the instigation of their area review committee on non-accidental injury, to start pilot schemes along these lines.

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SIR,—We would like to take the opportunity of commenting on the important points made by two recent correspondents, Dr P C Corry and Mr G T Meredith (23 April, p 1084).

There is certainly a need to look at larger samples than we were able to include in our study (5 March, p 624). However, we should not be deterred from considering preventive action simply because the percentage of newborns "at risk" seems to be so large. In our experience intervention frequently does not have to be massive. Open recognition of the parents' difficulties in caring for the child can result in a more positive attitude towards existing services, which can often be adapted to provide the help the family needs.¹

Our data were collected at the maternity hospital. Thus most information, not unnaturally, was concerning the mother. This does not mean that all the children were battered by their mothers. We have found that both parents are often deeply involved in the abuse of their child, irrespective of who actually inflicts the injuries; it is not necessarily helpful to concentrate on "who did it." Sometimes the parent who is not the identified abuser in one incident subsequently batters the same child or marries another battering partner.

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¹ Beswick, K, Lynch, M A, and Roberts, J, *British Medical Journal*, 1976, 2, 800.

Icelandic disease (benign myalgic encephalomyelitis or Royal Free disease)

SIR,—We were interested to read your expert's reply to the question on the nature of Icelandic disease (9 April, p 965). We should like to present the following additional information:

(1) None of the names in common use is completely descriptive of this syndrome, which is not truly benign, is not always myalgic, and has no proved connection with hysteria or neurasthenia.¹

(2) The sufferers are usually parents of young children or members of the armed Forces or of the teaching, nursing, or medical professions. The classic illness has a biphasic pattern with an initial episode of gastrointestinal or upper respiratory tract infection, with or without a rash and lymphadenopathy, affecting either the patient or juvenile members of the family.

(3) The most characteristic presentation is profound fatigue and muscular weakness coming on during the day and increasing in severity with exercise—a diurnal rhythm contrary to that found with other forms of depression.

(4) Other symptoms that may accompany the initial illness or a relapse include muscular fasciculation, paraesthesiae, and disturbance of vision, with which nystagmus may be seen. Autonomic disturbances such as orthostatic tachycardia, vasomotor instability, and episodes of pallor are often prominent. Encephalitic disturbances include severe depression, emotional lability, and difficulty in concentration.

(5) Physical findings may include atypical lymphocytes in the peripheral blood, hepatitis, electromyographic evidence of myopathic and neuropathic disorders, and abnormal glucose tolerance curves.^{2,3}

(6) Relapses are a typical feature of this illness and may occur even after several years of good health.⁴

Last year a research group was formed to study the aetiology and epidemiology of this syndrome. Our investigations so far indicate that the illness may accompany the more common viral infections and that the unique fatigue pattern may be due to mitochondrial damage.

As objective manifestations of the disease can still be present over 30 years after the initial illness we should be glad to hear from former sufferers who were members of the medical and nursing staff of the Royal Free Hospital or other institutions which have experienced outbreaks. Communications should be sent to the honorary secretary of the group (JVD) at the address below.

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¹ Ramsay, A M, *Update*, 1976, 13, 539.

² Parish, J G, *IRCS Journal of International Research Communications*, 1974, 2, 22.

³ Leon-Sotomayor, L, *Epidemic Diencephalomyelitis*. New York, Pageant Press, 1969.

⁴ Marinacci, A A, and Von Hagen, K, *Electromyography*, 1965, 5, 241.

Mechanism of chloroquine induced involuntary movements

SIR,—I read with interest the report by Drs E M Umez-Eronini and Elspeth A Eronini on chloroquine induced involuntary movements (9 April, p 945). May I throw some light on the probable mechanism of these movements?

