is therefore the normal position of choice; a pillow placed under the patient's buttocks gives the surgeon maximum room to manoeuvre without his being hampered by the edge of the couch. Under anaesthetic the left lateral position remains the position of choice unless sigmoidoscopy precedes a minor anal procedure about to be performed in the lithotomy position.

No mention is made of inspection of the anal canal prior to instrumentation. This is particularly important in relation to the diagnosis of a fissure, as any subsequent examination in these patients will be extremely uncomfortable and is better left until the patient is admitted for definitive treatment under anaesthetic. Sigmoidoscopy should always be performed prior to proctoscopy; this is stated at the end of the article but your title and layout suggest that proctoscopy comes before sigmoidoscopy.

The choice of instrument is obviously the prerogative of the surgeon but in a busy rectal clinic the standard stainless steel instrument is probably more practical than the disposable instrument; a fiberoptic light source is essential.

The presence of blood in the lumen is very important; but it should be stressed that blood seen on the way up is more important than blood seen on the way down, which may be secondary to instrument trauma.

I feel that it should also have been stressed that a negative digital examination merely means that nothing has been felt, not that there is no lesion; and it is also important to point out that sigmoidoscopy should always be performed prior to requesting a barium enema. Hopefully this sigmoidoscopy will be to 25 cm—that is, past the rectosigmoid junction. Failure to visualise the lower sigmoid will require a sigmoidoscopy under anaesthetic, or flexible sigmoidoscopy, if the barium enema is negative and symptoms are suggestive of a lesion in the lower sigmoid colon. The rectosigmoid junction is a blind area to the radiologists and tumours will be missed by the surgeon if he does not visualise the sigmoid colon between 15 and 25 cm from the anal margin.

ROGER GRACE

Royal Hospital,
Wolverhampton WV2 1BT

SIR,—With respect, the surgical instrument manufacturers have always considered a trocar to be a sharp pointed instrument for piercing a cavity wall in paracentesis.

The trade description of the component used for introducing a proctoscope tube into the anal canal (9 August, p 437) is surely the obturator.

THOMAS J HUGHES

Seward Surgical,
London SE1 9UG

Sudden infant death syndrome and ventilatory control

SIR,—The hypothesis that impaired chemoreceptor ventilatory drive is partly responsible for the sudden infant death syndrome, as outlined in your leading article (2 August, p 337), may be supported by two further observations.

Firstly, the common anaemia of infancy reaches its nadir between the second and fourth month of life,¹ when the majority of sudden infant deaths occur.² Haemoglobin accounts for only a third of the buffering

power of the blood, but is of great importance in disturbance of acid-base balance of respiratory cause.³ This fall in haemoglobin tends to be more profound where prematurity and adverse social circumstances exist, factors which are also associated with the sudden infant death syndrome.^{2,4}

Secondly, metabolic acidosis is recognised in premature infants and has been found to occur in apparently healthy full-term infants.⁵ It is more common among bottle-fed infants,⁵ as is the sudden infant death syndrome.²

Infants with impaired chemoreceptor ventilatory drive are prone to develop hypoxia and hypercapnia. If these infants also became anaemic and acidotic, they would have little reserve to withstand a combined respiratory and metabolic acidosis in the course of a respiratory infection.

Monitoring capillary blood gases and haemoglobin levels in high-risk groups⁴ might prove to be a simpler means of averting some cases of sudden infant death than monitoring ventilation.

ANGELA MOORE

Department of Paediatrics,
New Cross Hospital,
Wolverhampton WV10 0QP

¹ Matoth Y, Zaizov R, Varsano I. *Acta Paediatr Scand* 1971;60:317-23.

² Carpenter RG, Shaddick CW. *Br J Prev Soc Med* 1965;19:1-7.

³ Zilva JF, Pannall PR. In: *Clinical chemistry in diagnosis and treatment*. London: Lloyd-Luke (Medical Books) Ltd 1971:65.

⁴ Carpenter RG, Gardner A, McWeeny PM, et al. *Arch Dis Child* 1977;52:606-12.

⁵ Moore A, Ansell C, Barrie H. *Br Med J* 1977;i:129-31.

Perinatal and neonatal mortality

SIR,—Your leading article welcomed the report of the Parliamentary Social Services Committee on Perinatal and Neonatal Mortality (26 July, p 255). Of particular interest were its views on the thorny problem of the best place for delivery. The committee felt that an unresolvable dilemma existed in that many women still wish to be delivered in maternity units which do not have the facilities to ensure the lower possible perinatal and neonatal mortality. We have been particularly concerned with this dilemma recently in South Teesside and we wish to report our attempts at solving it.

