Lambert-Eaton myasthenic syndrome (n = 14, three had coexisting cerebellar degeneration and six had SCC). It is noteworthy that non-organ-specific ANA were found in 33% of patients with Lambert-Eaton syndrome who had cancer (n = 50). Patients with this syndrome have a variety of autoantibodies.[10] This emphasises the necessity of retesting and titrating any serum that is positive for neuronal nuclear reactivity on substrates of neural and non-neural tissues side-by-side to confirm neuronal specificity.

Immunoblot criteria have been used by some researchers to classify different types of anti-neuronal antibodies.[9 11] However, our experience with the immunofluorescence screening procedure described above validates the clinical usefulness of a positive result obtained with this simple test. Seronegativity does not exclude the presence of a malignant tumour. Occasionally atypical patterns of cerebellar staining are encountered, that is, not strictly fitting either of the two patterns described. At present no attempt is made to assign them clinical significance.

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Crying and laughing after brain damage: a confused nomenclature

Sir: Brain damage is commonly associated with abnormalities of emotional expression. This has long been recognised[1] and such changes have excited the interest of both neurologists[2] and psychiatrists.[2] Episodes of crying and unconstrained outbursts of laughter are most often described in connection with stroke,[3] multiple sclerosis,[4] cerebral tumour,[2] and motor neuron disease[5] but they are not specific to any particular neurological disorder and can follow most causes of brain damage.[6] Possible pathophysiological mechanisms have been discussed,[6] but their exact nature remains obscure. Lesions are usually multiple and bilateral and involve the corticobulbar tracts.[2] These changes are often distressing to patients and their carers and they can contribute to difficulties in management and diagnosis. There has been no systematic study of the phenomenology of these expressions and we know little about their physical or psychological correlates. What is striking about the existing literature is the multiplicity and confusion of the terminology. Communication would be facilitated by a consensus as to the most appropriate nomenclature. In this letter I will discuss the relative merits of existing terms and propose one of them as a candidate for future use.

Authors have often used different terms interchangeably, for example, "involuntary crying", "pathological emotionality", and "forced crying" may be construed as synonymous in describing the same phenomenon. In contrast, some authors use separate terms to refer to what they consider to be distinct phenomena, for example Poeck[6] makes a distinction between "pathological crying and laughing" on the one hand and "emotional lability" on the other. Some terms include reference to aetiology, as in "pseudobulbar affect" and "organic emotionalism", or to severity, as in "emotional incontinence." Others are more descriptive or judgmental as in "spasmodic crying," "inappropriate crying" and "pathological affect".

Some of these terms are clearly unsuitable or inadequate and should be abandoned. "Pseudobulbar affect", for example, can have only a limited application since the majority of patients with brain damage and episodes of crying or laughing will never have so called pseudobulbar signs. "Involuntary crying" alludes to absent or diminished control over the expressions but crying in non-brain damaged people is in a sense "involuntary" so the term is redundant. "Inappropriate crying or laughing" implies (wrongly) that it is possible to judge the appropriateness of an emotional expression under given circumstances. Also, this term detracts from making an effort to understand why the expression might be appropriate in any particular situation. "Emotional incontinence," although it invokes a vivid image of patients with extreme and uncontrollable episodes of crying or laughter (or both), has too many excretory connotations for regular use!

We are left with the terms: "pathological crying or laughing", "emotional lability" and "emotionalism". The first is the least satisfactory because of the problem of deciding what is "pathological"—is the emotional expression itself "pathological" because it (sometimes) appears different in some way to "normal" or does "pathological" refer to the underlying brain damage? "Emotional lability" is attractive in that it conveys meaning without being judgmental or confusing. The shortened form "lability" is also convenient for day to day use. However, the term is used widely in psychiatry to describe emotional states that are quite distinct from those seen in brain damage. The term "emotionalism" has been less widely used but unlike "emotional lability" it is not also employed to describe unrelated phenomena. It has the added advantages of being a single word, free of inaccurate aetiological inference and also non-judgmental.

In conclusion, the existing nomenclature for the abnormalities of emotional expression following brain damage is confused and inadequate. Consistency is required in order

to facilitate communication about such phenomena. No single term is entirely satisfactory but unless subtypes are adequately delineated, requiring further elaboration of the terminology, emotionalism seems to be the best choice.

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Sublingual apomorphine and Parkinson's disease

Sir: Subcutaneous injection of the dopamine receptor agonist apomorphine combined with oral domperidone is a safe, effective therapy for otherwise refractory disabling "off period" disabilities in patients with Parkinson's disease receiving long term l-dopa treatment.[1-4] After a therapeutic suprathreshold injection "unblocking" occurs after 5–10 minutes and lasts for 30–90 minutes. Although apomorphine can produce anti-parkinsonian effects when administered orally, very large doses (500 mg per day) are needed, the speed of action is much slower and unacceptable elevations in blood urea and plasma creatinine can occur.[5] We have therefore investigated the alternative possibility of administering the drug by the sublingual route.

Nine patients with idiopathic Parkinson's disease and severe refractory motor fluctuations agreed to participate after giving their informed consent. Their median age was 64 years, mean duration of disease 12 years (4–20 years), mean duration of l-dopa therapy 11 years (4–17 years), mean dose of l-dopa 600 mg, mean stage of Hoehn and Yahr when "off" 3·9. All the patients were known to respond to subcutaneous injections of apomorphine, mean dose 3 mg (1–5 mg). After pilot studies it was decided to give all the patients a standard 30 mg sublingual dose (10, 3 mg tablets), when fasting, after 48 hours of oral domperidone pre-treatment (20 mg tds) to ensure a maximum therapeutic response.

Anti-parkinsonian drugs were stopped for 12 hours before each study. Baseline assessments, tapping test and timed walking tests were carried out and were repeated every 10 minutes after the administration of sublingual apomorphine. A modified Webster scale (to include rising from a chair and balance) was carried out at baseline and then again at the time of maximum therapeutic response.[6]

All patients switched "on" with sublingual apomorphine with a therapeutic effect approximately comparable to that seen after subcutaneous apomorphine. Five of the patients were asked to let the tablets dissolve under the tongue whereas in the other four, the tablets were crushed up first. The mean time for dissolution was 33 minutes (20–45 minutes) and the mean time from complete dissolution to full switch on was a further 10 minutes. The therapeutic response lasted for a mean 73 minutes (30–110 minutes), the mean increase in tapping score was 16 per 30 seconds (from 26–42), the mean reduction in walking time was 15 seconds (from 30–15 seconds), tremor was abolished in all seven cases where it was present and drug induced dyskinesias were seen in six patients. Adverse reactions were mild sedation (six patients), slight nausea (two patients), and yawning (two patients). One patient whose tablets dissolved extremely rapidly almost fainted, with a significant drop in blood pressure.

These results confirm that sublingual apomorphine is an effective anti-parkinsonian preparation producing therapeutic responses qualitatively similar to those seen with oral levadopa. Although the latent period between therapeutic effects was longer than that seen after subcutaneous apomorphine injection, this was largely due to the time taken for the tablets to dissolve. We believe that this delay can be overcome by more efficient formulation. Preliminary work with liquid apomorphine administered sublingually suggests that only two or three times the dose of sublingual apomorphine would be necessary to produce comparable effects to those seen following parenteral administration. Sublingual apomorphine is likely to provide a viable alternative to intermittent injections of apomorphine in the treatment of "off period" disabilities in Parkinson's disease.

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