

CORRESPONDENCE

To the Editor:

I have read with interest Dr John Bancroft's monograph (supplement 24) on the premenstrual syndrome which appeared in *Psychological Medicine* in 1993. Dr Bancroft proposes a 3-factor model to account for the complex symptoms experienced by women with this disorder: a timing factor imposed by the ovarian hormone cycle, a menstruation factor, and a vulnerability factor that determines how vulnerable a woman will be to the first two factors. Recent observations on the effect of hysterectomy on women with PMS provide strong support for a menstruation factor as a trigger for premenstrual symptoms, but little support for a timing factor imposed by the ovarian hormone cycle.

Women who have no uterus are rarely troubled by late luteal phase symptoms. We have shown that only 18.9% of a representative sample of a population of hysterectomized women complained of PMS and that only 2.6% had confirmed PMS (Braiden & Metcalf, 1994). We have shown that the removal of the uterus makes little difference to the ovarian hormone cycle (Metcalf *et al.* 1992a). We have also shown that six of a group of seven women who had regular premenstrual symptoms before hysterectomy, lost their cyclical symptoms following surgery but preserved their ovarian hormone cycle (Metcalf *et al.* 1992b). The combined findings strongly suggest that for most women the ovarian hormone cycle contributes little to premenstrual symptoms, except in so far as it sustains the menstrual cycle.

We do not know why the symptoms of most women with PMS were relieved by hysterectomy. Many things change when a uterus is removed, any of which may have a beneficial effect; uterine secretions disappear, general health often improves, the pain and discomfort associated with menstruation ceases, cyclical concerns about fertility disappear, women without menstrual markers do not know when to expect to feel unwell. We know little of their

separate contributions to the disorder, neither do we know whether a similar effect might be achieved by removing the endometrium alone. What is clear is that factors associated with menstruation have a potent effect on women with PMS and that future research should be directed towards their identification and to the elucidation of the mechanisms by which they influence premenstrual tension. On present evidence a hysterectomy may be the treatment of choice for many women with PMS.

REFERENCES

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The Author replies:

Dr Metcalf goes too far, I believe, in her conclusions. While I would agree with her that the role of the uterus, or the 'menstruation factor', in PMS has been seriously underestimated, her conclusion that the ovarian hormonal cycle 'contributes little to premenstrual symptoms, except in so far as it sustains the menstrual cycle' is not justified by the evidence she presents.

While hysterectomy provides an excellent model for studying the impact of the uterus on

cycle-related symptoms, it is important to keep in mind that the operation is only likely to have been carried out in women with substantial menstrual problems. Therefore, she is considering a selected group of women who are heavily loaded on the 'menstruation factor'. It is to be expected that hysterectomy will be associated with improvement in their cycle-related symptoms for that reason. There are, however, many women who experience premenstrual symptoms who do not have menstrual problems as such. The interaction between the 'timing factor' and aspects of vulnerability are likely to be important in that group.

I shall be most interested to read Dr Metcalf's latest paper, currently in press, but on the basis of what she tells us in her letter, it is not clear what point she is trying to make – is it that women with intact uteri (and without problems of dysmenorrhoea and menorrhagia) will have a much higher prevalence of confirmed PMS than her 2.6%? If so, what is her evidence for this?

The hysterectomy studies of Dr Metcalf's group have been most informative and, as she will know, I made good use of them in my monograph in support of the concept of the menstruation factor. However, I would criticize them in one respect; they did not attempt to assess the level of pain or menorrhagic symptoms that the women had experienced before surgery. In a recent study from our group, also in press (Bancroft & Rennie, 1995), we have demonstrated a strong relationship between premenstrual pain, particularly pain starting in the premenstruum, and perimenstrual depressed

mood. The value of studies of hysterectomy in the future will be much enhanced if careful attention is paid to the pattern of perimenstrual pain and heavy bleeding, and their relationship with mood changes, that occurred preoperatively. If pain proves to be an important factor in the cause of perimenstrual depression, then we would expect improvement in mood following hysterectomy to be particularly marked in those women who experienced severe pain, premenstrually as well as menstrually, before surgery.

In the meantime, Dr Metcalf is surely premature in suggesting that hysterectomy (presumably she means without oophorectomy) may be the treatment of choice for many women with PMS. If she confines her attention to PMS sufferers who also experience severe pain or very heavy bleeding, then hysterectomy may eventually be proved to be a useful method of dealing not only with the pain and bleeding but also the mood change. More research is needed.

REFERENCE

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