ment. It would be very interesting if radiologists could do a three-month survey to find out what percentage of *x*-rays showed changes of any significance.

I suggest, therefore, that the Department of Health and Social Security, the Law Society, the defence societies, the B.M.A., and the nursing organizations get together for a significant reappraisal of what might and might not be considered negligence. If a more realistic standard was adopted some hundreds of millions of pounds could be put to much more effective use in the Health Service.—I am, etc.,

H. PIERCE HUGHES

Loughborough, Leics

Treatment of C.N.S. Involvement in Systemic Lupus Erythematosis

SIR,—Dr. Y. Levo (14 September, p. 689) proposes the use of intrathecal steroids and cytotoxic drugs in cases of systemic lupus erythematosus (S.L.E.). We believe this proposal to be illogical and potentially dangerous.

A true inflammatory vasculitis in the brain of patients with S.L.E. is rare[1] and despite recent immunological studies[2] there is no convincing evidence to support the view that the neuropsychiatric manifestations of S.L.E. are due to an immune disorder acting primarily within the brain. It should also be pointed out that corticosteroid drugs administered by mouth or intravenously do enter the central nervous system and there is no necessity to administer them intrathecally.[3] The development of C.N.S. leukaemia seems to be related to the failure of other antileukaemic drugs, such as mercaptopurine, to pass the blood-brain barrier in adequate amounts, coupled with the facility with which many leukaemic cell lines become resistant to corticosteroids. Thus the analogy between C.N.S. involvement in S.L.E. and in acute leukaemia is quite false. Dr. Levo's contention that the intrathecal administration of immunosuppressive drugs might benefit patients with S.L.E. therefore has little theoretical support.

More important is the fact that intrathecal cytotoxic drugs, while of undoubted benefit in the control of C.N.S. leukaemia,[3] may produce severe C.N.S. damage. The suggestion that in S.L.E. such treatment "might be given . . . even before the appearance of overt neurological manifestations" strikes us as grotesque. Intrathecal or intraventricular methotrexate, together with other cytotoxic drugs and irradiation of the neuraxis, has produced a necrotizing encephalopathy in a number of children suffering from acute leukaemia or brain tumours.[4-6]

At the Hammersmith Hospital six leukaemia patients treated in this way have developed severe neurological disease not attributable to leukaemia infiltration. These patients had leukaemia of the meninges and were treated with methotrexate administered by lumbar puncture, followed by insertion of a subcutaneous cerebrospinal fluid reservoir and the administration of 2,500 rads to the head. Further injections of methotrexate and cytarabine were made via the reservoir. Apparently complete control of the meningeal leukaemia was secured in all cases, but neurological complications occurred in each some weeks or months after the initiation of this therapy. The complications observed included aphasia and hemiplegia of rapid onset, progressive dementia, and progressive paraplegia. It is plain that only a consistently life-threatening condition such as meningeal leukaemia could justify therapy which entails the risk of such grave sequelae.

The necropsy findings in one patient, a youth of 15 with signs of bilateral cerebral involvement and a progressive dementia over four months, were extensive necrosis of white matter, without vascular involvement, in the anterior half of the cerebral hemispheres. In another case necrotizing leucoencephalopathy was found in the neighbourhood of the catheter which led from the subcutaneous reservoir to the right frontal horn.—We are, etc.,

P. D. LEWIS
ALEX S. D. SPIERS
C. A. PALLIS

Hammersmith Hospital,
London W.12

1 Johnson, R. T., and Richardson, E. P., *Medicine*, 1968, **47**, 337.
2 Lampert, P. W., and Oldstone, M. B. A., *Virchows Archiv; abt. A, pathologische Anatomie*, 1974, **363**, 21.
3 Broder. L. E., and Carter, S. K.. *Meningeal Leukemia.* New York, Plenum Press, 1972.
4 Kay, H. E. M., *et al., Archives of Disease in Childhood*, 1972, **47**, 344.
5 Shapiro, W. R., Chernick, N. L., and Posner, B., *Archives of Neurology*, 1973, **28**, 96.
6 Norrell, H., *et al.. Cancer*, 1974, **33**, 923.

