neuron disease, abnormal decrement such as myasthenia gravis has now been well documented. However, the facilitation they showed seems to be a false positive result.

In neuromuscular block in Lambert-Eaton syndrome, compound muscle action potential (CMAP) to the first stimulus is of very low amplitude. At low frequency stimulation, a waning pattern similar to that seen in myasthenia gravis is a common finding.[2] At high frequency repetitive stimulation, marked facilitation more than 200 to 300% is thought to be confirmatory. The true facilitation results from an increase in the number of ACh quanta released, which give rise to a larger end-plate potential. Electromyographers, however, have paid attention to the false facilitation erroneously seen without true neuromuscular block.

Figure 1 is an example of false facilitation, recorded from a healthy normal subject aged 30. At 2 Hertz stimulation little change was noted. At higher frequency stimulation, successive increase in amplitude was remarkable. The rate of increment was highest at 50 Hertz stimulation, up to 180% of the initial amplitude. The critical finding is that duration of the negative phase became concomitantly shorter when the amplitude increased. As a result, the area of negative phase remained relatively unchanged, which is a typical finding for false positive CMAP changes, either increment or decrement. In true facilitation in

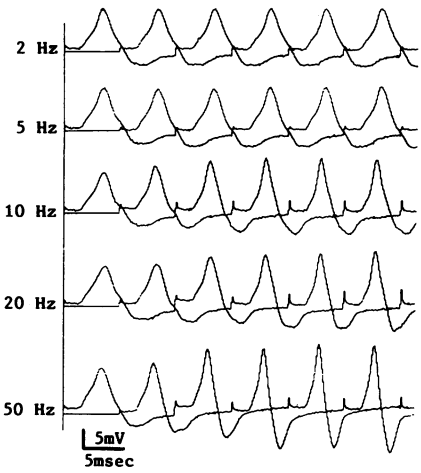

Figure 1 A train of responses recorded from the abductor digiti minimi with electrical stimulation of various frequency in a healthy subject. Smooth increment in amplitude is noted at higher frequency stimulation. As the duration became concomitantly shorter, the negative area remained relatively unchanged.

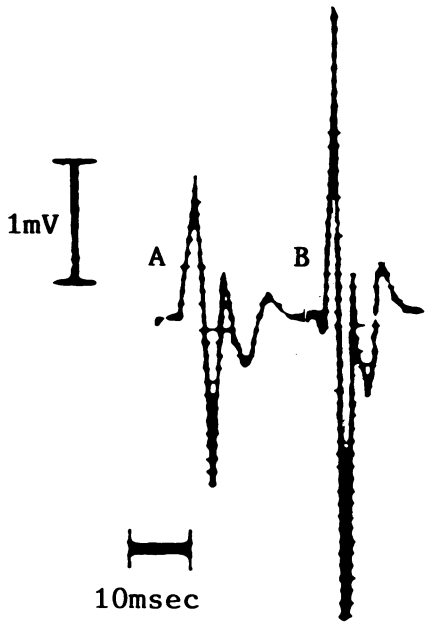

Figure 2 The initial CMAP (indicated by A) and the fifth one (B) to 50 Hertz stimulation in Guillain-Barré syndrome.[1] Instead of marked increase in amplitude, the duration of the fifth CMAP became nearly a half of that of the initial CMAP.

Lambert-Eaton syndrome or botulism, both amplitude and negative area of CMAP greatly increase.

One of the mechanisms of the false facilitation may be a physiological one due to an accumulation of calcium ion,[3] which may increase the amplitude by 10%. A more important factor is an artificial one, a change in spatial relationship of muscle and recording electrodes during testing. The muscle is sometimes very much shortened during tetanic stimulation, particularly when such high frequency stimuli as 30–50 Hertz are applied. The recording area of active electrode may change greatly as soon as the successive short interval shocks are given. This type of error can happen even at low frequency stimulation,[4] if excessive movements are not prevented.

Unfortunately, the facilitation shown by Drs Pullicino and Beck seemed to be a false one, because the increase in amplitude associated with concomitant reduction in duration of successive CMAPs as shown in fig. 2 (made from the original figure published in the journal). The negative area remained unchanged. At low frequency stimulation, they stated, that there was waxing instead of waning, which is an extremely unusual finding for impaired ACh release. We are therefore not convinced about the impaired neuromuscular transmission in GBS. Careful study will be required to confirm if there is true facilitation or not.

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Cost-effective investigations of patients with suspected TIAs

GJ Hankey and CP Warlow, discussed cost-effective investigation of patients with suspected transient ischaemic attacks.[1] The authors claim that the cranial CT scan in suspected TIA patients only allows exclusion of an underlying structural intracranial lesion, which may rarely be present (1%). The authors state that the prognosis of TIA patients with and without lesions on CT is similar.

In my experience the occurrence of structural intracranial lesions is higher than 1%. A personal study showed that cerebral transient attacks due to lacunes, large or medium size infarctions, tumours and haemorrhages reached 30%[2] of suspected TIA cases, and 13·4% of these patients suffered from lacunar infarction.[3] In another report the percentage of lacunar infarction was even higher, reaching 23% of all cerebrovascular diseases.[4] Patients suffering from lacunar infarction do have different prognosis from those with large infarctions and from TIAs without CT lesions.[5] The survival rate of patients with lacunar infarction is 479/1000, slightly higher than that of patients with completed stroke due to large or medium-size infarction, but considerably lower than that of patients with TIA with negative CT scan.[6 7] Therefore, cranial CT scan may modify the prognosis at least in one third of the patients with suspected TIA. Moreover, in some studies[6 7] mortality and morbidity are higher in TIA patients with respect to minor stroke.

In addition, some authors believe that therapy of patients suffering from lacunes is different from therapy of TIA with negative CT findings: TIAs and minor stroke need prophylactic therapy with anti-aggregating agents and careful evaluation for possible surgical therapy.

In conclusion I believe that a CT scan is a necessary test in all patients with suspected TIA and should be regarded as a very cost-effective strategy.

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Hankey and Warlow reply:

Professor Loeb has misinterpreted our distinction between a structural intracranial lesion (for example, a tumour or arteriovenous malformation) and a low density lesion (such as, a presumed infarct) on cranial CT scan. Our editorial indicates that, from the available data, about 1% of patients presenting with suspected TIA, who undergo a CT, have CT evidence of a structural intracranial lesion such as a tumour or AVM. In addition, about 10–30% of patients presenting with TIA (depending on which series you read) have cranial CT scan evidence of a low density lesion, such as a small deep infarct. Some of these hypodensities are clearly not related to the presenting TIA and are therefore asymptomatic (as they are located on the asymptomatic side of the brain) and some may be symptomatic (as they are located on the symptomatic side of the brain).

The key point is that, for TIA patients, the presence of focal hypodensity on CT has not been shown conclusively to be an important independent prognostic factor for subsequent important vascular events such as stroke, myocardial infarction or vascular death. It is therefore not that important to do a CT scan, just to see if a hypodensity is present or not (because it is of no prognostic value). It is, however, clearly important to identify underlying structural lesions such as a tumour, because the prognosis and treatment may be different. As such findings are infrequent (occurring in about 1% of suspected TIA patients), our editorial concludes by recommending further prospective studies of the yield and cost of CT scan in patients with TIA. Of course, if future studies show that the finding of infarction on CT is an important prognostic factor for stroke and other serious vascular events, then there would be greater purpose in performing cranial CT in patients with suspected TIA, and it may prove to be cost effective, as Professor Loeb suggests. At present, however, we do not have that information.

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BOOK REVIEWS

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