this is why it was chapter 19 of 19. He sadly continues the hospital myth of the "five minutes a patient" general practitioner who will try a couple of treatments but without a doubt will have to call in the specialist if it is anything out of the ordinary. To Dr Blau the general practitioner's skill is in choosing the right specialist.

His treatment of premenstrual tension with high dosage pyridoxine will raise a few eyebrows as will his advice for migraine

sufferers whose attacks are induced by sexual intercourse to "become less excited," but there are good points in this book. Dr Blau recognises that patients are responsible people and that they present with "headache" for reasons other than that symptom. Would I recommend this book to my patients? I think not. Why didn't a general practitioner write it?—GEORGE TAYLOR, general practitioner, Newcastle upon Tyne.

Clinical Algorithms

Menorrhagia

MARGARET REES

Heavy periods are a common clinical problem. It has been estimated that the annual consultation rate with general practitioners is 20.4 per 1000 women. The word menorrhagia is derived from the Greek *men* (month) *rhegynai* (to burst forth). Patients complain of increased menstrual loss, requiring more sanitary protection, or the passage of clots. Objective measurement of menstrual flow in representative populations has shown that the mean menstrual loss is about 35 ml during each period and the 90th percentile is 80 ml per period. Menstrual blood loss is therefore considered excessive if it is greater than 80 ml¹; such a loss tends to be associated with the development of iron deficiency anaemia.

Aetiology

Menorrhagia may be due to systemic, local, or iatrogenic disorders. Systemic conditions include endocrine disease (hypothyroidism, anovulation) or disorders of coagulation (haemophilia carriage, von Willebrand's disease). Local conditions include endometrial hyperplasia, endometriosis, pelvic inflammatory disease, and benign (leiomyomas, polyps) and malignant (endometrial, cervical) tumours. Iatrogenic disorders may be due to foreign bodies, such as intrauterine contraceptive devices, or poor control of anticoagulation treatment. In more than half of the women with menorrhagia no pathological cause is found. Excessive menstrual loss has been ascribed to abnormal endometrial concentrations of prostaglandins and plasminogen activator.^{2,3}

Assessment

Initial assessment begins with the medical and gynaecological history. The length and interval of periods and the duration of excessive bleeding should be ascertained. The method of contraception should also be noted since intrauterine contraceptive devices are associated with increased menstrual blood loss. General

examination, including bimanual pelvic and speculum examination, should be performed and a cervical smear obtained. A particular search should be made for polyps protruding through the cervical os and for enlargement or tenderness of the uterus or adnexae. A routine full blood count should be performed, and if there is any suspicion of abnormal coagulation a clotting screen should be obtained. Thyroid function tests should be performed if clinically indicated.

The next step is to examine the endometrial histology. This is mandatory in women over 40 years of age but may be deferred in younger women unless the bleeding is severe, does not respond to treatment, or is associated with intermenstrual bleeding. A sample of endometrium may be obtained by suction curettage in the outpatient department without anaesthesia or by dilatation and curettage with anaesthesia. In specialised centres hysteroscopy may be used to reveal endometrial polyps or submucous leiomyomas, which are sometimes missed at curettage.

Treatment

Treatment may be either medical or surgical. Medical treatment is indicated when there is no obvious pelvic abnormality and the woman wishes to retain her fertility. Surgical treatment may be necessary to deal with pelvic abnormalities such as leiomyomas, chronic pelvic inflammatory disease, or endometriotic masses. Operations should be as conservative as possible in women who wish to remain fertile. Surgical treatment is also indicated when medical treatment has failed.

MEDICAL TREATMENT

Prostaglandin synthetase inhibitors, such as mefenamic acid, and antifibrinolytic agents, both taken during menstruation, reduce menstrual blood loss.^{4,5} Progestogens, such as norethisterone, given during the luteal phase are also used to reduce menstrual bleeding, but the effect has not been documented by measurement of blood loss. Oral contraceptives are particularly useful in women under 35 who also require contraception. In cases of menorrhagia associated with anovulation clomiphene has been used to stimulate ovulation, but there is no objective evidence showing a reduction of menstrual blood loss. It is probably reasonable to reserve clomiphene for those patients who wish to become pregnant. Finally, danazol

Menorrhagia

taken continuously reduces menstrual blood loss and may produce amenorrhoea.⁶ It should always be remembered that menorrhagia tends to cause iron deficiency anaemia, which requires treatment.

SURGICAL TREATMENT

Surgical treatment includes removal of cervical or endometrial polyps, curettage, myomectomy, and, ultimately, hysterectomy. Endometrial polyps and submucous fibroids may be removed under hysteroscopic control in specialised centres. Dilatation and curettage reduces menstrual blood flow in the next period but seems to have no permanent effect on measured menstrual loss. It is best regarded as a diagnostic rather than a therapeutic procedure. Hysterectomy is offered more often to younger women whose families are complete because many are reluctant to take treatment for several years. In some older women prostaglandin inhibitors or antifibrinolytic drugs may be used until the menopause supervenes.

