poorly controlled diabetes mellitus, anticoagulant therapy, infection, and hypermobility syndromes are absolute contraindications to spinal manipulation.⁷ Despite these various precautions, many potentially vulnerable patients will be unidentified and it is likely that patients will continue to present with neurological complications after chiropractic manipulation.

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- 1 Mohr PD, Strang FA, Sambrook MA, *et al*. The clinical and surgical features in 40 patients with primary cerebellar ectopia (adult Chiari malformation). *Q J Med* 1977;181:85-96.
- 2 Paul KS, Lye RH, Strang FA, *et al*. Arnold-Chiari malformation, review of 71 cases. *J Neurosurg* 1983;58:183-7.
- 3 Smith RA, Estridge MN. Neurologic complications of head and neck manipulation, report of two cases. *JAMA* 1962;182:528-31.
- 4 Mueller S, Sahs AL. Brainstem dysfunction related to cervical manipulation. *Neurology* 1976;26:547-50.
- 5 Rinsk LA, Reynolds GG, Jameson MM. A cervical cord injury following chiropractic manipulation. *Paraplegia* 1976;13:223-7.
- 6 Heffner JE. Diaphragmatic paralysis following chiropractic manipulation of the cervical spine. *Arch Intern Med* 1985;145:562-4.
- 7 Lee KP, Carlini WG, McCormick GF, *et al*. Neurologic complications following chiropractic manipulation: a survey of California neurologists. *Neurology* 1995;45:1213-15.

random sample of the adult population of the United Kingdom, The Netherlands, and Sweden.² As a result, the summary index score does not quantify the respondent's value of their own health, but rather the value that the general population would place on the respondent's health. By contrast, the visual analogue scores are direct measures of the value a respondent places on his or her own health. It is not surprising that the summary index scores and visual analogue scale scores are somewhat different in the study by Schrag *et al*.

These properties of the EQ-5D make it an indicated measure of health status for certain applications, particularly estimating health utility for cost-utility analysis. It is, perhaps, a fortunate accident that it is also a valid measure of quality of life in patients with Parkinson's disease.

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- 1 Schrag A, Quinn N, Jahanshahi M, *et al*. The EQ-5D—a generic quality of life measure—is a useful instrument to measure quality of life in patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry* 2000;69:67-73.
- 2 EuroQol Group. EuroQol—a new facility for the measurement of health-related quality-of-life. *Health Policy* 1990;16:199-208.

Selai *et al* reply:

We thank Siderowf and Werner for their interest in the EQ-5D and our work. They raise important points about the use and interpretation of generic quality of life instruments.

The valuation of health states raises many complex methodological and ethical issues and it is the topic of considerable debate in the literature.¹ Although we have participated in this debate,² we did not enter into a discussion of these issues in our recent paper because this was beyond the scope of that study.

The EQ-5D is a generic measure that has three distinct components, each providing separate data. The first part yields a simple descriptive profile of the respondents' own subjective health status in five dimensions. Secondly, the respondents next rate their own health on a visual analogue scale, calibrated 0-100. Thirdly, according to how the respondents have rated themselves on the descriptive profile, a utility value can be ascertained. Thus, the EQ-5D generates a cardinal index of health, giving it considerable potential for use in both economic evaluation and for ascertaining a person's subjective perspective of their own health status. The EQ-5D classification system defines a fixed number of health states, which may include health states valued worse than death, but leaves open the issue of what value should be assigned to each of those states.³ Valuation data sets have been obtained in several countries, both European and non-European.

The evaluation of health related quality of life (HR-QOL) of patients with a given disease is generally measured using a disease specific instrument,—for example, the PDQ-39 in patients with Parkinson's disease. As these instruments are only applicable to patients with a particular disease, they do not allow comparisons across health conditions

and are of limited use in economic studies. It is therefore recommended that a generic HR-QOL instrument be used in addition to disease specific measures. The generic measure must be tested and validated before use in the respective patient population. The purpose of our study was to test the validity and feasibility of two generic measures of HR-QOL (the EQ-5D and the SF-36) in patients with Parkinson's disease. The EQ-5D, a simple generic instrument, was shown to have good validity and feasibility and performed better than the SF-36 in this group of patients with Parkinson's disease.

