haematoma as incidental. There was no computed tomogram before treatment, the aetiology of the haematoma was not discussed, and results of examination of the cerebrospinal fluid were not given. In addition, the second reference is in supplement 1 of *Cancer Treatment Reports* 1981 and not in the main journal.

Rather than postulate a second, previously unreported cause of this patient's neurological condition it would be simpler to follow Occam's razor and explain that which is obvious. The intracerebral haematoma may have resulted from hypertension, aneurysm, or haemorrhage into a deposit, in which case the administration of methotrexate would then probably be incidental.

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1 Hughes PJ, Lane RJM. Acute cerebral oedema induced by methotrexate. *Br Med J* 1989;**298**:1315. (13 May.)

AUTHORS' REPLY,—We are sorry that Dr Ellis finds our interpretation of this case unsatisfactory. Perhaps the important changes in the initial computed tomogram were unclear. Though there was indeed a frontal haematoma with some surrounding focal oedema, the important point is that there was generalised cerebral swelling, the lateral ventricles and prepontine cisterns being almost completely obliterated. The second tomogram taken after treatment with osmotic diuretics and steroids showed the focal oedema around the haematoma to be even greater, but the generalised cerebral swelling had decreased, the lateral ventricles returning towards normal. Focal cerebral oedema around an intracerebral haemorrhage does not normally respond to such treatment, and it would be hard to explain the rapid clinical improvement seen on this basis.

We are unsure of the relevance of a computed tomogram before treatment in this case. It was not clinically indicated. Although the cause of the spontaneous haematoma was not established, such complications are not unknown in haematological malignancies, and the pathogenesis is to some extent irrelevant to the point of the case. Results of examination of cerebrospinal fluid were not reported as a lumbar puncture was not performed; we consider a spinal tap to be contraindicated in the presence of brain swelling and an obvious focal cerebral lesion.

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Neck sprains after car accidents

SIR,—I would like to contribute to the debate arising from Mr K M Porter's editorial on neck sprains after car accidents.[1]

Of 102 consecutive patients with acute neck sprain injuries, two thirds had no symptoms at two months and were cured. One third had symptoms at two years, but only 10% had persistent symptoms.[2] These patients with moderate to severe symptoms were found at three year follow up to have minimal disability even though they still had symptoms. None took up the offer of further investigation and treatment and none were pursuing legal claims.

We attempted to elucidate the pathology of the condition in four patients with acute neck sprains with magnetic resonance imaging at Guy's Hospital. All patients had moderate to severe symptoms and in all four the result of the imaging was negative. Similar negative results were reported in 15 patients with whiplash injuries by Rofo.[3] The evidence so far suggests that the uncomplicated whiplash injury is a musculoligamentous strain or sprain to some or all of the 23 joints of the cervical spine.[4]

We found, however, that immobilisation with a cervical collar and taking analgesics were effective in the initial phase of the injury (first and second weeks). Early mobilisation and physiotherapy speeded up the resolution of symptoms.[5]

We carry out regular reviews and an assessment at around three months. Patients with symptoms of persisting neurological deficit or who had preexisting symptomatic cervical spondylosis have a protracted course. They seem to have a different pathology and natural progression of their symptoms and they should be treated as a separate entity, as suggested by Dr Pearce.[4] We find such a management regimen helpful in distinguishing patients with genuine symptoms from those who might exaggerate or prolong their symptoms in order to enhance their financial compensation.

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1 Porter KM. Neck sprains after car accidents. *Br Med J* 1989;**298**: 973-4. (15 April.)
2 Maimaris C, Barnes M, Allen M. Whiplash injuries of the neck: a retrospective study. *Injury* 1988;**19**:393-6.
3 Rofo NMR. Tomography of the cervical spine. *Fortschr Rontgenstr* 1986;**145**:657-60.
4 Pearce JMS. Neck sprains after car accidents. *Br Med J* 1989;**298**:1581. (10 June.)
5 McKinney LA, Dornan JO, Ryan M. The role of physiotherapy in the management of acute neck sprains following road traffic accidents. *Arch Emerg Med* 1989;**6**:27-33.

