'mongoloid', which fell into disfavour in the context of the previous designation of Down's syndrome as 'mongolian idiocy'). I will therefore refer in this letter to black people.

We have no difficulty with the designation of black people who are nationals of West African countries. It is also easy to see that black people of West African extraction who are nationals of the Caribbean islands are Afro-Caribbeans, African-Caribbeans or, as some prefer to call them, West Indians (though this latter designation is less precise insofar as it necessarily includes Caribbean people of other racial backgrounds, including the indigenous peoples of those islands, such as the Caribs). How then do we designate black people, of West African or African-Caribbean parentage, who are born in the United Kingdom and carry UK passports?

It is clearly incorrect to refer to them as West Africans on the one hand, or as West Indians or African-Caribbeans on the other, any cultural identification with people of these backgrounds notwithstanding. I think that the findings of our research would be on firmer ground if we recognised that there are three distinct subgroups of West Africans and people of West African extraction: the West Africans themselves, the African-Caribbeans (or West Indians) and the Afro-Britons or African-Britons, this last group being black people who are UK nationals.

The tendency to lump all African-Caribbeans and African-Britons into one large group as 'West Indians' muddies the waters in our research, and we should aim for greater clarity in the use of these terms.

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Episodic dyscontrol

SIR: I read with interest the article by Lewin & Sumners (*Journal*, August 1992, **161,** 261–262) in which they reported a patient who developed episodic dyscontrol after a road-traffic accident and responded well to carbamazepine. Although they considered intermittent explosive disorder (DSM–III–R) as a differential diagnosis, I wonder why they did not mention organic mood disorder (DSM–III–R). Since there was evidence of bilateral frontal and temporal lobe lesions in their patient and he suffered from depressive states, it seems likely that his episodic dyscontrol might be due to underlying organic mood disorder. Assuming that this was the case, it is less surprising that he responded well to carbamazepine.

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Applying Roberts' framework

SIR: Using the framework described by Roberts (*Journal*, September 1992, **161,** 293–308) I should like to present a case to illustrate the origins of delusion.

Case report. A 38-year-old woman with no previous psychiatric history sustained a severe head injury, and nine months later developed a paranoid state. Computerised tomography scan initially revealed diffuse cerebral contusions, but later there was no focal abnormality. Her intelligence quotient decreased (WAIS–R full scale 87–97, NART 114) and frontal lobe dysfunction was indicated by disinhibition and impulsivity and findings on the Wisconsin Card Sorting Test of idiosyncratic reasoning and gross impairment of abstract thinking and shift of mental set.

The injury acted as a non-specific precipitant (stage 2). Eight months later the prodrome (stage 3) was characterised by complaints of persistent confusion and impaired memory which, together with a psychological response to the trauma, led to depression and weepiness and to her belief that her brain was abnormal and about to discharge a lot of electricity. This could be seen as a rational attempt to explain abnormal experiences, but was not successfully adaptive as she entered a highly anxious state with concerns that she had a deep vein thrombosis and was about to die of a pulmonary embolus. She repeatedly called out her GP and dialled 999, but failed to be reassured and came to believe that she was being badly treated by the medical profession. This mental set could have engendered further persecutory beliefs by influencing her interpretation of events (Garety, 1991; Fleminger, 1992) and by priming preattentive processes to bring potentially threatening stimuli to attention (Anscombe, 1987).

In stage 4 she reported that her telephone was making strange noises and that she had been cut off in the midst of calls. On being told by a British Telecom engineer that the fault was under investigation, she interpreted this as meaning that she was under investigation and became established in the belief that she was being bugged. This misinterpretation arose because of the heightened significance given to the word 'investigation', possibly because intentional priming of preattentive processes was bypassed (Anscombe, 1987) or may not have been recognised in the way that willed intentions may not be in patients with positive symptoms (Frith & Done, 1988). This alone may have been sufficient for her to apply an improbable explanation, but that she did so implies a failure of hypothesis evaluation. Jumping to a conclusion (Garety, 1991) could reflect

a lack of tolerance of ambiguity and the need to resolve the anxiety generated by competing hypotheses as well as a failure to apply knowledge about the likely causes of events. That the latter played a role in this case is suggested by the fact that when it returned her delusions began to disappear. Thus she dismissed as nonsense the previously held belief that doctors had put bugs under her forearm plaster, because it was highly improbable that they would do so.