The heavily industrialised and socially deprived South Tees Health District has a population in excess of 300 000. Middlesbrough, administratively the "centre" of the district, is situated geographically in the north-western corner and so there are several substantial pockets of population more than 20 miles away from the hospital centres or, more relevantly perhaps, 90 minutes away by public transport. This combination of unfavourable social and environmental factors has resulted in a delivery rate outside the consultant units of almost 30%. In 1978, out of 4332 deliveries, 1223 (28.2%) occurred in one of these isolated general practitioner units. This contrasts with a rate of 5.9% for England and Wales as a whole. These factors are, not surprisingly, associated with a relatively unfavourable perinatal death rate (19.9 compared with 15.5 per 1000 nationally).

In response to these problems a plan has been evolved, the central feature of which is the gradual concentration of deliveries into the consultant unit. As a first phase in this process the most isolated of these units was closed. The unit was housed in an old converted house, with inadequate facilities and services. It was more than 15 miles from emergency consultant help. Despite the obvious disadvantages it was well loved by some of the community it served and by the GPs and midwives who staffed it. The protests provoked by its closure were of such intensity that reverberations were felt as far away as the Elephant and Castle

and the Minister of State himself came to survey the scene.

It had been recognised that the closure of this unit would cause inconvenience and additional expense to the patients and their relatives. To minimise their effects, a new consultant antenatal clinic was established in the area and the existing clinics in adjacent areas were expanded. By taking the "consultant mountain" to the "patient Mohammed" the patients' travelling time has been greatly reduced and so their acceptance of care and attendances have been increased. There have been other less obvious benefits. Both hospital and district staff have benefited from the closer working relationships established. With this closer co-operation it has been possible to produce a more selective and appropriate early discharge policy after delivery. It is planned to improve further on this policy by establishing postnatal beds in the community hospitals. When these beds are available we hope to operate a "delivery only" policy at the central unit for suitable cases. The patient will then be transferred back to her own community and caring team within a few hours.

We believe that this policy should ensure the maximum acceptance by patients living in these outlying areas of intensive central hospital facilities for the vital hours of labour and delivery, while preserving as much as possible the homely local unit atmosphere for antenatal care. Perhaps these proposals represent one partial solution to the Parliamentary Social Services Committee's "unresolvable dilemma."

RAY GARRY
JOHN ATKINS

Department of Obstetrics and
Gynaecology,
South Tees Health District,
Middlesbrough General Hospital,
Middlesbrough, Cleveland TS5 5AZ

Progesterone, fluid, and electrolytes in premenstrual syndrome

SIR,—We take strong issue with the conclusions drawn from the paper by Mr P M S O'Brien and others (10 May, p 1161) that progesterone deficiency is probably not the cause of premenstrual syndrome and that treatment with progesterone is illogical unless a deficiency is detected.

The subjects in this study consisted of the same population used in a previous investigation,¹ in which spironolactone was compared with placebo. The group consisted of volunteers from the hospital environment, who may have had prior knowledge of symptoms; and the method of assessment is a further problem as only a full Moos menstrual distress questionnaire has been considered as really reliable.² Estimation of premenstrual mood index (PMI) was made when the subjects were on placebo and is a most unsatisfactory way to define groups for further comparison, as inevitably both contain a mixture of "symptomatic" and "asymptomatic" patients. There are two reasons for this: firstly, symptomatic patients may be included in the asymptomatic group because of a placebo effect and, secondly, asymptomatic subjects have a one in four chance of being included in the symptomatic group, since the probability of randomly obtaining two positive PMI scores in two placebo cycles is one-quarter. In addition, the whole validity of the subsequent conclusions is called into question on account of the small and unequal groups of subjects—namely, 18 and 10.

With regard to the progesterone levels, only four estimations were made—on days 4, 12, 18, and 24 of the menstrual cycle—which obviously bear no relation to ovulation or menstruation unless every subject had an identical 28-day cycle. Two blood levels, therefore, at some stage of the luteal phase is scant evidence on which to base a strong claim. Furthermore, the authors have taken the individual

subjects' figures and tried to represent them graphically, purporting to present a degree of precision that does not exist. The points on the graph in both groups give the appearance of representing mean concentrations on most days of the cycle in all subjects, whereas one point may represent the progesterone level in only one subject, and be joined to that of a totally different subject on the next day. The findings that the authors considered to be most significant, between days 10 and 12 premenstrually, may represent only single readings in three subjects. Additionally, the graph shows the high level of progesterone in symptomatic subjects compared with the asymptomatic group during the immediate three premenstrual days, whereas the table indicates the reverse.