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- 1 Drummond MF, O'Brien B, Stoddart GL, *et al*. *Methods for the economic evaluation of health care programmes*. 2nd ed. Oxford: Oxford Medical Publications, 1997.
- 2 Selai CE, Rosser RM. Good quality quality? Some methodological issues. *J R Soc Med* 1993;86:440-3.
- 3 Kind P. The EuroQol instrument: an index of health related quality of life. In: Spilker B, ed. *Quality of life and pharmacoeconomics in clinical trials*. Philadelphia: Lippincott-Raven, 1996.

Cerebral malaria

Two items in the review of cerebral malaria by Newton *et al*¹ warrant comment. The first relates to corticosteroids in the treatment of cerebral malaria, and the second involves the definition of cerebral malaria.

The authors, citing Warrell *et al*² and Hoffman *et al*,³ stated that steroids are contraindicated in cerebral malaria because they add risks without providing any benefit.

In late December 1965, I arrived in Vietnam as a neurologist with the United States Army Medical Corps., and for the next 6 months I was the only neurologist serving United States forces in Vietnam. I was attached to the 93rd Evaluation Hospital, which opened only 2 weeks before my arrival. As recounted in a recent article about my Vietnam experience,⁴ two American soldiers with cerebral malaria died at the hospital during those 2 weeks, and the internal medicine specialists requested my involvement in all future cases. After reviewing the treatment protocols of the two soldiers, I decided that adding dexamethasone to the usual antimalarial regimen would be reasonable in patients with severe cerebral malaria. There was, I think, no literature at the time on the use of steroids; if there was, I certainly had no access to it in the combat zone.

During the next 10 months, we saw 19 patients with cerebral malaria, and they all recovered without any residual neurological dysfunction.⁵ Our success with steroids prompted all the United States medical units in Vietnam to adopt the practice. A decade after the end of the Vietnam conflict, Warrell *et al*,² in a double blind trial, concluded that dexamethasone was both ineffectual and deleterious in cerebral malaria, and Hoffman *et al*³ concurred. These two papers^{2,3} are those usually cited by tropical medicine specialists (Newton and Warrell, 1998; White, 1999. See Daroff⁶ for citations) in recommending

CORRESPONDENCE

The EQ-5D—a generic quality of life measure—is a useful instrument to measure quality of life in patients with Parkinson's disease

We read with interest the recent article by Schrag *et al*,¹ in which the authors showed that the EuroQol-5D (EQ-5D) is a valid measure of quality of life in patients with Parkinson's disease. However, the authors neglected to mention two important aspects of the EQ-5D that differentiate it from typical quality of life instruments.

Firstly, the EQ-5D is a preference based measure. The summary score of the EQ-5D captures the strength of a person's preference for a given health outcome relative to other possible outcomes. These preferences (also called utilities) are measured on a scale from 0 to 1 where 0 represents death and 1 represents perfect health. The values derived from the EQ-5D can be used to compare health states in a quantitative way. For example, a health state with a value of 0.5 is half as desirable as perfect health. The scoring rule for the EQ-5D permits scores less than 0, implying that some health states may be worse than death.

Secondly, the index scores for the EQ-5D are intended to approximate general population preferences rather than the respondent's own health values. The EQ-5D values were developed based on ratings by a large,

against the use of steroids in adult patients with cerebral malaria. Given the importance of the papers^{2,3} buttressing the "no steroid" mandate, I will summarise them.