Conclusions

The diagnosis and management of menorrhagia are based on the patient's complaint of excessive menstrual bleeding. Women are unreliable judges of their menstrual blood loss, however, and one study found that only 38% of women with a convincing complaint of menorrhagia had measured losses greater than 80 ml.⁸ Some women are therefore treated for a condition that cannot be shown objectively. It could be argued that objective measurement of

menstrual blood loss should be available routinely to ensure that treatment is restricted to women in whom menstrual bleeding is excessive. Women can reliably detect a change in their menstrual loss, however, and not all whose complaint of menorrhagia is associated with a normal menstrual loss actually welcome this knowledge; many want a return to the loss that they previously regarded as normal.

Assessment should therefore take into account the woman's complaints and expectations. Although mainly determined by the clinical findings, management should also be influenced by the patient's wishes.

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A 30 year old woman takes the progestogen ethynodiol diacetate 500 µg (Femulen) for contraception. She also suffers from premenstrual irritability and depression but does not wish to change to a combined pill. Progesterone deficiency has been postulated as one cause of premenstrual tension, so should progesterone or its derivatives be used to help this patient? Another woman taking a similar dose of the drug is still able to pinpoint ovulation by changes in the mucus present near the introitus. Does this mean that the dose is insufficient and that contraception is inadequate? Would increasing the dose of a progestogen only pill increase its efficacy?

The key to both these questions lies in understanding that there is a whole range of possible responses to the progestogen only pill. In a group of women studied by Landgren and Diczfalussy in 1980, 40% of cycles showed almost no change from normal with apparently normal ovulation, minimal shortening of the luteal phase, and progesterone concentrations within normal limits.¹ In another group (21%) there was a normal follicular phase but pronounced shortening of the luteal phase with lower progesterone concentrations for a shorter time than expected in a normal cycle. In the third group (23%) follicular activity was noted, often with higher peak oestrogen concentrations than usual but no ovulation at all and no progesterone production. Finally, at the limit of the range, 16% of the women had reduced follicular activity, low oestrogen concentrations, no corpus luteum formation, and no endogenous progesterone production. The menstrual cycle in women taking progestogen only pills therefore varies: from normal, unaffected (group one as just described); through the middle two groups who tend to have irregular bleeding; to the fourth group who are amenorrhoeic. More recent studies at the Margaret Pyke Centre and in Dundee (P Howie, personal communication) have been largely confirmatory but suggest that even among the women with regular cycles ovulation may not be fertile. In the middle two groups follicular activity is abnormal, and absent as expected in the fourth group.

Probably (though this is not stated) the first woman is in the first group, seeing reasonably regular menses; and her premenstrual syndrome is presumably caused in the same (unknown) way as the premenstrual syndrome of the normal menstrual cycle (without exogenous progestogen). Although the evidence is against progesterone deficiency as the cause of this syndrome,² some women do respond for unknown reasons to this treatment. Empirically, therefore, the doctor could try the effect of giving two tablets of Femulen daily during the second half of each 28 day cycle of taking the progestogen only pill. Otherwise any of the treatments suggested in the recent algorithm article² could be tried while the woman continues to take one progestogen only pill a day.

The fact that a woman taking such pills can detect slippery "ovulatory" mucus at or near the introitus does not of itself prove that she is ovulating. Possibly she comes from the third group described above with high oestrogens sufficient to overcome the effect of the progestogen only pill on the mucus—yet by definition such women are not ovulating and will later produce no progesterone. Hence one would be concerned only about the contraceptive efficacy if this woman was also experiencing extremely regular periods. Confirmation would be possible by a luteal phase progesterone or ovarian ultrasound; but if in doubt, in my view a trial of two tablets of the same brand of progestogen only pill daily with careful follow up would not be unreasonable if this remained the woman's preferred method. —J GUILLEBAUD, senior lecturer in gynaecology, London.

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It is fashionable to eat certain brassicas raw, usually shredded in salads. What quantities might be expected to exert a clinically significant antithyroid effect?

Many brassicas contain substantial quantities of the glucosinolates that may be converted to substance with thyroid activity by the enzyme myrosinase.¹ Whereas the amounts in a substantial intake of cooked Brussels sprouts (150 g/day) showed no effect on thyroid hormone concentrations the consumption of large amounts of raw brassicas with the myrosinase enzyme still active may possibly affect thyroid function. There is little evidence that any harm has arisen from the consumption of raw brassicas, probably because the amounts consumed are not usually large and because those brassicas customarily eaten raw tend to have lower glucosinolate concentrations than Brussels sprouts.² In the study cited above about 150 g Brussels sprouts were used. These contain somewhere between two and ten times the amount in some other common brassicas such as white cabbage, so intakes of several hundred grams a day would be necessary to provide levels that might give cause for concern. It should be emphasised, however, that there are no direct observations on which to base a more definite answer.—D A T SOUTHGATE, head, nutrition and food quality research, AFRC Institute of Food Research, Norwich.

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