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). 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. 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 significantly shorter when the amplitude increased. As a result, the area of negative phase remained relatively unchanged, which is a typical finding for false 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.
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 present study suggests that there is no advantage in the use of CMAPs. When moderate and high frequency stimulations are used, the amplitude remains 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.


Hankay 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 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 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 occur 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.