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

One of the mechanisms of the false facilitation may be a physiological one due to an accommodation phenomenon, which 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, if excessive movements are not prevented.

Unfortunately, the facilitation shown by Dr 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.


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 presence of TIA patients with and without lesions on CT is similar.

In my opinion the occurrence of structural intracranial lesions is higher than 1%. A personal study showed that cerebral transient ischaemic attacks due to lacunes, large or medium size infarctions, tumours and haemorrhages reached 30% of suspected TIA cases, and 13–4% of these patients suffered from lacunar infarction.1 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 TIs with 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 Therefore, cranial CT scan may modify the prognosis at least in one third of the patients with suspected TIA. Moreover, in some studies2,7 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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