helped the mentally disabled over the past 150 years.

JOHN CRAMMER

South Grange
Steeple Aston
Oxon

Reference

CRAMMER, JOHN (1990) *Asylum History: the Buckinghamshire County Pauper Lunatic Asylum – St Johns*. London: Gaskell.

Who was Jellinek?

SIR: Hore (*Journal*, 1990, **157**, 786–789) has provided a fair-minded reappraisal of Jellinek's *The Disease Concept of Alcoholism* (Jellinek, 1960). There are three issues on which he might perhaps be able to give us some further thoughts.

The first question is simply "Who was E. M. Jellinek?" Amazingly, the answer to that query remains obscure. Jellinek has been described as a biostatistician, but his understanding of statistical inference was limited, on the evidence of his published research (Jellinek, 1952). He held no medical or psychological qualifications, and *The Disease Concept* suggests that he was not well versed in issues relating to psychiatric taxonomy. At the same time, Jellinek's professional influence and the personal impact of his warmth and enthusiasm, are beyond doubt – we all stand in his debt.

Secondly, there is a question to be explored in relation to the historical antecedents of Jellinek's ideas. There is little in *The Disease Concept* which is not to be found in 19th-century authorities. Anyone who has, for instance, read Kerr (1888) or Crothers (1893) is likely to find in Jellinek a sense of *déjà vu*. Alcoholism was as much a 'disease' to those earlier writers as to Yale in the 1960s, and Kerr and Crothers had their typologies. Furthermore, and just as with Jellinek, the 19th-century activists confused 'disease' as a campaigning slogan, with disease as scientific formulation.

Thirdly, one might question whether Dr Hore is right in suggesting that the dependence syndrome (Edwards & Gross, 1976) "incorporates" Jellinek's views. It would, of course, have been impossible to write anything on alcoholism in the 1970s without an awareness of Jellinek, but those who put forward the syndrome formulation were also influenced by many other currents in the flow of contemporary science – learning theory formulations for instance (Edwards, 1986), and the epidemiological research which pointed to the shifting and multifarious nature of drinking problems within the community (Room, 1977). To suggest that the dependence syndrome was the disease concept reincarnate would be ahistorical.

On a more minor note, one might wish to correct the record as to the year of the *British Journal of Addiction*'s first publication under one of its several earlier titles – 1884, not 1892.

GRIFFITH EDWARDS

Addiction Research Unit
National Addiction Centre
Institute of Psychiatry
London

References

CROTHERS, T. D. (1893) *The Disease of Inebriety*. New York: E. B. Treat.

EDWARDS, G. (1986) The alcohol dependence syndrome: a concept as stimulus to enquiry. *British Journal of Addiction*, **81**, 171–183.

—— & GROSS, M. M. (1976) Alcohol dependence: provisional description of a clinical syndrome. *British Medical Journal, i*, 1058–1061.

JELLINEK, E. M. (1952) Phases of alcohol addiction. *Quarterly Journal of Studies on Alcohol*, **13**, 673–684.

—— (1960) *The Disease Concept of Alcoholism*. New Haven: College and University Press.

KERR, N. (1888) *Inebriety*. London: H. K. Lewis.

ROOM, R. (1977) Measurement and distribution of drinking patterns and problems in general populations. In *Alcohol-Related Disabilities* (eds G. Edwards, M. M. Gross, M. Keller, J. Moser & R. Room). Geneva: World Health Organization.

Depression and the menopause

SIR: Neither the letter from Studd *et al* (*Journal*, 1990, **157**, 931–932), nor the original review paper by Ballinger (*Journal*, 1990, **156**, 773–787) considers another important aspect of depression and hormone replacement therapy (HRT), namely progestogen-induced depression.

The majority of menopausal women have intact uteri and so require additional treatment with progestogen to protect against putative endometrial cancer. Progestogen-induced depression is a well recognised complication of such treatment (Holst *et al*, 1989). The symptoms may be severe, including suicidal ideation, and specific antidepressant drugs then become necessary.

Gath & Iles (1990) have made a distinction between "depressed mood" and "depressive disorder" suggesting that the former will respond to oestrogen replacement but not the latter. They further state that, "if the diagnosis is depressive disorder the primary treatment is not oestrogen but standard psychiatric treatment, whether pharmacological or psychological, or both". Dr Ballinger's review refers to the treatment of "depressive illnesses" and so perhaps the argument should be confined to the illness rather than the emotion.

In this context, clinical trials of oestrogen therapy in climacteric depression should be judged on the precision of the psychiatric criteria employed. Established diagnostic procedures for depressive illness should be used, for example, those outlined in DSM–III–R (American Psychiatric Association, 1987). Furthermore, the severity of psychiatric morbidity and its response to any form of treatment needs to be assessed by standard psychiatric rating scales. At least in one study, which utilised the Beck Depression Rating Scale, no significant difference was demonstrated between oestrogen (Premarin) and placebo (Campbell, 1976).