In stage 5, defects in reasoning underpinned elaboration of her beliefs which, in accord with Bleuler's view (cf. Winters & Neale, 1983), was consequent upon errors in logical thinking in that she believed she was being investigated for drugs dealing because her boyfriend had been to the Far East. Happily, with neuroleptic treatment her delusions resolved and she went home.

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Botulinium toxin in a case of severe tardive dyskinesia mixed with dystonia

SIR: Botulinium toxin appears to be an effective and safe treatment for cervical dystonia (Jankovic & Schwartz, 1990; Blackie & Lees, 1990; Jankovic & Brin, 1991; Poewe *et al*, 1992). It is used in patients who failed to improve with standard medications. To our knowledge, it has not been used for patients with tardive dyskinesia (Burke & Un Jung Kan, 1988). We report a case of severe mixed syndrome of tardive dyskinesia and dystonia.

Case report. The patient is a 41-year-old, divorced, right-handed man. In 1969, with no significant previous history (personal or family) of neurological or psychiatric illness, he presented with an acute schizophrenic episode, characterised by auditory and somatic hallucinations, agitation, thought withdrawal, persecutory delusions, periodic mutism, grimacing, and schizophasic episode. He received neuroleptic therapy for the next 23 years, with intermittent pauses of less than 2 years. The following antipsychotic medication was well-tolerated and improved the main schizophrenic symptoms: he received in sequence regular dosages of chlorpromazine, trifluoroperazine, haloperidol, thioridazine, methotrimethazine, haloperidol, and fluphenazine, and was given central anticholinergics (trihexiphenidyle or procyclidine) for correcting drug-induced extrapyramidal reactions. He received also diazepam and clonazepam.

The patient first noticed abnormal movements in 1973, occurring in intermittent fashion, first localised to the right shoulder but progressing to both shoulders. The abnormal movements were most prominent in the evening, and associated with stress and fatigue. Blepharospasms followed eventually, as well as uncontrollable mixed dyskinetic and dystonic movement of the face and jaw. By 1984, head and neck dyskinesia had appeared, including significant neck extension dystonic movements. Haloperidol tapering in 1986 accentuated the movements, which became continuous and extended to the trunk and upper limbs, thus confirming the diagnosis of tardive dyskinesia mixed with tardive dystonia. These movements tended to decrease or disappear with directed activity requiring significant concentration, and disappear during sleeping. Gait instability ensued, with a few resulting falls.

In 1987 treatment with reserpine (up to 6 mg/day) was tried and discontinued. Then the patient received lecithine, followed by loxapine, and finally the neuroleptic medication was discontinued, except a trial with risperidone with positive but no lasting effects, possibly due to an interaction with valpoic acid which had to be given because of withdrawal seizure related to benzodiazepine discontinuation. A treatment with tetrabenazine during the two last years mildly improved the syndrome.

The patient has no history of exposure to chemical or industrial toxins. Wilson's disease was ruled out on the basis of an ophthalmological examination, as well as ceruloplasmin and copper levels. Isotopic, computerised tomography and nuclear magnetic resonance scans were all normal. Neuropsychological examinations, Wisconsin Card Sorting Index, Hooper, and mirror reading reflected frontal and basal ganglia dysfunction. Neurological examination, in 1992, revealed severe abnormal dyskinetic movements of the face and the trunk with blepharospasms, hyperextension of the neck, and episodic opisthotonos and a choreoathetosic right-arm movement. The psychiatric examination revealed, at this time, no psychotic symptoms and only a dysphoric mood. Extrapyramidal scales (AIMS, Ross-Chouinard) illustrated a severe movement disorder.

Jankovic & Schwartz (1990) have demonstrated the efficiency of botulinium toxin injection in the treatment of primary cervical dystonia on a population of 205 patients. Long-term treatment with these toxins has been studied for up to five years, with relatively minor adverse effects and development of resistance. Botulinium toxin A is a potent pre-synaptic neuromuscular blocking agent, widely used in many spastic disorders. Three botulinium toxin injections were performed, in the hope of preventing a cervical fracture, in our patient with severe dystonic posterior hyperextensions. The objective was to weaken extensor contraction and thus to avoid dangerous and paroxysmal extension. 75 units of toxin A were injected into the following muscles: left splenius capitis, right and left trapezius. The therapeutic results were spectacular. Adverse effects included a flexed position of the neck, and intermittent dysphagia during the first ten days after injection. Injections