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Premenstrual tension syndrome

There is nothing pleasant about menstruation. At best it is a physiological inconvenience; at worst it contributes to chronic ill health through menorrhagia and dysmenorrhoea, and there are other associations. Frank[1] and more recently Dalton[2] have described a premenstrual tension syndrome with a wide range of physical and psychological symptoms. Clare[3] has suggested that the syndrome is not a single entity but that various "symptom profiles" occur in different combinations, some responding to one treatment and some to another. Whatever the symptoms there is a common, predictable relationship to menstruation, and this suggests a hormonal basis.

Frank,[1] one of the first to link premenstrual symptoms with the second half of the menstrual cycle, postulated an imbalance between the ovarian steroids oestradiol and progesterone. Others[4–6] have supported this view, but evidence of progesterone deficiency in the second half of the cycle has been based largely on clinical observations,[7] and only recently have radioimmunoassay techniques brought a new accuracy to the measurement of steroid hormones. A group working at St Thomas's Hospital showed a lack of progesterone in the second half of the cycle in 30% of women suffering from severe symptoms when compared with normal controls.[8 9] Similar results have been reported elsewhere,[10] but what of the 70% of women with normal progesterone values? Some may have raised oestradiol concentrations, but there is no evidence that women with prolonged or high oestrogen activity (such as occurs in metropathia haemorrhagica) suffer unduly from premenstrual tension.[7] More likely is a relatively minor imbalance in hormones including not only oestradiol and progesterone but also aldosterone and prolactin. The results so far reported have been conflicting. Brush's[8] work on plasma aldosterone concentrations in patients suffering from premenstrual tension showed no significant abnormalities. Raised prolactin concentrations have been claimed to account for at least some of the wide range of premenstrual symptoms,[11–13] but other investigators have failed to confirm any rise in prolactin concentrations in patients with premenstrual tension.[14 15]

Even the widely held belief that women—and especially those with symptoms of premenstrual tension—gain weight premenstrually has been challenged. In a study of 20 patients with severe premenstrual symptoms and 20 controls without symptoms Andersch *et al*[16] found that in neither group were there significant changes in water or body weight in the premenstrual period. Possibly the symptoms of premenstrual tension may be due to a redistribution of fluid—an increased flow from the intravascular to the extravascular compartment.[17] Certain tissues more than others may be sensitive to very slight alterations in their fluid environment, but we cannot yet monitor precisely the movement of water between intracellular and extracellular spaces.[16]

The continuing uncertainty over the cause of premenstrual tension is reflected in the many treatments offered. Dalton[2] claimed success with natural progesterone rather than synthetic progestogens, but the natural hormone is expensive and difficult to administer long term.[18] Taylor[7] identified a group of patients with premenstrual symptoms who had low progesterone values in the luteal phase and treated them with several progestational agents active by mouth; the most successful was dydrogesterone, which relieved many, though not all, symptoms in 70% of patients. Kerr[18] used pyridoxine in 70 patients with apparently normal progesterone or prolactin concentrations, with real benefit to between 50% and 60% of the women, especially in relieving premenstrual headache. Some success has been claimed for bromocriptine,[12] a drug which inhibits the release of prolactin, but Andersch *et al*[16] were unable to show that bromocriptine had any effect on weight or body water in their control patients or in those suffering from severe premenstrual symptoms; in the same two groups of patients a diuretic (bumetanide) was similarly ineffective.

There is no evidence, indeed, that one treatment is more effective than the others. What we need is a more precise definition of the syndrome with well-planned, carefully controlled trials of various treatments—including simple reassurance, mild sedation, or tranquillisers. Whatever else we offer these patients they are likely to appreciate an understanding and sympathetic approach to their problem, and this is where husbands, relatives, and friends as well as doctors can be of considerable help.

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Hazards of fibreoptic bronchoscopy

The reported complications of fibreoptic bronchoscopy are legion, but fortunately most of them are trivial, preventable, or readily amenable to treatment. The minor complications include epistaxis (if the conventional nasal route is used), transient laryngospasm, aphonia, vasovagal reactions, fever, minor cardiac dysrhythmias, and psychotic reactions.[1 2] Rarely, lignocaine (the drug generally used for local surface anaesthesia) produces toxic effects, particularly if precautions are not taken to limit its total dosage. General anaesthesia, too, has potential hazards. Respiratory infection, including cross-