The management of menorrhagia—current concepts: Part I

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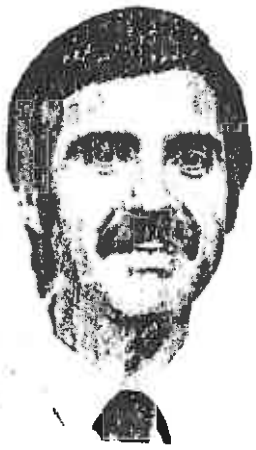
Although modern societies regard menstruation as a normal physiological process, the phenomenon of excessive menstrual bleeding has unfortunately become surrounded by numerous medical myths which have created a limited, almost superstitious approach to management of the patient who complains of this. The innumerable negative diagnostic curettages under general anaesthesia which have been performed to exclude serious pathology (ie endometrial cancer), elucidate the cause of abnormal uterine bleeding and hopefully cure a proportion of cases, bear testimony to this view. Critical assessment suggests that heavy menstrual loss is rarely caused by genital tract cancer, most endometrial examinations are normal and, for the purpose of treatment, after an initial post curettage light loss, the previous menstrual pattern returns. (Nilsson & Rybo, 1971, Haynes, 1979).



Reasons for "Superstitions"

THERE are probably four main reasons why patients complaining of heavy menstrual loss are currently offered a limited approach to management with hysterectomy so often providing an early final solution. These will be discussed under the following headings:—

(i) Cumbersome terminology. (ii) Wide variability in interpretation of dysfunctional uterine bleeding. (iii) Unsubstantiated extrapolation of known facts gained from small groups and (iv) Lack of definite knowledge of the cause of menorrhagia.

Terminology

Because of difficulty in remembering the precise definition of terms such as epimenorrhoea (polymenorrhoea), epimenorrhagia (polymenorrhagia), metrorrhagia (the same as metrostaxis) and impossible ones such as menometrorrhagia, it has become widely accepted that any abnormal and/or excessive uterine bleeding is termed menorrhagia.

Although, through general default a case exists for dispensing with these unmanageable terms it is important to realise that clues to specific aetiologies and hence the direction of management are usually provided in the history. Furthermore, reasons for patient complaint and their expectations from the medical profession will vary depending upon the way in which the abnormal menstrual loss is disturbing them.

This article will deal purely with the problem of amplified menses; that is where the amount and/or duration of blood loss is considered excessive but where the cycle retains its normal rhythm.

Dysfunctional uterine bleeding

One may collect an immense variety of documented interpretations of what is encompassed by the term dysfunctional uterine bleeding. Although this may be as interesting as philately to the ardent collector, it has not provided a stable base for the development of understanding of the subject of abnormal menstrual bleeding.

Two of the shorter and more popular definitions are presented and will exemplify the reason why certain beliefs held today control the management approach.

"Dysfunction l uterine bleeding may be defined as abnormal bleeding from the uterus unassociated with tumour, inflammation or pregnancy". (Novak & Woodruff, 1976)

"Dysfunctional uterine bleeding is defined as abnormal bleeding not due to genital tract pathology or systemic disease and is anovulatory". (Southam & Richart, 1966)

Because of the possibilities implied in the first definition, EUA (examination under anaesthesia) and D & C (dilatation and curettage) has been regarded as necessary to exclude benign and malignant tumours of the uterus, inflammatory processes of the endometrium and pelvis and possible complications of pregnancy which may constitute a remediable cause or at least an explanation for the abnormal bleeding.

From the second definition one is able to attribute other genital tract diseases and even systemic ones, in the causation of abnormal uterine bleeding. Furthermore, if none are disclosed then the management should obviously be directed against the problem of anovulation. This view still has universal support as judged by the most recent edition (1978) of Postgraduate Obstetrics & Gynaecology, edited by Professor Sir J Dewhurst. In this highly esteemed book, Professor Davey states "The present concept is that in the majority of cases of dysfunctional uterine bleeding the essential defect is in the secretion of oestrogen and progesterone by the ovary".

These conclusions are currently begining to be contested.

Unsubstantiated Extrapolations

Sutherland in 1949 and later Kistner (1964) both produced remarkably similar results in analysing curettings from patients with abnormal uterine bleeding where there was no obvious cause. The results are summarised in Figure 1.

Figure 1: Histology of Endometrium in Dysfunctional Uterine Bleeding from two sources.

