after their partners' death (Bornstein & Clayton, 1972) probably are common, though are mostly so mild as not to require the attention of psychiatrists. My own study suggests that age-correspondence-precipitated reactions are rare. When they do occur it is important that they be recognised as such, since they can sometimes present as florid psychotic states (Hilgard & Newman, 1959). I would be interested to know if any readers have experience of one.

JOHN BIRTCHNELL

MRC Social Psychiatry Unit
Institute of Psychiatry
De Crespigny Park
London SE5 8AF

References

BIRTCHNELL, J. (1981) In search of correspondences between age at psychiatric breakdown and parental age at death—"anniversary reactions". *British Journal of Medical Psychology*, **54**, 111–120.

BORNSTEIN, P. E. & CLAYTON, P. J. (1972) The anniversary reaction. *Diseases of the Nervous System*, **33**, 470–472.

HILGARD, J. R. & NEWMAN, M. F. (1959) Anniversaries in mental illness. *Psychiatry*, **22**, 113–121.

Depression and Urinary Free Cortisol

SIR: B. J. Carroll (*Journal*, February 1986, **148**, 218) questions the use of the radio-immuno assay (RIA) procedure and suggests that the urinary free cortisol (UFC) levels were unusually high in the patients in our study (*Journal*, October 1985, **147**, 429–433). He states that the values were "all well within the range expected for patients with Cushing's Disease".

In fact, only 38 of the 72 patients had a 24 hour UFC secretion above the normal range (25–130 μmg/24 hours). The mean pre-dexamethasone 24-hour UFC was 158.6 μgm/24 hours (SD = 77.8, range 33–378) which was higher than that found by Diegbold *et al* (1981) (mean = 122 μgm/24 hours). One likely explanation for this is the fact that the patients in our study were essentially drug free in comparison to the above study and that of Carroll *et al* (1976) in which patients were treated with psychotropic medication, including benzodiazepines, which have been shown to lower cortisol levels.

The NIMH study (Stokes *et al*, 1984) on drug-free patients reported mean pre-dexamethasone UFC levels of 148 μmg/24 hours in unipolar depressed patients, a result very similar to our own. Moreover, the post-dexamethasone UFC results in the two studies were remarkably similar with the NIMH study reporting levels of 59 μgms per 24 hours, (and 65 μgm per 24 hours in patients with unipolar depression), as compared to 65.4 μgm per 24 hours in our study of mainly unipolar depressed patients (Calloway *et al*, 1984).

Carroll also questions the validity of using RIA for measuring plasma cortisols in the DST. It has been established that RIA gives comparable results to competitive protein binding (Wilens *et al*, 1983) and RIA is now the most widely used method for assaying cortisols in DST studies of depressed patients (e.g., Stokes *et al*, 1984).

S. P. CALLOWAY

Fulbourn Hospital, Cambridge CB1 5EF

R. J. DOLAN

Maudsley Hospital, London

References

CALLOWAY, S. P., DOLAN, R. J., FONAGY, P., DE SOUZA, F. V. A. & WAKELING, A. (1984) Endocrine changes and clinical profiles in depression. *Psychological Medicine*, **14**, 749–65.

CARROLL, B. J., CURTIS, G. C., DAVIES, B. M., MENDELS, J. & SUGERMAN, A. A. (1976). Urinary free cortisol excretion in depression. *Psychological Medicine*, **6**, 43–50.

DIEBOLD, K., KICK, H. & SCHMIDT, G. (1981) Urinary free cortisol excretion in endogenously depressed and schizophrenic patients. *Psychiatria Clinica* (Basel), **14**, 43–48.

STOKES, P. E., STOLL, P. M., KOSLOW, S. H., MAAS, J. W., DAVIS, J. M., SWANN, A. C. & ROBINS, E. (1984) Pretreatment DST and hypothalmic-pituitary-adrenocortical function in depressed patients and comparison groups. A multicenter study. *Archives of General Psychiatry*, **41**, 257–267.

WILENS, T. E., ARANA, G. W., BALDESSARINI, R. J. & CREMENS, C. (1983) Comparison of solid-phase radioimmunoassay and competitive protein binding method for postdexamethasone cortisol levels in psychiatric patients. *Psychiatry Research*, **8**, 199–206.

Pseudodementia: Facts and Figures

SIR: The article by Drs Bulbena and Berrios (*Journal*, January 1986, **148**, 87–94) referred to the fact that speculation abounds in relation to the concept of pseudodementia. We decided to examine their observation "that there is no consensus on the use and application of the diagnosis of 'pseudodementia". We will avoid commenting on how one *diagnoses* a presentation. We were also interested in seeing how closely our findings approached their "stringent criteria"—cognitive impairment of the dementia-type, absence of a relevant organic disorder, and reversibility.