In contrast to these authors, at least eight publications, including those of Bäckström,^{3,4} have discussed an imbalance between oestradiol and progesterone in the second half of the cycle or reported an absolute lack of progesterone in a much greater number of subjects. Mr O'Brien and his colleagues do not report oestrogen levels in their paper, and so we do not know whether a relative deficiency of progesterone actually exists. It has been further suggested⁵ that the obvious cause of premenstrual syndrome is a change in plasma hormone values from the time of ovulation. Changes in progesterone levels from high after ovulation to low premenstrually are certainly claimed by the authors, the high levels preceding changes in mood; could it be, therefore, that administration of progesterone from around the time of ovulation to menstruation would flatten this variation and relieve symptoms?

Irrespective of the aetiology of premenstrual syndrome the most effective therapy should be employed. Although Sampson's study² of progesterone versus placebo showed no difference, a suppository or pessary was used, the treatment period was short, and the number of patients was small. Perhaps the most work and the best results to date have been reported for dydrogesterone,⁶⁻¹⁰ which is orally active—although there is still need for further study and much work is under way.

A J MARTIN
L J DOWNEY

Duphar Laboratories Limited,
Southampton SO3 3JD

¹ O'Brien PMS, Craven D, Selby C, Symonds EM. *Br J Obstet Gynaecol* 1979;**86**:142-7.

² Sampson GA, Jenner FA. *Br J Psychiatr* 1977;**130**: 265-71.

³ Bäckström T, Cartensen H, Sodergard R. *J Steroid Biochem* 1976;**7**:473-6.

⁴ Bäckström T, Cartensen H. *J Steroid Biochem* 1974;**5**:257-60.

⁵ Studd J. *Br Med J* 1979;**ii**:410.

⁶ Taylor RW. *Curr Med Res Opin* 1977;**4**, suppl 4: 35-40.

⁷ Strecker JR. *Fortsch Med* 1980;**98**:145-7.

⁸ Romanini C, Liverani A, Arduini D, Lafuente G, Manfredi L. *Ann Oset Ginecol Med Perinat* 1979; **100**:250-65.

⁹ Taylor RW, James CE. *Curr Med Res Opin* 1979;**6**, suppl 5:46-51.

¹⁰ Day JB. *Curr Med Res Opin* 1979;**6**, suppl 5:40-5.

*We sent this letter to the authors, and Mr O'Brien's reply is printed below.—ED, *BMJ*.

SIR,—Our recent article appears to have aroused a mixture of predictable reactions, many of which contradict each other. Although the study shows subjects which have a measurable increase in the classical symptoms of the syndrome, specifically during the premenstrual phase, Dr Katharina Dalton suggests that they are not adequately defined. In her definition (5 July, p 61) she states that in premenstrual syndrome "symptoms should recur regularly at the same phase of each

menstrual cycle, followed by a symptom-free phase in each cycle"—a definition which does not even require that symptoms occur premenstrually. In her previous writings¹ she has included women whose symptoms occur during ovulation and menstruation and even implied that those who are postmenopausal may suffer.

While Drs Martin and Downey (above) recognise that I have defined the subjects using the premenstrual mood index (PMI) this is not to their satisfaction. The "symptomatic" group were initially self-referred for treatment and subsequently volunteered for the study. The PMI² was used as a scientific method for defining the groups and excluding women without significant premenstrual syndrome—that is, those with so-called "menstrual distress." Even so, the serum progesterone values for the same subjects when self-defined differ only marginally from those reported in the paper (that is, defined by the PMI). In symptomatic subjects (n=20) the mean ($\pm$ SE) serum progesterone was 22.3 ± 3.9 nmol/l (69.7 ± 12.2 ng/ml) whereas in control subjects (n=8) it was 8.9 ± 2.7 nmol/l (27.8 ± 8.4 ng/ml). This was again statistically significant ($p < 0.025$).

Although Drs Martin and Downey point out quite rightly that blood samples in each phase of the cycle cannot be related to ovulation, this has been overcome by retrospectively redating the values from the onset of the next menstrual cycle. These were represented graphically and again the difference in progesterone values was shown. We were, however, careful to point out that daily samples for each patient would be necessary to confirm the finding. Not surprisingly, the inclusion of menstrual cycles of normal duration meant that the majority of day 18 samples fell in the important days of the study.

