ordeal. The mother experiences the tail-end of post-traumatic dysphoria as a reaction to the gladness and serenity of the first few days.

This hypothesis predicts that the blues will not be experienced by those for whom childbirth is an unhappy event. In them the only positive element is surviving an ordeal, which is also present in post-operative patients, and one would not expect their pattern of symptoms to differ from the surgical group.

One would expect a greater magnitude of mood change after delivery of the first child, and there is some support for this, at least for depression scores, in the data of Kendell et al (1981).

The theory is compatible with the great individual and collective variation seen in the timing of the blues, which has been reported to occur on the third day, the fourth day, the fifth day, the sixth and seventh days, any time during the first ten days, or not at all (references on demand). It is compatible with the association with neuroticism found by Kendell et al (1984), if 'neuroticism' is related to strong emotional reactions, with accentuation of both euphoric and dysphoric components. It does not readily explain why the blues predicts post-natal depression independently of neuroticism (Kendell et al, 1981, 1984), but studies of this prediction should exclude patients with chronic dysthymic states.

It follows from this interpretation of the maternity blues that we should study the two factors independently. The dysphoric factor may be related to the severity of tissue damage, pain, steroid production, etc., while the euphoric factor is related to emergence from a time of apprehension and suffering, and to pride and pleasure in the newborn. It would be interesting if a syndrome thought to be due to obscure hormone changes was due (in part) to normal happiness and excitement – but it is a reflection on the transience of human joys that the blues occurs so early in the puerperium.

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Characteristic Plasma Hormone Changes in Alzheimer's Disease

SIR: We read with interest the report by Christie et al (*Journal,* May 1987, 150, 674–681). The paper describes elevated TSH levels in a group of patients suffering from Alzheimer's disease (at three time points) in comparison within an elderly depressed group and, for females only, significantly higher TSH levels in the Alzheimer group compared with the elderly control group. We would like to make the following points.

In the group with Alzheimer's type dementia (ATD), two were still working and three were living alone, indicating probable early mild dementia. The point is made that a younger group was chosen because "younger patients with ATD have a more extensive loss of noradrenaline and somatostatin". However, this resulting group of ATD is atypical both in terms of age and severity. Consequently, it is questionable whether it is valid to extrapolate from such a group and suggest that these findings provide a generally useful test for Alzheimer's disease.

Although basal TSH levels and the TSH response to TRH can be affected by recent weight change, no mention is made of weight for any of the groups studied.

They fail to point out that the elderly control group is significantly younger ($P < 0.05$) than the ATD group, which may be of significance as TSH levels in females may increase with age (Tunbridge et al, 1977).

Another point not made in the paper is that the TSH values of the depressed group are significantly lower ($P < 0.02$) than those of the elderly control group. This will have the effect of accentuating the difference between the demented and depressed groups.

To draw attention to the raised TSH levels particularly in females is unfortunate. The study cannot really tell whether the effect is present in both sexes or confined to females, since they have studied only two healthy male controls.

The TSH data of Christie et al contain a gross outlier. They admit to this, justify its non-rejection, and carry out Mann-Whitney tests, which are not impaired by even such gross departures from the normal model. Nevertheless, they just quote summary statistics leaving this subject in. Also, the paper quotes standard errors rather than standard deviations. When standard deviations are included as well, the grossly skewed distribution becomes even more apparent.

It is clear that the *gross* differences reported in the paper are mainly due to the single outlying value.

Nevertheless, the standard deviation remains high in the ATD group and suggests that there is still considerable positive skewness, even if this single outlier is removed. Since it is not essential to remove the outlier, the Mann-Whitney tests remain valid and show a statistically raised TSH level at each time of day, in both sexes together and in females in particular. This has come about because there is a general shift upwards as well as a single grossly elevated value. What is incorrect is to interpret Table II in Christie *et al's* paper as meaning that TSH is raised *threefold* in females suffering with ATD.

Although the authors comment that this one patient with a high TSH level was euthyroid, they have not documented this satisfactorily. Hypothyroidism, reflected in elevated TSH levels, can occur in the presence of normal tri-iodothyronine (T_3) and thyroxine (T_4) levels and in the absence of overt clinical features. However, under these circumstances, elevated TSH levels are strongly correlated with the presence of thyroid antibodies (Tunbridge *et al*, 1977). The measurement of thyroid antibodies would therefore have helped to clarify the thyroid status of this one patient with high TSH levels. Furthermore, accurate assessment of clinical and biochemical thyroid status (including auto-antibodies) is mandatory in any study of this type, particularly in the presence of marginally elevated TSH levels.

In general, the basal TSH levels seem considerably higher in absolute terms than the levels one would expect to see in the most sensitive TSH assays. This does raise some questions about the actual TSH assay used. What is the normal cut-off for primary hypothyroidism with this assay?

Our group has also investigated thyroid function in 21 elderly patients with severe senile dementia of the Alzheimer type (Thomas *et al*, 1987). There was no substantial or statistically significant difference in basal TSH levels between patients and controls. The TSH response was blunted in the patient group, but all differences were small in biological terms and were within the laboratory's normal range, emphasising (in our study) the relative normality of neuroendocrine function, particularly thyroid status, in ATD.

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SIR: Dr Thomas and colleagues challenge the presentation of TSH data in our report. We would like to respond with some additional data that clarify and extend our finding of raised TSH concentrations in women with presenile Alzheimer-type dementia (ATD).

Firstly, we have deliberately studied a population of demented patients with a presenile onset and do not suggest that we can at this stage extrapolate our finding to the senile ATD population. Secondly, the patients studied were at an early stage of illness, but it is precisely at this time that there is a need for improved diagnostic tests, to help especially in the differential diagnosis of dementia and when distinguishing early dementia from depression.

We have considered whether recent weight change may have influenced our finding by examining the weekly weight records of the ATD patients during their repeated admissions to the research ward, and found that the weights of ATD patients remain remarkably constant over periods of up to 4 years. Weight gain was rarely, if ever, reported by the relatives of any of our ATD patients at the time of referral but, of course, weight loss is common during the latter stages of dementia and in the depressed patients and may tend to lower TSH concentrations in that group.

We have now extended our original study, and Dr Thomas's next four points are best answered by examining the original in conjunction with additional data for morning TSH concentrations. In the female subjects, morning TSH concentrations (mean of 3 samples) were: ATD, $n = 19$, median = 5.5 mU/l, range = 3.4 to > 50 mU/l; major depressive disorder (MDD), $n = 16$, median = 3.8 mU/l, range = 2.6 to 4.9 mU/l; other dementia patients (primarily multi-infarct dementia), $n = 8$, median = 4.7 mU/l, range = 3.3 to 5.7 mU/l; healthy control subjects, $n = 15$, median = 4.2 mU/l, range = 2.1 to 5.9 mU/l. TSH concentrations greater than 6 mU/l were found in 9 female ATD patients (6.3, 7.3, 8.0, 8.1, 8.3, 8.8, 10.2, 15.1 and > 50 mU/l respectively) but in none of the MDD, other dementia patients, or control subjects. The TSH concentrations in our ATD women are skewed and, as before, non-parametric statistics were used (the Kruskal-Wallis