We sent questionnaires, a covering letter, and s.a.e's. to a random sample of Irish psychiatrists of senior status ($n = 65$). We placed a five week deadline for the return of correspondence. Twenty-nine questionnaires were returned.

The questionnaire consisted of three questions. The answers given to these, including only those given by more than one respondent (absolute numbers in brackets) were as follows:

1. What do you understand by the term pseudodementia?

Resembles an organic dementia (18), potentially reversible with/without treatment (6), functional (9), depressive (9), found usually in elderly (5), memory impairment an important component (12), and "hysterical", including Ganser's syndrome (7).

2. What do you consider to be its cause(s)?

Depression, especially in the elderly (21), anxiety/stress/loss (8), "hysteria"—dissociative (7), "Ganser's syndrome" (3), schizophrenia/schizophreniform (5), malingering (2). Parkinsonism, hypovitaminoses, hypothyroidism and other metabolic causes, and "age-related brain changes" were mentioned by individual respondents.

3. Please comment on the current use and validity of the term.

A reminder to the physician to search further (6), a retrospective diagnosis—with or without the use of treatment (4), a useful term if criteria are stated (4), should be confined to elderly depressives with a dementia-like syndrome (3), a useful term (3), a useless/confusing term (3), a term of rare applicability (3), and, lest we forget, depression and dementia may co-exist (2).

Our general impression was that pseudodementia, like "pseudodepression" (Feinberg & Goodman, 1984), is a loosely held concept amongst psychiatrists. We would suggest that it be replaced by a clearer statement of the findings. As an illustration of how this might be done we would refer to Feinberg & Goodman's (1984) "four 'Ideal Types' of depression plus dementia syndromes": depression presenting as dementia ("depressive pseudodementia"), dementia presenting as depression ("pseudodepression"), depression with secondary dementia ("dementia syndrome of depression"), and dementia with secondary depression ("depressive syndrome of dementia").

BRIAN O'SHEA
MARTIN RAHILL
CATHERINE MCCOLLAM

Newcastle Hospital
Greystones
Co. Wicklow

Reference

FEINBERG, T. & GOODMAN, B. (1984) Affective illness, dementia, and pseudodementia. *Journal of Clinical Psychiatry*, **45**, 99–103.

Huntington's Chorea Without Dementia

SIR: We would like to comment on the case report of Turner (*Journal*, May 1985, **146**, 548–550) concerning a patient who developed chorea in middle age, had a positive family history of Huntington's chorea, but who showed no evidence of dementia on formal psychological testing. The author questions the value of genetic counselling in this case and in particular whether it was justified, given that there were doubts about the diagnosis. We agree that the likely diagnosis for this patient is Huntington's chorea and would not be surprised by the lack of dementia, particularly with a relatively late age at onset. Indeed, lack of mental deterioration in some cases was noted by Davenport & Muncie (1916). Caution is required if there is a family history or non-progressive choreiform movements from childhood without dementia, since a likely diagnosis is benign hereditary chorea (Harper, 1978).

Genetic counselling should be part of the management of any family in which a relative is diagnosed to have Huntington's chorea, and the consequences of its deficiency are well documented by Martindale & Yale (1983). The form of the counselling is important and a general family meeting is inadequate; risks for an individual need to be considered separately and worries and fears dealt with on an individual basis. We question the statement that the seven living siblings had outlived their major risk period, since 10% of Huntington's chorea patients have an age of onset after 60 years (the age of the eldest sibling). The risk to the youngest sibling aged 42 years is considerably higher—approximately 38%, based on the life table method of Newcombe (1981).

Concern was expressed about the daughter of the index case, who may have broken off her engagement for a number of reasons. A full discussion of the genetic aspects might have eased her problem; in particular, information about the recent developments in DNA technology which may allow her to have children at low risk for developing Huntington's chorea, without changing the risks to herself (Harper & Sarfarzi, 1985).

O. W. J. QUARRELL
PETER S. HARPER

University of Wales College of Medicine
Department of Medicine
Heath Park
Cardiff CF4 4XN

References

DAVENPORT, C. B. & MUNICE, E. B. (1916) Huntington's chorea in relation to heredity and eugenics. *American Journal of Insanity*, **73**, 195–222.

HARPER, P. S. (1978) Benign hereditary chorea, clinical and genetic aspects. *Clinical Genetics*, **13**, 85–95.

—— & SARFARAZI, M. (1985) Genetic prediction and family structure in Huntington's chorea. *British Medical Journal*, **290**, 1929–1931.