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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-

infection from inadequate sterilisation of the instrument, may follow fibreoptic bronchoscopy, but it has serious consequences only occasionally,[3 4] and usually responds promptly to treatment with antibiotics.

If the purpose of the examination is merely to inspect an intrabronchial lesion and take a biopsy specimen from it the risk of the procedure is very small indeed—so long as the patient's respiratory function is adequate, too large a dose of opiate is not used for sedation, and the operator is not foolish enough to occlude a tracheal stenosis with the tip of the bronchoscope. The fibreoptic bronchoscope (unlike the rigid bronchoscope, which is an open tube) acts as a space-occupying lesion within the main airway and impairs respiratory function both in patients with obstructive airways disease and in normal persons.[5 6] The partial pressure of oxygen in arterial blood (PaO_2) falls, but the changes are usually small and are reversed soon after the bronchoscope is withdrawn.[6] Nevertheless, a protracted examination by an inexperienced operator in a patient with severe airways obstruction—particularly if heavy sedation has been given—could well produce a critical reduction in PaO_2 to a level at which it might precipitate respiratory and cardiac arrest. Such events are known to be rare in specialised units where many examinations are performed and serious complications are fully recorded,[1 2 7] but they may be less uncommon elsewhere—and remain unreported—when the procedure is undertaken by "occasional" bronchoscopists. The rule should be that before any patient with exertional dyspnoea is submitted to fibreoptic bronchoscopy under local or general anaesthesia his respiratory function must be assessed by simple measurements of ventilatory capacity and arterial blood gas studies. Only then is it possible to weigh the risk of the procedure against the benefit the patient is likely to derive from it and to decide whether oxygen should be administered during the examination.

Two further serious hazards are pneumothorax (from penetration of the visceral pleura by biopsy forceps) and haemorrhage from a bronchial artery. Both are inherent in the technique of transbronchial lung biopsy for either localised or diffuse pulmonary lesions. In skilled hands the risks of this procedure are slight and are usually acceptable, because they are less than those of percutaneous needle biopsy and open thoracotomy. In one recent survey[2] the incidence of pneumothorax was reported to be 5%. This is a low figure, and the complication, if quickly recognised and treated, should not pose any major problems; but nevertheless to take biopsy specimens from both lungs at the same session in patients with diffuse pulmonary disease seems unwise. There is a strong case for performing every transbronchial lung biopsy under radioscopic control (which is, of course, essential in the case of localised lesions) because this must reduce the risk of penetrating the visceral pleura with biopsy forceps. As a further prudent precaution a chest radiograph should be taken before the patient leaves the *x*-ray department at the end of the procedure, so that pneumothorax can be detected without delay and an intercostal tube inserted if the pneumothorax is large or is causing any respiratory discomfort.

Haemorrhage of over 50 ml is uncommon during transbronchial lung biopsy[1 2 8] (and is indeed hardly mentioned in many reports), but the very nature of the procedure requires transection of the wall of a small bronchus. The neighbouring bronchial artery is unlikely to escape damage, so that some bleeding is inevitable, and, though it usually abates within a few minutes, few bronchoscopists will have been spared the uneasy experience of a total loss of vision when the distal lens of the bronchoscope is suddenly and completely obscured by blood which cannot be cleared by suction through the narrow instrument channel. If bleeding of this degree continues the patient may be in serious danger, and a rigid bronchoscope or an endotracheal tube should always be available so that large accumulations of blood can be removed quickly by suction through a wide-bore aspirating tube or catheter. Facilities for cardiac resuscitation should also be at hand.

Fibreoptic bronchoscopy has much to offer in the diagnosis of bronchial and pulmonary disease, particularly tumours in the segmental divisions of the upper lobe bronchi, peripherally situated lesions, and diffuse pulmonary abnormalities. Published reports suggest that it has a very low morbidity and mortality, but this may not always be the case if proper precautions are not strictly observed—as indeed they should be in every invasive investigative procedure.

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Typhoid fever

In Britain typhoid fever frequently produces the same reaction from the press as much more serious infections such as smallpox and Lassa fever. This is unjustified. The mortality rate from major smallpox and the viral haemorrhagic fevers may be as high as 30%, and there is no specific treatment available for these virus infections; in contrast, typhoid fever responds to several antibiotics, and, provided the diagnosis is made in time, no patient should die from this disease. Further, whereas smallpox is readily transmitted by the airborne route, direct person-to-person spread of typhoid rarely occurs.

Notifications of typhoid fever in Britain are rising annually, and every year there are one or two deaths. There were 199 notified cases of typhoid fever in 1977, and the number is likely to be considerably higher for 1978. In the past decade almost all the typhoid seen here originated in Asia, but in recent months the disease has again been seen in holiday-makers recently returned from Spain.

Early diagnosis is vital in typhoid fever; delay in starting treatment may result in complications such as perforation of, or haemorrhage from, the small bowel, which may be fatal. The diagnosis of typhoid may be delayed by the blind use of an antibiotic such as ampicillin, amoxycillin, or co-trimoxazole for treating an undiagnosed febrile illness. Recent reports from Britain have shown that delays of this kind may be as long as four weeks.[1 2]

The early symptoms of typhoid fever are non-specific, being related to the septicaemic phase of the illness. Sustained high fever is common, though swinging fever may occur; repeated rigors are less frequent than in other septicaemic illnesses. Headache is almost invariable at some stage. During the first week the spleen may be palpable, but apart from fever there may be no abnormal physical sign. That classic sign of typhoid fever, "rose spots," is frequently absent.[1–3] The rash is pink and may not be noticed on a pigmented skin. As the disease progresses further symptoms and signs develop; multiple