Warrell *et al*² studied Thai patients in a village hospital. There were two groups of 50 patients each; eight in the steroid treated group died, and nine of the untreated controls died. Hoffman *et al*³ studied Indonesians in a provincial hospital with a mean age of 10.2 years, and found that four of 19 patients in both the treatment and control groups died. The two studies^{2,3} had mortality rates of 17%–21%, and neurological residua in the survivors, which is in sharp contrast to the experience with United States troops in Vietnam. As mentioned, we had no deaths or residual neurological dysfunction in our 19 patients⁵ and Dr Andrew Carr, the second neurologist in Vietnam, had an almost identical experience in 1966–7 using steroids in the sicker patients (personal communication). Blount (see Daroff⁴ for citation) treated 24 patients with cerebral malaria with steroids and had no deaths. The three United States army hospital experiences in Vietnam in 1966–7 included a total of 62 patients with cerebral malaria liberally treated with steroids, with no deaths. There were no neurological residua in our series or Carr's⁴; Blount did not comment on residua. This compares with the 17% death rate in the study by Warrell *et al*², 21% in that of Hoffman *et al*³ and residual dysfunction in their survivors.

The cerebral malaria experience in the second world war is particularly relevant. Mortality rates in American troops were lower than in their Asian counterparts, and the rates in adults were lower than in children.⁵

Thus, I don't think that the conclusions of Warrell *et al*², Hoffman *et al*³ and Newton *et al*¹ are applicable to western combat troops who are usually in excellent health before contracting malaria, and I continue to recommend steroids for an adult patient with cerebral malaria manifesting significant neurological dysfunction. I specify adults, as we lacked experience with children.

The definition of cerebral malaria is the other issue I have with Newton *et al*.¹ We diagnosed cerebral malaria when a patient with confirmed *Plasmodium falciparum* parasitaemia displayed signs of cerebral dysfunction that could not be explained by hyperpyrexia or detectable metabolic abnormalities.⁵ The United States Army in the second world war also used this simple diagnostic criteria and experienced a similar occurrence rate. Our 19 patients with cerebral malaria were among about 1200 cases of falciparum malaria admitted during a 10 month period. This occurrence rate of about 1.6% is concordant with reports from United States hospitals in the second world war, which ranged from 1.2% to 2.3%.⁵

The neurological manifestations in our patients fell into five broad groupings.⁵ Disturbance of consciousness was the most common (eight patients), and ranged from extreme lethargy to coma; on awakening, the patients displayed the expected transient cognitive disturbances before gradually returning to normalcy. Four patients had delirium, three movement disorders (tremor, myoclonus, chorea), one had a unilateral cerebral hemispheric syndrome, and three had acute personality changes (manifesting in two as a paranoid psychosis and in the other as a delusional state). In each of the these three,

psychometric testing showed organic dysfunction that ultimately normalised (Blocker *et al* 1968; Kastl *et al* 1968. See Daroff⁴ for citations). Previous studies, dating back to Anderson's 1927 monograph (see Daroff *et al*⁴ for citation), reported similarly diverse manifestations in cerebral malaria.

Newton *et al*¹ commented about the loose definitions of cerebral malaria in the literature, often without evidence that confounding secondary causes of encephalopathy were excluded. We are confident that this criticism is not applicable in our series, nor others emanating from the medical experience among United States forces in Vietnam. Nevertheless, Newton *et al*¹ suggested a strict definition of cerebral malaria, requiring "a deep level of unconsciousness" or coma. Such restricted criteria might be justified in studies comparing treatment protocols, but to insist that a cerebral condition lacks degrees of severity is contrary to the experience of any neurologist. The all or none definition of cerebral malaria is simply not acceptable.

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- 1 Newton CRJC, Hien TT, White N. Cerebral malaria. *J Neurol Neurosurg Psychiatry* 2000; **69**:433–41.
- 2 Warrell DA, Looareesuwan S, Warrell MJ, *et al*. Dexamethasone proves deleterious in cerebral malaria. A double-blind trial in 100 comatose patients. *N Engl J Med* 1982; **306**:313–19.
- 3 Hoffman SL, Rustama D, Punjabi NH, *et al*. High-dose dexamethasone in quinine-treated patients with cerebral malaria: a double-blind, placebo-controlled trial. *J Infect Dis* 1988; **158**: 325–31.
- 4 Daroff RB. Neurology in a combat zone. Vietnam 1966. *J Neurol Sci* 1999; **170**:131–7.
- 5 Daroff RB, Deller Jr JJ, Kastl Jr AJ, *et al*. Cerebral malaria. *JAMA* 1967; **202**:679–82.