Chloroquine and phenothiazines combine avidly with melanin both in vitro and in vivo; this is believed to be based on a charge transfer reaction facilitated by the presence of electrons in the fused coplanar ring structures of both chloroquine and phenothiazines.¹ Melanin is a derivative of DOPA (phenylalanine → tyrosine → dihydroxyphenylalanine (DOPA) → DOPA-quinone → melanin). Chloroquine, a 4-aminoquinoline derivative, crosses the blood-brain barrier, and the brain and spinal cord contain 10-30 times the amount present in plasma.² It is quite logical to infer from the structure-activity relationship that chloroquine may also avidly combine with the dopaminergic receptors (like melanin) and thus by blocking those receptors (like phenothiazines, butyrophenones, metoclopramide, etc) in the nigrostriatal system it may produce involuntary movements. It is another example of drug-induced extrapyramidal disorders.

In the light of this it is better not to give patients with chloroquine induced involuntary movements phenothiazines like chlorpromazine, which may aggravate the situation. Anticholinergic anti-Parkinsonian drugs like bztropine, benzhexol, orphenadrine, ethopropazine, procyclidine, etc should be given in these cases as their action is not dependent on the dopaminergic receptors in the nigrostriatal system.

It needs to be mentioned that other members of 4-aminoquinoline family like amodiaquine, cycloquine, and hydroxychloroquine may also

produce involuntary movements in the same way as chloroquine.

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¹ Potts, A M, in *Physiological Pharmacology*, ed W S Root and F G Hofmann, vol II, part B, pp 329-397. New York, Academic Press, 1965.

² Rollo, I M, in *The Pharmacological Basis of Therapeutics*, ed L S Goodman, A Gilman, A G Gilman, and G B Koelle, 5th edn, p 1051. New York, Macmillan, 1975.

Insurance companies' attitude to psychiatric illness

SIR,—In recent issues you have printed letters from two psychiatrists expressing concern at the harsh attitude towards minor psychiatric illness taken by insurance companies, and the untoward experiences of five neurotic patients are mentioned. Dr J T Hutchinson (9 March, p 775) describes excessive loading against those who seek life insurance and Dr A B Sclare (16 April, p 1031) from his experience with employees of insurance companies becoming patients stresses the lack of confidentiality, punitive attitudes towards mental illness, and absence of discrimination between mild and serious psychiatric illness.

Although I would agree that the attitudes of the companies should be enlightened and informed, it is worth mentioning that the neuroses do carry a slightly increased risk of premature mortality as demonstrated in the studies by Babigian and Odoroff,¹ by Innes and Millar,² by Keehn *et al*,³ and by me.⁴ Further findings on the extent of this increased death risk will be published soon, but figures have generally been about 1.5 times those for the matched general population.

It would seem reasonable that psychiatrists should ask the insurance companies to take the psychiatric diagnosis into account in their assessment of weighting so that neuroses and other disorders can be differentially weighted. This would result in a slightly increased premium for neurosis which should by no means be prohibitive.

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¹ Babigian, H M, and Odoroff, C L, *American Journal of Psychiatry*, 1969, 126, 470.

² Innes, G, and Millar, W M, *Scottish Medical Journal*, 1970, 15, 143.

³ Keehn, R J, Goldberg, I D, and Beebe, G W, *Psychosomatic Medicine*, 1974, 36, 27.

⁴ Sims, A C P, *Lancet*, 1973, 2, 1072.

Maintenance digoxin

SIR,—Concerning Dr B J O'Driscoll's comments (16 April, p 1028) on our paper (19 March, p 749), comparison with pioneering studies is difficult because of historical emphasis on digitalisation rather than maintenance therapy, the fact that digitalis used to be pushed until cardiotoxicity occurred, and differences in concomitant therapy. Although Sir James Mackenzie¹ in 1910 obtained the best response in failure associated with "rapid" atrial fibrillation, he did not consider that decreasing the rate was the primary mechanism: "The good results obtained by the use of digitalis are doubtless due to the specific action of the drug on the function of tonicity." Windle² in 1917 demonstrated the value of digitalis in patients with pulsus alternans, while