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Office Gynaecology

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The term gynaecology means the treatment of diseases peculiar to the female genitalia, and office gynaecology, of course, refers to the management or treatment of the diseases peculiar to the female genitalia and these diseases are such that one is able to properly manage or to treat them in the office. Besides this, of course, there are certain diagnostic procedures which may be carried out in the office. The same rule applies in the office as in the hospital, namely, before you can properly treat any disease you must firstly arrive at your diagnosis. There are three cardinal essentials for aiming at a proper diagnosis:

(a) *a good clear cut history.*
(b) *a general physical examination.*
(c) *the functional inquiry.*

With these in mind you are prepared to proceed with your diagnostic investigation.

ESSENTIAL OFFICE EQUIPMENT

For a proper and thorough examination of any gynaecological patient you will need at least certain essential articles of equipment, such as: a private and well-lighted room, an examining table with a moderately firm mattress, a goose-neck light or one similar, adequate supply of clean linen, including sheets for draping the patient, a tray on a table beside the examining table and this tray should include the following at least: a bivalve speculum, sponge forceps, gauze or cotton wipes, waste dish, culture tubes, glass slides for smears, sterile applicators for taking smears and a pair of clean gloves.

Special articles of equipment: This would include a biopsy punch (sterilized), an Ayres spatula for taking cervical smears, a microscope and suitable stains, a small incubator is very handy, insufflator for treating trichamona and some special solutions or powders used for specific treatments of trichamona and the yeast fungus, an electric cautery for cervical catarrh cases. Other special items may be added as found necessary.

Your gynaecological patients may be divided into three common groups:

(a) *the young adolescent patients.*
(b) *patients in the child-bearing age.*
(c) *the menopausal and post-menopausal patients.*

Before carrying out a pelvic examination for the younger patients, you must have the patient's mother or guardian present and obtain her consent before doing a pelvic examination. Such a patient may be examined by rectum at first and if necessary the vaginal examination may be done under an anaesthetic. This patient must be 18 years of age before she accepts responsibility herself regarding pelvic examination. The common reasons for the younger patients seeking advice from a gynaecologist are as follows:

(a) *late puberty.*
(b) *irregular periods.*
(c) *epimenorrhea.*
(d) *meno-metrorrhagia.*
(e) *vaginal discharge.*
(f) *lower abdominal pain.*
(g) *enlargement of the lower abdomen.*

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We usually do not worry about late puberty until the girl is 16½ years old, providing she is a reasonably healthy girl. Also the irregular periods need cause no special concern until she is 16½ years old. Meno-metrorrhagia requires a thorough investigation — including a general physical examination, a pelvic examination and usually always a dilatation and curettage.

The women in the child bearing period of life present themselves with more multiple complaints and of course require meticulous investigation. The commoner complaints might be summarized as follows:

(a) *having missed a period.*
(b) *irregular periods.*
(c) *vaginal discharges.*
(d) *pain or discomfort in lower abdomen and back.*
(e) *urinary symptoms.*
(f) *any combination of these.*

If a woman misses a period at this time of life she is pregnant until you prove she is not. Meno-metrorrhagia or epimenorrhea require a diagnostic curettage always. Suspicious cervices should be biopsied. The vaginal discharges should be treated according to the causative organism. Pain is often a baffling problem and will try your investigative powers to the fullest. The commonest causes of pain in this age group may be:

(a) *extrinsic.*
(b) *intrinsic.*

Extrinsic causes are those outside the female genitalia or pelvic viscera. Intrinsic causes are those within the female genitalia or pelvic viscera, such as endometriosis, pelvic inflammatory diseases and ectopic pregnancy.

Older women at or after the menopause come to you with a different symptomatology, e.g., vaginal discharges, vulvar irritations, vaginal bleeding, urinary symptoms and a feeling of something coming down, and bleeding. Post-menopausal bleeding means carcinoma from some pelvic organ until you prove it is not. A definite diagnosis of such bleeding will usually always require a cervical biopsy and/or a dilatation and curettage.

SOME FINAL HELPFUL LESSONS

1. A good history is invaluable.
2. Do not hurry with the history or examination — if you do, you will miss something important.
3. Remember that about 25% of your patients will not have any very serious gynaecological lesion at all, but will be emotionally disturbed. Proper psychological management of these patients will pay dividends. Remember also that about half of the 25% of patients will not have any organic gynaecological lesion whatsoever and only need moral help.
4. All women 30 years old or older should have a thorough pelvic examination every six months or more often if necessary for selected cases.
5. Pre-requisites for a pelvic examination — a cooperative patients in proper position and draped, an empty rectum and bladder. If the bladder is full pass a catheter and not an opinion. If the rectum is full give an enema before giving a gynaecological opinion.
6. Post-menopausal bleeding means cancer until you prove it is not.
7. Remember there are three possible sources of bleeding whenever a woman complains of pelvic bleeding:

   (a) *from the cervix or uterus.*
   (b) *from the urethra or bladder.*
   (c) *from anus or rectum.*

   Check all three carefully before making a final decision.

