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 accidental release of ACh, when an increase in 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 waning 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 experience the occurrence of structural intracranial lesions is higher than 1%. A personal study showed that cerebrovascular events, particularly lacunar infarction 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. In another report the percentage of lacunar infarction was even higher, reaching 23% of all cerebrovascular diseases. Patients suffering from lacunar infarction do have different prognosis from those with large infarctions and from TIA without CT lesions. 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. Therefore, cranial CT scan may modify the prognosis at least in one third of the patients with suspected TIA. Moreover, in some studies mortality and morbidity are higher in TIA patients with respect to minor stroke.

In addition, some authors believe that therapy of patients with lacunar infarction from lacunes is different from therapy of TIA with negative CT findings: TIA 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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