Squint

SIR,—With reference to your leading article on this subject (17 August, p. 430), recent developmental paediatric and ophthalmic work has shown that visual acuity is developed by between two and three years of age. Similarly, binocular function is well advanced by this age. In view of this it is vital that all deviations of the eyes are treated expeditiously to obtain parallelism in the shortest possible time. Failure to carry out treatment at that time will hazard, if not completely mar, the possible development of binocular vision and possibly sight.

With regard to myopia of congenital or early onset, it should be noted that the deviation is usually convergent, as the child has never had the visual stimulus or need to diverge the eyes from the near position for a distant object. As a general rule operation should be undertaken early, when indicated, in the hope of developing some natural fusion. One should not wait until five years of age before operating since orthoptic treatment at this age is supplementary, by consolidating the binocular function.—I am, etc.,

G. V. CATFORD

London W.1

SIR,—It is unfortunate that your recent leading article (17 August, p. 430) fails in certain important aspects to provide an accurate and up-to-date assessment of the subject.

The definition of "concomitant" as applied to squint is somewhat misleading because it applies essentially to a type of squint wherein the deviation remains the same in all directions of gaze, and though it is frequently associated with a sensory anomaly, this is usually the result of the squint rather than its cause. The paragraph which deals with the management of a paralytic squint is most misleading because such a squint may occur at any time of life, and it is by no means common for the squint to become rectified spontaneously even when it is possible to treat the underlying cause of the squint. It is, of course, essential in an incomitant squint to delay active treatment for a sufficiently long period to ensure that there is going to be no spontaneous resolution of the squint, and during this time it may be necessary to advise the occlusion of each eye in turn to avoid the inconvenience of a persistent diplopia, but there is certainly no indication to give advice about the turning of the head, because a compensatory head posture is adopted reflexly by the patient if this is sufficient to avoid diplopia by maintaining the eyes in a position of gaze which permits fusion (bifoveal fixation). It should be stressed that the ultimate treatment of many incomitant squints is of a surgical nature in order to restore binocular vision in the primary position and in as much as possible of the binocular field of fixation, though in certain circumstances an operation may be avoided by the use of prisms, particularly the new form of membranous Fresnel prisms.

It is well recognized that the amblyopia which occurs in the uniocular squint (strabismic amblyopia) is confined to a squint which occurs in early childhood, but there is little prospect of obtaining any satisfactory relief of the amblyopia if the treatment is delayed until the age of about 7 years, particularly because of the likelihood of the development of an associated anomaly of retinal fixation (eccentric fixation), so that occlusion of the good eye is only a worthwhile procedure if it is carried out at a stage when the point of eccentric fixation has not become unduly established, with a chance of a shift of fixation from the eccentric point to the fovea or to an area of the retina adjacent to the fovea (parafovea) simply by effective occlusion of the fixing eye.

It is of course well recognized that the myope is liable to develop some form of divergent squint, which is usually of a latent type (exophoria). However, when the myopia is present in infancy so that it is essentially of a congenital nature the squint which may develop is almost invariably of a convergent nature because, in this position of the eyes, the child is able to obtain a clear form of binocular single vision in the presence of uncorrected myopia.

It is suggested in the leading article that, except in congenital cases, an operation for squint is usually deferred until about the age of 5 years so that it is not carried out until the child is able to take part in orthoptic treatment. This is an attitude which has had little credence in the United Kingdom for a considerable period of time because there is no evidence that orthoptic treatment alone is able to provide a normal form of fusion. It is usual, therefore, for the surgical treatment of a squint in childhood to be carried out as soon as possible after there has been a relief of the amblyopia and any anomaly of retinal fixation of the squinting eye by occlusion and also a correction of any underlying refractive error by spectacle lenses. In this way the eyes are placed in a position in which it is possible to obtain some form of fusion, even though this may prove to be of an