	Sutherland, A M (1949)	Kistner, R W (1964)
	%	%
Normal	63.5	57.5
Hyperplasia	30.8	30.8
Irregular Ripening	3.0	—
Irregular Shedding	1.5	2.2
Chronic Menstruation	—	7.8
Atrophy	1.2	1.7

The conclusions were that anovulation accounted for a large proportion of these cases as judged by over 30 per cent displaying the pattern of endometrial hyperplasia. Very small numbers showed the features of irregular ripening and/or irregular shedding, as described by McElvey & Samuels (1947) and which are thought to be the result of an inadequately functioning corpus luteum causing irregular ripening or a persisting, but failing, corpus luteum giving rise to irregular shedding. It was suggested that these states were undoubtedly associated with abnormal hormone release from the ovary and furthermore, if more endometrial biopsies were performed during the active bleeding time, many of the normal cases might be seen to belong in this group with corpus luteum defects.

Although these eminent views were generally accepted, possibly because they provided a rational basis for conservative hormonal therapy, they have not been substantiated by recent investigation. Indeed, the selection of patients for the reports in Figure 1 may have been biased towards a hormonal abnormality and the numbers who clearly had menorrhagia without other menstrual abnormality is not known. It is not common experience in general gynaecological clinics today to find 30 per cent of patients with menorrhagia displaying hyperplasia in their endometrial curettings.

Indeed, Nedoss (1971) showed that over 80 per cent of endometrial biopsies performed in the luteal phase displayed the normal secretory pattern. Recent work in Oxford (Haynes, 1979) analysing daily hormonal patterns (gonadotrophins, oestrogen and progesterone) of women whose complaint was purely of menorrhagia, had patterns similar to normal controls and were obviously ovulating without hormonal or other evidence of any functional corpus luteal defect.

It is possible that widespread, long term oral contraceptive usage may have acted prophylactically suppressing anovulatory causes of menorrhagia, but hard data supporting this hypothesis is not available.

Aetiology of menorrhagia

Keeping in mind that this article is concerned with the problem of heavy and/or prolonged menstrual bleeding, the following account takes into consideration recent developments influencing current concepts.

Beginning with the Swedish population studies of Hallberg *et al* reported in 1966 were the first large scale studies attempting to define normality of menstrual loss, based on the blood extraction method of Hallberg and Nilsson (1964). Their results were remarkably similar to a British study undertaken in a Northumbrian mining community in 1971 (Cole *et al*). The mean menstrual blood loss is 35-40 mls. However, the range of blood loss is remarkably wide varying from less than 1 ml. to 280 mls.

Women who complain of excessive blood loss or who develop symptoms secondary to excessive bleeding are usually losing in excess of 80 mls. per cycle. It is important to realise, however, there is no distinction between heavy and light losers (*ie* there are not two groups), but that patients complaining of menorrhagia are at the right-hand end of a curve with positive skew. The loss is greater in those women of larger stature and rises with parity, proportional to baby weights. Heavy losses are more common in the last half of the fifth decade but the influence of age is unclear and probably minimal if parity and pelvic pathology are taken into account. The Northumbrian study clearly showed that there was reduced loss in oral contraceptive users (mean 12.7 mls.) and an increased loss in those using an intrauterine device (mean 56.3 mls. and much wider variation).

(i) Associated Pelvic Pathology

Certain pelvic diseases have traditionally been regarded as causing menorrhagia. Commonly, benign tumours such as fibroids are considered to cause heavier bleeding because of a larger endometrial surface area. However, hysteroscopists have been impressed with the relative thinness of endometrium overlying submucous fibroids (see Figure 2). There is an unpredictable associ-

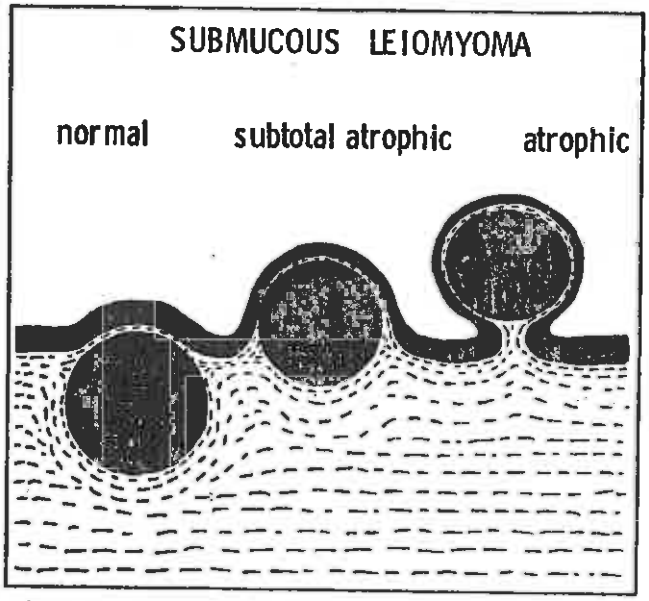


Figure 2. Relationship between submucous leiomyoma and overlying endometrium. Sugimoto, 1978.

ation of menorrhagia with fibroids although it does appear that the more centrally located the tumour in the uterus the more likely is there to be heavy menses. The true mechanism is probably associated with derangement of the venous drainage mechanism known to occur within the fibroid uterus (Farrer-Brown *et al*, 1970). This may also be the basis of menorrhagia associated with adenomyosis and other bulky uterine states.

