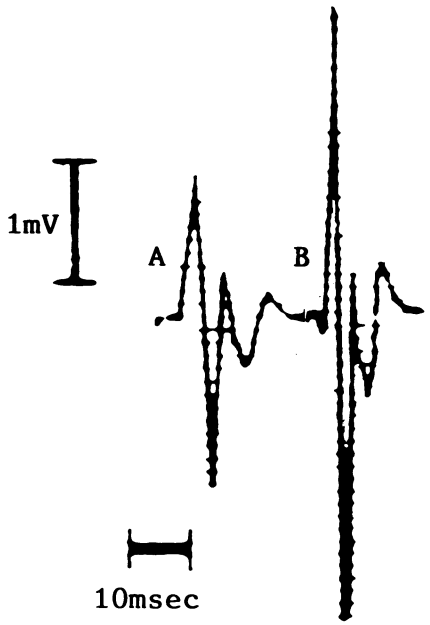

Figure 2 The initial CMAP (indicated by A) and the fifth one (B) to 50 Hertz stimulation in Guillain-Barré syndrome.[1] Instead of marked increase in amplitude, the duration of the fifth CMAP became nearly a half of that of the initial CMAP.

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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1 Pullicino P, Beck N. Incremental response to repetitive stimulation in Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 1992; 55:233–4.
2 Keesey JC. Electrophysiological approach to defects of neuromuscular transmission. *Muscle Nerve* 1989;12:613–26.
3 Desmedt JE. The neuromuscular disorder in myasthenia gravis. 1. Electrical and mechanical response to nerve stimulation in hand muscles. In: Desmedt JE, ed. *New developments in electromyography and clinical neurophysiology*, Vol 1. Basel:Karger, 1973: 241–304.
4 Kimura J. Techniques of repetitive stimulation. In: *Electrodiagnosis in diseases of nerve and muscle*, 2nd ed. Philadelphia: FA Davis, 1989:184–207.

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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1 Hankey G, Walton C. Cost-effective investigations of patients with suspected-transient ischaemic attacks. *J Neurol Neurosurg Psychiatry* 1992;55:171–6.
2 Loeb C. The diagnosis of TIA and RIND: basic requirements in Cerebrovascular Disease. In: Lechner H, Meyer JS, Ott E, eds. *Cerebrovascular disease: research and clinical management.* Elsevier. Amsterdam, 1986:231–6.
3 Loeb C, Gandolfo C, Mancardi GL, Primavera A, Tassinari T. The lacunar syndromes. A review with personal contribution. In: Lechner H, Meyer JS, Ott. E, eds. *Cerebrovascular Disease: research and clinical management.* Vol 1 Amsterdam: Elsevier, 1986.
4 Mohr JP, Caplan LR, Melski JW, *et al.* The Harward Cooperative Stroke Registry: a prospective registry. *Neurology* 1978;28: 745–62.
5 Gandolfo C, Caponnetto C, Del Sette M, Santoloci D, Loeb C. Risk factors in lacunar syndromes: A case control study. *Acta Neurol Scand* 1988;77:22–26.
6 Loeb C. Transient ischemic attack, protracted transient ischemic attack and completed stroke. *Eur Neurol* 1983;22:68–73.
7 Falke P, Stavenov L, Young M, Lindgard F. Differences in mortality and cardiovascular morbidity during 3 years follow up of transient ischemic attacks and minor stroke. *Stroke* 1989;20:340–4.

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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