White and Newton reply:

Daroff has queried the World Health Organisation (WHO) definition of cerebral malaria that we quoted in our paper.¹ We pointed out that a patient with falciparum malaria with any impairment of consciousness or other sign of cerebral dysfunction should be treated as a medical emergency with parenteral antimalarial drugs. The point about the definition proposed by WHO,² is to allow a direct comparison between studies in which the clinical syndromes are precisely defined.

This point is well illustrated by Dr Daroff's comments on studies of corticosteroids in malaria; he compares his studies on cerebral malaria, in which the cerebral involvement was described in terms of "extreme lethargy", "delirium", or "stupor"³ to those in whom cerebral malaria was strictly defined.^{4,5} It is difficult to compare the mortality in such disparate groups.

As for the continued use of corticosteroids in cerebral malaria, Daroff's recommendation is based on anecdotal experience during the second world war and Vietnam in the 1960s. There is little theoretical basis for using corticosteroids.⁶ The two double blind, randomised control trials of dexamethasone failed to demonstrate any benefit from corticosteroids in adults with a precise definition of cerebral malaria.^{4,5} Indeed the studies were associated with a significant increase in gastrointestinal bleeding,^{4,5} infections,⁴ and duration of unconsciousness.⁴

It is difficult to assess historical anecdotal evidence as there are many factors which may contribute, such as differences in the definitions of the clinical syndrome, drug resistance,

and patient groups. Although the two randomised trials do not exclude a potential benefit, there is little substantial evidence to support the use. The onus is on the people who think that corticosteroids are beneficial, to provide more substantial data to support their conjecture, preferably as the results of a randomised trial.

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- 2 Warrell DA, Molyneux ME, Beales PF. Severe and complicated malaria. *Trans R Soc Trop Med Hyg* 1990; **84**(suppl 2):1–65.
- 3 Daroff RB, Deller JJ Jr, Kastl AJ Jr, *et al*. Cerebral malaria. *JAMA* 1967; **202**:679–82.
- 4 Warrell DA, Looareesuwan S, Warrell MJ, *et al*. Dexamethasone proves deleterious in cerebral malaria. A double-blind trial in 100 comatose patients. *N Engl J Med* 1982; **306**:313–19.
- 5 Hoffman SL, Rustama D, Punjabi NH, *et al*. High dose dexamethasone in quinine treated patients with cerebral malaria: a double blind, placebo controlled trial. *J Infect Dis* 1988; **158**: 325–31.
- 6 World Health Organisation. Severe falciparum malaria. *Trans R Soc Trop Med Hyg* 2000; **94**(suppl 1):S1–1/74.

Treatment of paroxysmal sympathetic storm with labetalol

Do *et al* present a patient with paroxysmal sympathetic storm, and include a sample of the patient's ictal EEG recording.¹ It is stated that the absence of clear epileptiform activity serves as evidence against these episodes being epileptic in nature. However, if indeed the EEG recordings during an attack show significant slowing, as depicted in the EEG sample, this EEG change from a presumably normal EEG background rhythm at other times, would rather indicate that these events are, indeed, of seizure origin.

Deep seated epileptic foci very often do not project any sharply configured waveforms to the scalp surface; neither do they provide reliable localising information and can appear rather in a generalised fashion. In such cases, any reproducible and reliably observable EEG changes from the background rhythm that coincide with the clinical event are typically interpreted as evidence in favour of epileptic activity. Possible candidates for surgical treatment of their epilepsy would be further investigated with implanted electrodes in an effort to obtain EEG recordings from the closer vicinity of the presumed focus. This is obviously not a justifiable approach in this patient as surgical resection is not an option in this location.

Neither should successful treatment of specific symptoms during an attack with medication other than antiepileptic drugs necessarily be interpreted as evidence against the event being epileptic. In this case report, the patient's autonomic disturbances during the episode did respond to labetalol; however, this does not necessarily exclude a possibly epileptic origin as labetalol could only have obscured the clinically observable manifestations.

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