Cigarette smoking on increase

SIR,—In January as part of its prebudget antismoking campaign the BMA claimed that in 1988 smoking had increased by 2·2% compared with the previous year. This claim was attributed to Ms Joy Townsend of the Medical Research Council's statistical unit and was subsequently withdrawn apparently because it could not be substantiated, but not before mischief had been done with questions asked in the House of Commons and an early day motion laid. Fortunately, government departments do their own calculations on up to date data and do not rely on the Medical Research Council.

A recent article by Ms Joy Townsend mentioned data from the Central Statistical Office, which is already in the public domain.[1] Ms Townsend then attempted to develop an argument suggesting that the industry by timing the reporting of its data may influence these statistics. In fact, the data from the Central Statistical Office are always subject to revision, and the office publishes revision dates. Ms Townsend's article contains a direct misquote of the expenditure on tobacco in the first quarter of 1988, which was £1696m not £1969m as she suggested (incidentally, that was total expenditure on tobacco and not cigarettes alone), revised in December to £1667m, a drop of £29m not the £300m implied by the article.

What the industry can say with confidence based on its sales to the trade is that in 1988 sales of manufactured cigarettes to the trade were about 1% lower than in the previous calendar year. This trend accords with data from the Customs and Excise and the Central Statistical Office, which broadly confirm that the tobacco market is static. For Ms Townsend to draw conclusions based on non-significant trends, which led to the headline "Cigarette smoking on increase" is extremely surprising and certainly misleading.

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1 Townsend J. Cigarette smoking on increase. *Br Med J* 1989;**298**: 1272. (13 May.)

AUTHOR'S REPLY,—Mr Owen's letter exemplifies the problem of the tobacco industry's approach to statistics. It is not true that the 2·2% increase in smoking quoted by the BMA was attributed to me and subsequently withdrawn. This figure is the year on year increase from the first quarter of 1988 to the first quarter of 1989 published in the *Monthly Digest of Statistics* from July to November 1988. The 1988 figure was then revised by the Central Statistical Office (not by the Medical Research Council, which does not produce such statistics) in January 1989 for the December 1988 digest. The reason given by the Central Statistical Office was that the tobacco industry had decided in 1988 to stop providing data, then changed its mind and provided the lower estimates in retrospect. The original data and the revised data were both published by the Central Statistical Office. To imply that they originated from the Medical Research Council is misleading.

The Central Statistical Office gives consumers' expenditure on cigarettes in 1988, adjusted for price changes, as £5935m, which is £33m (in 1985 prices) higher than its figure for 1987. It is also a significant change in direction from the decreases in cigarette consumption of the previous seven years. (The expenditure on tobacco for the first quarter of 1988 was misprinted in the article as £1969m; the figure supplied was £1696m.)

The Customs and Excise publish data on cigarettes released for home consumption. It is a volatile series and not related to consumption in the short term, even over a year, as the tobacco industry well knows. Cigarettes released to consumption in 1987 and 1988 show, however, a 3% increase over the previous two years, and these are the only two years to show an increase this decade. This is further evidence of a new upturn in cigarette consumption.

The tobacco industry can be the only industry that is so sensitive to suggestions that sales of its product are increasing.

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Anteroposterior radiography in cervical pain of non-traumatic origin

SIR,—Dr Janet E Page and colleagues report that in some circumstances an anteroposterior view of the cervical spine is not justified.[1]

We have recently carried out a survey to investigate the same problem and also found that the anteroposterior film is of little value in routine cervical spine assessment for similar symptoms and concluded that its use could justifiably be curtailed. One hundred consecutive general practitioner referrals to our department for cervical spine assessment with presenting symptoms of neck pain, neck stiffness, headache, or arm paraesthesia were studied. Two radiologists independently reported on every patient, using either the lateral film alone or both the anteroposterior and lateral films but without any clinical information.

In 92 patients an abnormality was detected. Loss of lordosis (72 patients), narrowing of the disc space (68), osteophyte formation (64), and facet joint degeneration were apparent in the lateral film. Uncovertebral degeneration (48 patients) and cervical ribs (3) were seen only in the anteroposterior projection. There was no evidence of neoplasia, fracture, dislocation, soft tissue swelling, or erosions in this group. Uncovertebral degeneration was invariably associated with degenerative changes at similar levels seen in the lateral film, and its demonstration was of no extra diagnostic value. Cervical ribs were seen in three patients, but none of these had documented symptoms in the arms.