Polyps within the uterus (benign or malignant) tend to have a rich, fragile, arteriole arrangement which may be traumatised during menstrual contractions and lead to increased blood loss at the time of menses.

Malignancy of the endometrium will rarely be the cause of heavy regular menses. Ulceration of the endometrium causes an irregular pattern of bleeding. It is an uncommon tumour under 45 years of age unless the previous menstrual pattern was one of high or unopposed oestrogen activity such as with Stein-Leventhal syndrome, feminising ovarian tumours and possibly patients given unopposed oestrogen therapy.

Other pelvic conditions such as congenital uterine abnormalities, endometrial infection, chronic pelvic inflammatory disease, endometriosis and chronic pelvic venous congestion may be associated with menorrhagia. The mechanism for these remains somewhat obscure. Possibilities are discussed in a review article by Yovich, 1978.

(ii) Blood Dyscrasias

Certain distinct hereditary haemostatic disorders are invariably associated with menorrhagia (Factors V, VII and X deficiencies, von-Willebrand's disease and certain platelet disorders such as thrombopathic thrombocytopenia and Glanzmann's syndrome). However, the role played by coagulation factors in controlling menstrual blood loss is still not fully understood. There are also some acquired haemostatic disorders which may present or be associated with menorrhagia. Most likely candidate are idiopathic thrombocytopenic purpura, acute leukaemias and aplastic anaemia.

iii) Endocrine Disturbances

Dysfunction of the hypothalamic-pituitary-ovarian axis will produce an influence on the endometrium and lead to defects of the menstrual cycle. The various anomalies have been classified by Vorys *et al* (1975) but, as indicated earlier, there is considerable doubt that heavy and/or prolonged regular menses are often caused by these "dysfunctions". Endometrial hyperplasia and metropathia haemorrhagica are the end result of anovulation if this is associated with high, unopposed oestrogenic stimulation. However, the clinical pattern is usually one of heavy irregular bleeding following a variable time of amenorrhoea and usually succeeding oligomenorrhoeic cycles. From R Schröder (1954), Figure 3 is presented showing the age distribution of endometrial hyperplasia as seen over a lifetime of gynaecological work. These cases constituted 6.6 per cent of all gynaecological pathology material gathered in his time.

Specific endocrine disorders may also cause menorrhagia. Early pituitary tumours, feminising tumours of ovarian or adrenal tissue and oestrogen ingestion may

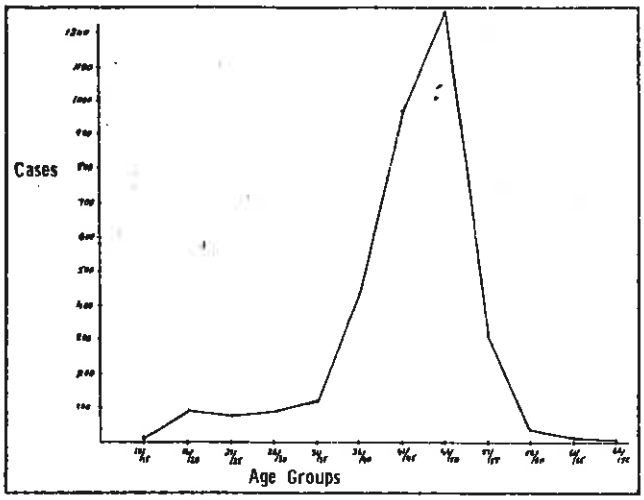


Figure 3. Age distribution of endometrial hyperplasia. From Schröder, 1954.

upset the hypothalamic-pituitary-ovarian axis. Hypothyroidism is commonly associated with menorrhagia, but the mechanism is obscure.

(iv) Aetiology in the Majority

Most patients complaining of heavy periods will not have one of the above causes present. There have been two areas of advance in the past decade which are beginning to shed light on the subject and offer a rational basis for alternative management (*ie* other than D & C, progestagens and hysterectomy). These advances have been the finding of increased endometrial fibrinolytic activity and advances in knowledge concerning prostanoid metabolism in the menstrual cycle. These factors will be examined in part two of this article, which will include the references.