

---

## LETTERS

---

### Premenstrual syndrome

Sir,

I read with interest the letters by Drs Simpson and Dalton (January *Journal*, p.41) in response to my editorial on premenstrual syndrome (PMS) (October *Journal*, p.533). The discerning reader will have noted that in themselves they illustrate one of the main themes of this condition: disagreement among those who write about it. Dr Simpson feels that the diagnosis is made very simply and has no time for a recording of day-to-day experiences, while Dr Dalton considers that such recording is the very cornerstone of the diagnosis.

To take Dr Simpson's letter first, this is, unfortunately, full of didactic statements, none of which is supported by a formal reference. He describes PMS as a straightforward progesterone deficiency disease and seems to regard it as akin to, say, hypothyroidism. I think we may take it that if the situation were so simple as that there would no longer be controversy about the nature of the condition or its treatment. He goes on to assert that about 95 per cent of general practitioners have little knowledge of PMS and implies that most of us still tell women to pull themselves together when consulted about this problem. I do not recall meeting a general practitioner who has this attitude, so perhaps Dr Simpson could let us have some supporting facts.

Dr Dalton's letter is, of course, a weighty criticism from a doctor who has published very extensively on this subject. The difficulty is that so many women who have premenstrual symptoms either do not have a dramatic postmenstrual relief or suffer different symptoms from month to month. While Dr Dalton and Dr Simpson may claim that this excludes such women from the diagnosis of PMS, in practice this is not really the point at issue. The question is how best the general practitioner can help women who consult about symptoms which are present, or exacerbated, premenstrually. This problem is among those increasingly recognized by a better informed public, who are not themselves too concerned whether they conform strictly to the definition of 1953.

The editorial and Professor Clare's monograph on which it is based do not deal in any detail with treatment. Nowhere is it denied that there are patients who may benefit from progesterone therapy. However, Dr Dalton herself says at the start of the chapter on treatment in her book *Premenstrual syndrome and progesterone therapy*:<sup>1</sup> 'Not all patients with PMS require progesterone treatment, which is expensive and there is no justification in treating those whose symptoms do not warrant it.' I therefore stick to the contention of the editorial that for most women who consult a general practitioner in an everyday surgery (as opposed to attending a world expert or a clinic for those with especially severe problems) the woman and the doctor are most likely to be served by a careful assessment of symptoms, attention to other problems which may have affected the decisions to consult and an acknowledgement both of the woman's distress and her right to the final decision as to whether she wants treatment at all.

N.T.A. OSWALD

East Barnwell Health Centre  
Ditton Lane  
Cambridge CB5 8SP

### Reference

Dalton K. *Premenstrual syndrome and progesterone therapy*. London: Heinemann Medical Books, 1984.

### Familial hypercholesterolaemia

Sir,

Inserted with the November issue of the *Journal* was a booklet entitled *Familial hypercholesterolaemia — notes for general practitioners*.<sup>1</sup>

We read it with interest and were dismayed to find that only one original source was quoted and that this was unreferenced. Presumably the authors thought it proper to make dogmatic statements without original references, the assumption being that general practitioners are unwilling or unable to assess evidence critically.

The authors base their case for treatment on the Lipid Research Clinics trial<sup>2</sup> and point out that the active treatment group had a lower rate of cardiovascular events including deaths from coronary heart disease. They omit to mention that the authors state: 'All cause mortality was only slightly, and not significantly, reduced in the cholestyramine group' (71 deaths in controls, 68 deaths in treated men.)<sup>3</sup> This inconvenient finding was ascribed to chance.

Curiously, other studies which have attempted to lower cholesterol have had no effect on total mortality. This includes the WHO clofibrate trial<sup>4</sup> and the multiple risk factor intervention trial<sup>5</sup> in which all cause mortality was higher in the intervention groups. It seems that lower levels of cholesterol are associated with decreased deaths from coronary heart disease but with increased deaths from other causes, especially cancer.<sup>5,6</sup>

This pamphlet is in unhappy contrast with Clifford Kay's thoughtful and well referenced review which you published in the same issue of the *Journal*.<sup>6</sup>

JAMES S. McCORMICK  
PETR SKRABANEK

Department of Community Health  
University of Dublin  
196 Pearse Street  
Dublin 2  
Ireland

### References

1. Familial Hypercholesterolaemia Association. *Familial hypercholesterolaemia - notes for general practitioners*. London: FHA, 1984.
2. Rifkind BM. Lipid Research Clinics coronary primary prevention trial: results and implications. *Am J Cardiol* 1983; **54**: 30c-34c.
3. The Lipid Research Clinics. Coronary primary prevention trial results. *JAMA* 1984; **25**: 351-364.
4. Committee of Principal Investigators. WHO cooperative trial on primary prevention of ischaemic heart disease with clofibrate to lower serum cholesterol: final mortality follow-up. *Lancet* 1984; **2**: 600-604.
5. Rose G, Shipley MJ. Plasma lipids and mortality: a source of error. *Lancet* 1980; **1**: 523-526.
6. Kay CR. Latest views on pill prescribing. *J R Coll Gen Pract* 1984; **34**: 611-613.