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We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

Creutzfeldt-Jakob disease and hospital care

SIR,—There is often uncertainty among health care personnel and health administrators about the possible occupational hazards of caring for patients with Creutzfeldt-Jakob disease (CJD), despite several articles[1-3] analysing critically this potential danger according to the knowledge available. These have called, though with reasonable reservations, rather for awareness of the admitted potential risk and for maintaining general precautions[1] during examination and nursing of patients with CJD.

The uncertainty is justified by the uniformly fatal outcome of CJD; its transmissible nature; the unusual properties of the virus; reported iatrogenic transmissions (which might occur in about 10% of cases)[4]; and growing evidence of the presence of the agent not only in the central nervous system but also in visceral organs, superficial tissues of the eye, the cerebrospinal fluid, and the blood.[1 5] The Clinicopathological Conference on a case of Creutzfeldt-Jakob disease (17 March, p 729) touched only briefly on the infectivity of the disease, and it was stated that there has been no indication of transmission by peripheral inoculation. Certainly there has so far been no direct evidence that it can occur in man by peripheral infection. But the only evidence strongly suggestive of possible transmission by contact yields at least three sets of cases in spouses. An epidemiological survey of 218 familial CJD cases, constituting over 15% of patients reported, indicates a greater likelihood of some form of microenvironmental contamination[4] than of other mechanisms of infection. Similarly, the present evidence of at least eight apparent foci of high incidence of CJD suggests common exposure to the virus.[6]

In the same survey, Masters *et al* show that, of 308 patients whose occupation was ascertained (from a total of 1435 CJD cases studied), 18 (6%) were in the health professions; but, of the health professionals, nearly half of those affected were nurses (six nurses and one nursing aide). This appears as more than an unfortunate chance finding and seems to support the recently proposed programme[7] of active surveillance of those exposed to the causal agent in the difficult nursing of patients during the dementia and myoclonic phase of CJD (mean duration 3·9 months, range 0·5-36 months).[5] This programme would be considerably more effective if performed in adequately designed and equipped facilities, with the staff deliberately perfecting and maintaining a rigid regimen of precautions.

Such a system, providing for a nation-wide centralisation of CJD cases, is now being put into practice in Czechoslovakia. We believe that only the most rigid safety requirements, at least until more hard facts are available, will reduce the danger of potential exposure to the causal agent of CJD and help to set our minds at rest. The precautions advised earlier[1] were extended recently in order to meet certain situations in families at high risk of developing transmissible encephalopathy.[5] Our efforts so far are concerned with overt CJD cases, which have been diagnosed more frequently in recent years.[6]

The epidemiological significance of the non-characteristic prodromal phase (mean duration 3·5 months, range 0·5-24 months),[5] observed in 80% of CJD cases,[8] remains to be evaluated. Nevertheless, the early phase of the disease observed in primates in whose central nervous system the CJD agent replicated after experimental inoculation closely approximated to the prodromal symptoms seen in human disease.[9]

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[1] Gajdusek, D C, *et al*, *New England Journal of Medicine*, 1977, **297**, 1253.
[2] Brown, P, *et al*, *Revue Neurologique*, 1978, **134**, 277.
[3] *British Medical Journal*, 1978, **1**, 463.
[4] Masters, C L, *et al*, in *Slow Transmissible Diseases of the Nervous System*, ed W J Hadlow and S B Prusiner. New York, Academic Press, in press.
[5] Cook, R H, and Austin, J H, *Archives of Neurology*, 1978, **35**, 697.
[6] Masters, C L, *et al*, *Annals of Neurology*, 1979, **5**, 177.
[7] Mayer, V, *et al*, in *Slow Transmissible Diseases of the Nervous System*, ed W J Hadlow and S B Prusiner. New York, Academic Press, in press.
[8] Brown, P, and Cathala, F, in *Slow Transmissible Diseases of the Nervous System*, ed W J Hadlow and S B Prusiner. New York, Academic Press, in press.
[9] Court, L, *et al*, *Revue d'Electroencéphalographie et Neurophysiologie Clinique*, 1975, **5**, 335.