On the other hand, Studd *et al* quote one of their own papers (Montgomery *et al*, 1987) on a placebo-controlled study of the beneficial effect of oestradiol implants on depression, in a group of women attending their menopausal clinic. However, the measuring instrument for depression that they used was the SRD-30 (self-rating scale of distress; Kellner & Sheffield, 1973), which is little known to psychopharmacology research workers. Whether or not it represents a valid measure of depressive illness is debatable, since the following items which would be considered indispensable in most psychiatric depression rating scales are missing: suicidal tendencies, retardation, loss of libido, somatic gastric symptoms, weight loss, hypochondria, insight, and diurnal variation (although many believe the last named to be a diagnostic criterion rather than a measure of severity). These are all items to be found in the universally accepted Hamilton Rating Scale for Depression (HRSD), which is that most frequently employed internationally for the measurement of severity of depressive illness.

The relief of menopausal misery has been revolutionised by the introduction of HRT, but it is not a universal panacea. When depression occurs at the menopause, whether idiopathic or iatrogenic, specific antidepressant drug therapy should not be spurned. As Studd *et al* (1990) have so rightly emphasised, there is a considerable need for properly controlled clinical trials with both hormones and antidepressants, or even a combination of both.

DAVID WHEATLEY

The Stress Clinic
The Maudsley Hospital
London SE5 8AZ

References

CAMPBELL, S. (1976) Double-blind psychometric studies on the effects of natural estrogens on post-menopausal women. In *Management of the Menopause* (ed. S. Campbell). MTP Press.

GATH, D. & ILES, S. (1990) Depression and the menopause. *British Medical Journal*, **300**, 1287–1288.

HOLST, J., BACKSTROM, T., HAMMERBACK, S., *et al* (1989) Progestogen addition during oestrogen replacement therapy – effects on vasomotor symptoms and mood. *Maturitis*, **11**, 13–20. Ireland: Elsevier Scientific Publications.

KELLNER, R. & SHEFFIELD, B. T. (1973) Self-rating scale of distress. *Psychological Medicine*, **3**, 88–100.

MONTGOMERY, J. C., APPLEBY, L. & BRINCAT, M. (1987) Effect of oestrogen and testosterone implants on psychological disorders in the climacteric. *Lancet*, *i*, 297–299.

STUDD, J., WATSON, N. R. & MONTGOMERY, J. (1990) Depression and the menopause. *British Medical Journal*, **300**, 1653.

SIR: The importance of temperamental differences in gender-mediated clinical features of depressive illness is supported by women's less active, more ruminative responses (*Journal*, December 1990, **157**, 835–841), linked to dysfunction of the right frontal lobe in which the metabolic rate is higher in females (Friedman & Jankovic, 1990). The role of gender-related cerebral asymmetries was supported by fluvoxamine and fluoxetine-induced apathy and indifference in a predominantly female sample (4 of 5) with panic or depression (Hoehn-Saric *et al*, 1990), and by fluoxetine-induced bradycardia accompanied by faintness or syncope in two women (Ellison *et al*, 1990) which may have been due to serotonergic-mediated inhibition of dopamine lateralised to the right hemisphere (Friedman & Jankovic, 1990). This hypothesis was supported by suppression of the right hemisphere decreasing motor-exploratory aspects of directed attention (Spiers *et al*, 1990), and by unilateral cerebral inactivation producing differential left/right heart-rate responses (Zamrini *et al*, 1990).

ERNEST H. FRIEDMAN

1831 Forest Hills Boulevard
East Cleveland, OH 44112-4313

References

ELLISON, J. M., MILOFSKY, J. E. & ELY, E. (1990) Fluoxetine-induced bradycardia and syncope in two patients. *Journal of Clinical Psychiatry*, **51**, 310–311.

FRIEDMAN, E. H. & JANKOVIC, J. (1990) Tardive oculogyria (letter and reply). *Neurology*, **40**, 728.

HOEHN-SARIC, R., LIPSEY, J. R. & MCLEOD, D. R. (1990) Apathy and indifference in patients on fluvoxamine and fluoxetine. *Journal of Clinical Psychopharmacology*, **10**, 343–345.

SPIERS, P. A., SCHOMER, D. L., BLUME, H. W., *et al* (1990) Visual neglect during intracarotid amobarbital testing. *Neurology*, **40**, 1600–1606.

ZAMRINI, E. Y., MEADOR, K. J., LORING, D. W., *et al* (1990) Unilateral cerebral inactivation produces differential left/right heart rate responses. *Neurology*, **40**, 1408–1411.

Racism and psychiatry

SIR: Dolan & Evans (*Journal*, December 1990, **157**, 936–937) wilfully misunderstand our comments on racism and psychiatry (Lewis *et al*, *Journal*, 1990, **157**, 410–415). Unlike the correspondents, we as