illness and to answer the initial anxious questions as well as they can at this early stage of investigation. All those small but essential details are the houseman's lot. House surgeons assist at their patients' operations and perform a few operations under supervision, such as appendicectomies and stripping varicose veins. They also have their own operating list for minor operations such as the removal of lumps in the skin.

The senior house officer, registrar, or senior registrar is soon on the scene to check the houseman's findings, to agree initial treatment, to work out a strategy of investigation before the consultant's next visit, or to phone him to discuss the situation when matters cannot or should not wait.

Housemen work long hours, but shorter than they did; soon they will not be permitted to be on first or second call for a total

of more than 80 hours a week. Shifts are not worked as they are in many countries. The week is five days long for a start and is likely to include one night first on call for emergency admissions and one second on call. When first on call the houseman may be up for most of the night, but this gives no dispensation from the next day's work. One half day is supposed to be free for study or relaxation. One weekend in three the houseman lives in on first or second call. Two weeks' holiday are allowed in each six month post.

When two house jobs have been completed satisfactorily, as they almost invariably are, the first period of a doctor's training is complete and he or she can apply for registration with the General Medical Council. Now the first career decisions must be made.

Personal Paper

Polyhydramnios: threat of a handicapped child

ANNE WALLING

Consideration of the patient's emotional needs has been a feature of many of the leading papers on antenatal screening for neural tube defects, but few accounts have been written by patients. I do not pretend that my experience can be regarded as typical, and, since it happened in 1977, it is in many respects already out of date, but it may be useful and interesting to those concerned with such patients.

About 24 weeks into my second pregnancy I developed polyhydramnios. Neither my husband nor I (both doctors) was willing to recognise that anything was wrong until at 27 weeks our general practitioner, who was also a partner and friend, pointed out that I was 36 weeks' size and he intended immediately to convert "shared care" into "consultant only." The appointment with the consultant was arranged for the next day. Examination was followed by ultrasound scan, x ray examination, and a long and frank discussion with the obstetrician and radiologist. Polyhydramnios had always been a favourite question in our medical school, and I found to my horror that I could recite a list of causes and associated conditions under three headings—maternal, fetal, and miscellaneous. This was indeed a case of medical nemesis.

Polyhydramnios in a west coast Scot alerted everyone to neural tube defect. I particularly remember the kindness of the radiologist who carefully discussed the x ray film and frankly concluded that it looked normal but that a lumbar spina bifida could not be completely excluded. There seemed to be little to do but follow the ubiquitous antenatal regimens of rest and more frequent attendances at the clinic. Just as we were leaving, the consultant suggested sending blood to a laboratory in London

where he knew there was an experimental test for neural tube defect. It seemed a wonderful opportunity to put our minds at rest. I do not recall contemplating the possibility that the test could be anything but beneficial.

Discomfort for the patient

One feature that all my book learning on polyhydramnios had not mentioned was how uncomfortable it is for the patient. To reach term size 10 weeks early is very wearing. My girth was 45 in (115 cm) at 26 weeks, and as I am only 1.57 m (5 feet 2 in) tall I must have looked like a version of the Michelin man. Apart from the expected discomfort of legs, abdomen, and pelvis, it was difficult to sleep owing to pressure on the ribs. My most worrying symptoms, however, were those of hypotension. Lying down or even flopping into a chair brought on nausea, dizziness, and a feeling which I can describe only as a spinning into a vortex. Loss of consciousness quickly followed unless I rolled over on to one side. These episodes at their worst occurred several times daily and sometimes had no obvious precipitant. My practically minded 4 year old son calmly went for a neighbour whenever mummy fainted. Alone at home with a young child and having to drive to reach shops, etc, made the situation potentially dangerous. It was a great relief to be admitted to hospital and to give up the unequal struggle of "resting at home."

In the meantime, the first blood α -fetoprotein concentration had been reported as, "Almost undetectable levels—is patient pregnant?" a question that was really quite funny in view of my size. Nothing daunted, another sample was sent. This time the result somehow got lost and only after telephone calls were we told, "Just outside upper limits of normal". We were left with the worst possible situation of a substantial threat of abnormality at more than 30 weeks' gestation.

Surprisingly, our reaction was to stop trying to find out. We thought there was no alternative but to proceed with the pregnancy and to make the best of any handicap that resulted. That at least was my publicly expressed opinion. Privately, I remem-

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bered a patient with polyhydramnios who had an intrauterine death, and I was convinced that this would be the happiest outcome for all concerned.

Waiting in hospital

The weeks spent in hospital were uncomfortable, but instructive. The antenatal ward was situated between the labour rooms and the paediatric ward in an E shaped building. Thus on long, warm, sleepless nights we could listen to grunts, crashes, and occasional screams from the labour suite or a child in the paediatrics ward crying for his mother. As most of us had left children at home, these noises were particularly distressing. One child with whooping cough seemed to cough or cry all night.

Being a doctor patient made for some awkwardness with nurses and medical staff. I was very aware of a need not to make a fuss. Perhaps this is why I never asked to speak to the paediatricians before delivery, although I desperately wanted to know what would happen should the baby have spina bifida. For the other patients I was an instant second opinion. It was a revelation to hear the patient's versions of "what the doctor said." All in all, patients, staff, and visitors behaved exactly as described in my textbooks of medical sociology.

I went into labour at 37 weeks and delivered a normal boy weighing 3175 g—plus an amazing amount of fluid. The last complication on my long memorised list was our only problem. He inhaled a small amount of fluid but recovered quickly in the special care unit. He is now a boisterous, intelligent young man. We never did find an explanation for the polyhydramnios.