Dr Martin's argument is inconsistent. He questions the validity of the findings because of the small unequal groups and the omission of the Moos menstrual distress questionnaire for symptom measurement. At the same time he attempts to support his argument by citing several studies with unequal groups of smaller size,³ which did not use the Moos menstrual distress questionnaire and even failed to define the syndrome. These studies concluded that symptoms correlated more closely with oestrogen than with oestrogen:progesterone ratio or progesterone deficiency. Moreover they looked only at the premenstrual six days and therefore the changes follow the onset of symptoms in the majority of patients.

The most surprising fact is that these letters quote results of studies^{4,5} which were not double blind and in some cases not even controlled by placebo therapy—a major omission since there is a well-established placebo effect in this syndrome, frequently reported to be greater than 40%.⁶

P M S O'BRIEN

University Department of Obstetrics
and Gynaecology,
City Hospital,
Nottingham NG5 1PB

¹ Dalton K. *The premenstrual syndrome and progesterone therapy*. London: Heinemann Medical Books, 1977:61.

² O'Brien PMS. *Endocrine changes in the premenstrual syndrome*. MD thesis: 1979.

³ Bäckström T, Cartensen H. *J Steroid Biochem* 1974;**5**:257-60.

⁴ Taylor RW. *Curr Med Res Opin* 1977; **4**, suppl 4: 35-40.

⁵ Greene R, Dalton K. *Br Med J* 1953;**ii**:1007-14.

⁶ Day JC. *Curr Med Res Opin* 1979;**6**, suppl 5: 40-5.

Symptoms in middle life and the menopause

SIR,—While one admires the techniques employed by Dr G T Bungay and others and reported in their recent article (19 July, p 181) it is impossible not to be disappointed by their neglect of basic features of their subject matter. In this they are unfortunately not alone, for most studies of menopausal symptoms repeat the same mistakes.

Symptoms are by definition the complaints of the patient. They are means of communication by which she secures the attention of her doctor, asks for his help, and explains her plight to him. They are not data. They are not a scientific report on unpleasant sensations caused by physical factors. In acute conditions such sensations are likely to overshadow everything else: the patient's complaints may then be clinically very informative. Not so in subacute or chronic illness. The patient then often utilises bodily sensations only to communicate her discomfort or illustrate her malaise. Moreover she always does so with a changing emphasis, which depends on her emotional state, her social position, and her culture. Studies which investigate symptoms of this type as if they were data are sure to produce distorted results.¹

Symptoms vary with the situational factors obtaining at the meeting between patient and doctor.^{2,3} They depend on the relationship between them, different symptoms being presented by the same patient, in the same illness, to different doctors. Symptoms always have, moreover, an important non-verbal aspect. This may, in fact, often be much more significant than their verbal component. Medical practitioners have always known it; they have always recognised that what the patient does not verbally say, is even not completely conscious of, may be of greater importance than what she says. Questionnaires can obviously never probe this aspect of semeiology, or be utilised to study the totality of the patient's symptoms.

The position in the related field of evaluation of the reactions to complex stimuli may therefore be of interest. USA advertising firms are, for instance, now increasingly turning away from questionnaires as a tool of market research. Instead they investigate the impact of their advertisements by examining the encephalograms of people who watch television advertising under conditions similar to those obtaining in their homes. No verbal comment is sought. Researchers in medicine, however, still place the same reliance on symptoms as they did when Cullen devised his nosology.⁴ Compounding misunderstanding, they pluck them out of context, in the medical interview, in repeated attempts at analysis by impersonal questionnaires, treating them as if they were census data.

What do these studies prove? At most they show that women and men in Oxford, or among similar populations, have a cultural pool of verbal symptoms which they utilise in middle life—that Western women in the climacteric pay special attention to vasomotor sensations and utilise them, when disturbed, to express their dissatisfaction. It may even be justifiable to conclude that this pool of verbal symptoms and their non-verbal counterparts provides the material from which a "folkloric expression of the menopause" is formed. This may then be presented as a syndrome by women who are unable to cope with social stress at this time.⁵ Had the study been carried in another country the results would have been very different. Not only do social stresses vary in different societies, but modes of communication are different in different cultures. So are symptoms and illnesses.⁶

As the authors remark, their study provides no "evidence concerning the effectiveness of hormone treatment." Evaluation of improvement in symptoms, in the context of the medical interview, by the same therapist, is, however, as amply proved by generations of doctors, a reliable method of assessing the treatment of illness. This, needless to say, can never be unifactorial. It should always include both pharmacological agents aimed at