Value of α fetoprotein testing

In retrospect two things stand out. Firstly, there was the distress to my family and friends in coping with the threat of a

handicapped child. As the patient, I had an obvious role as the focus of attention and care, but it was more difficult for the others, in particular the immediate family. They were obviously under great strain and it hurt to see them trying to overcome their own distress in order to avoid worrying me.

The second aspect was the false positive α fetoprotein result. We had hoped the test would relieve doubt but in fact it compounded and confused the issue. Some writers assume that a false positive result is so devastating to the patient as to be an argument against screening. I would maintain that it is an argument for improving screening, not abandoning it. Our distress was caused by uncertainty and reluctance to discuss with the family what to do for the different potential outcomes. Much of this reticence was due to the special circumstances of both parents being doctors. Since my pregnancy, the many improvements in the technical and organisational aspects of α fetoprotein screening should make false positive results less likely. It is equally important to develop support and follow up for patients with positive results to maximise the benefits of screening. This is particularly vital when screening asymptomatic patients. We already knew there was a risk of handicap because of the polyhydramnios. With a screening programme, the first indication of trouble is a positive blood test result. This offers the family choices and, if they opt to proceed with the pregnancy, the time to prepare mentally and physically for a handicapped child. The alternative is the sudden shock at birth with all the ensuing adjustments and trauma at a time when the family and in particular the mother is least well prepared. Screening for neural tube defects offers a valuable and humane service to families. For the medical profession, there remain two tasks—firstly, to continue to improve the sensitivity and specificity of the tests and, secondly to develop those skills in communication and counselling which will enable the full benefits of the programme to be available to patients.

(Accepted 10 June 1983)

What are the indications for hormone replacement therapy at the menopause? Can it be given routinely and what are the risks of this treatment?

These questions are still causing debate, and the American Medical Association has recently issued guidelines.¹ Of the many symptoms attributed to the menopause, only two are specifically relieved by oestrogen treatment—vasomotor flushes and vulvovaginal atrophy.² Vasomotor symptoms ("hot flushes") affect about 80% of menopausal women,³ though many can cope without treatment. Oestrogens relieve hot flushes in 90% of cases and treatment can usually be tapered off after a year or two.³ Vulvovaginal atrophy (and some bladder symptoms) are reliably relieved by systemic or local oestrogens, which may have to be continued for four to 12 weeks³ and may be required intermittently thereafter. (Local treatment may have systemic effects because oestrogen is absorbed through the skin.) So far as other symptoms such as depression are concerned, oestrogens are no more effective than placebo. "Routine" oestrogen treatment has been suggested because it slows postmenopausal osteoporosis² and may protect against fractures in later life.¹⁻³ Treatment must begin before osteoporosis becomes severe, and after treatment is stopped bone resorption continues at a rapid rate.³ The pathogenesis of postmenopausal osteoporosis is complex,^{2,3} and because oestrogen prophylaxis has to be continued indefinitely it is not generally recommended.^{1,2} There is no conclusive evidence that postmenopausal oestrogens alter the risk of coronary heart disease in either direction.^{2,3} The main risk of hormone replacement therapy, endometrial carcinoma, appears after two to five years' treatment,^{1,2} but the condition is usually diagnosed early (when the prognosis is good¹⁻³) and the risk may be reduced by giving treatment cyclically along with a progestogen.³ Another problem is uterine bleeding, which must always be investigated by curettage.¹ There have been suggestions that 10 years or more of oestrogen-only treatment increase the risk of breast cancer, but these have not been confirmed.³ There is an increased risk of gallbladder disease¹⁻³ but no risk of hypertension except in occasional idiosyncratic

cases.³ The American Medical Association recommends annual checks during treatment, with cervical smears (and perhaps endometrial biopsy), breast examination, and blood pressure recording.¹ Treatment is contraindicated by a history of an oestrogen dependent breast tumour,^{1,3} thromboembolic disease, or acute liver disease.³ Before treatment its benefits and risks must be discussed with the patient, who may then decide that prolonged treatment is unnecessary. —JAMES OWEN DRIFE, senior lecturer in obstetrics and gynaecology, Leicester.

¹ Council on Scientific Affairs. Estrogen replacement in the menopause. *JAMA* 1983;249:359-61.

² Mosher BA, Whelan EM. Postmenopausal estrogen therapy: a review. *Obstet Gynecol Surv* 1981;36:467-75.

³ Hammond CB, Maxson WS. Current status of estrogen therapy for the menopause. *Fertil Steril* 1982;37:5-25.

Is there any evidence that commercially made brown bread causes headaches?

There are a large number of isolated individual reports of adverse reactions, of varying degrees of severity, to individual foods.¹ These reports are difficult to confirm as to do so requires clear identification of the food responsible and recurrence of the reaction on a blind reintroduction of the food to the diet.² There is no evidence to suggest, for example, that the additives permitted in brown bread are associated with any adverse reactions. Clearly, however, there are well documented idiosyncratic reactions to particular foods and in the absence of detailed investigation to prove that the food is involved the only course for the person is to avoid the food in question.—D A T SOUTHGATE, head, nutrition and food quality division, Norwich.

¹ Lessof MH, Wraith DG, Menett TG, Menett J, Buisseret PD. Food allergy and intolerance in 100 patients—local and systemic effects. *Q J Med* 1980;49:259-71.

² Jones VA, McLaughlan P, Shorthouse M, Workman E, Hunter JO. Food intolerance: a major factor in the pathogenesis of irritable bowel syndrome. *Lancet* 1982;iii:1115-7.