8. Psychological disturbances are common around the time of menopause and will require actually more skill in their management than most of the organic lesions.
9. Never tell a patient she cannot get pregnant without 100% good reason, because she may some day come back pregnant for sure.
10. Sprinkle a little common horse sense in all your therapy and it will pay dividends every time.

THE DIAGNOSTIC ACCURACY OF THE VAGINAL SMEAR IN CANCER OF THE UTERINE CERVIX

Olle Kjillgren; Acta Obstetrica et Gynecologica, Scandinavica, Vol. 34, No. 3, 1955.

A brief review of the development of exfoliative cytology with special references to the gynecological field and Papanicolaous' technique opens this article. A discussion of the techniques of taking smears, of staining, and of the examination of smears is followed by a description of the celluar elements of a normal smear, the criteria of malignancy, and the celluar types found in cases of uterine cervical carcinoma.

There are two major diagnostic errors possible in the technique: (a) false negative diagnoses are those with negative smears but positive cancer on biopsy; (b) false positives with positive smears but no cancer present.

The present study involves 257 cases of uterine cervical cancer found in the examination of 3000 smears. The following statistics were derived:

(a) 244 or 95% were squamous cell carcinoma, only 13 cases or 5% were adenocarcinoma.

(b) There were 241 correct and 16 false negatives diagnosed from the smears, that is the percentage of false negatives was 6.2% plus or minus 1.5%. This was corrected to 2.0% plus or minus 0.9% by the removal of those cases in which the presence of malignant cells could have been masked by recent previous treatment.

(c) Of the 16 false negatives, 5 were adenocarcinoma; that is 5 of 13 cases or 38% of adenocarcinoma were incorrect while 11 of 244 cases or 4.3% of squamous cell carcinoma were incorerct.

According to the author's statistical analysis, this difference, though large, is not significant, being compatible with the results of other investigators.

(d) Thirty-eight cases or 15% were of endocervical origin. Of the 16 false negatives, 9 were endocervical; that is 9 of 38 cases or 24% of endocervical carcinoma were missed or incorrectly diagnosed, while only 3.2% of portio vaginalis carcinoma were so misdiagnosed. This difference is statistically significant, probably due to the higher incidence of adenocarcinoma of the endocervix.

The author feels that false negative diagnoses would be reduced if the smears were taken from the endocervical region rather than from the vaginal pool.

—Bob McLachlin, Meds '57

Normal Menstrual Physiology

Kay Sandor, Meds '58

INTRODUCTION

The phenonema of estrus in the lower animals and the menstrual cycle have been well known for many years. However, the exact mechanism and processes involved were comparatively unknown until recently.

In 1917, Stockard and Papanicolaou discovered that the vaginal mucosa undergoes a cyclic histological change during the menstrual cycle. This evidence stimulated a further study of the cycle. Allen and Doisy produced an estrogenic substance capable of producing estrus in ovarectemized rats in 1923, followed by the crystalization of estrogen in 1929 by Doisy, and finally the isolation of estrogen from ovarian tissue in 1935. Even after the isolation of the estrogenic substances, it was found that the menstrual cycle could not be experimentally produced. Corner and his associates in 1929 demonstrated the presence of the second ovarian hormone which was produced by the corpus luteum and which when used with estrogen was capable of experimentally reproducing the menstrual cycle.

There are still many facts in dispute, but the author of the following paper has set forth to elucidate and review the present concepts of the normal physiology of the menstrual cycle.

HISTOLOGY

The normal human uterus is described histologically as consisting of three coats from without inwards:

1. Serous coat or visceral peritoneum
2. Muscular coat or myometrium
3. Mucous membrance or endometrium

Menstruation is more particularly concerned with the endometrium; this layer will be given a more detailed description. The endometrium or the lining of the uterine cavity has an epithelium of columnar cells and a lamina propria, usually referred to as endometrial stroma, of mesenchymal cells enmeshed in a network of reticular fibres. The columnar epithelium also lines the simple tubular glands which are scattered through the stroma and which open into the uterine cavity.

The endometrium consists of two distinct layers, the deeper basal layer and the superficial functional layer. The thin compact basal layer remains unchanged during the menstrual cycle and regenerates the thicker but more loosely constructed functional layer which is sloughed off during the bleeding period.

Blood Supply

The uterine arteries, branches of the internal iliacs, supply the endometrium. The functional and basal layers, however, receive separate arteriolar blood supplies via the straight arteries to the basal layer, and the coiled or spiral arterioles to the functional layer. The straight and spiral arterioles pass from the myometrium to the endometrium, the straight arterioles ending directly in the basal layer while the coiled or spiral arterioles end in a spray of precapillary arterioles in the functional layer.

Length of the Menstrual Cycle

The menstrual periods begin at puberty, between the ages of ten to fifteen years, and continue at regular intervals until the menopause, which occurs between the ages of forty-five to fifty. In the very early years of menstruation, it is not abnormal or unusual if there is no regular per-

iodicity of menstrual bleeding and amenorrheal periods of several months to a year may occur toward the end of the teens. However, a regular interval between menstrual periods is usually established.

The interval between successive menstruations is usually accepted to be twenty-eight days for academic discussions of the menstrual cycle. Clinically, a cycle ranging from twenty-one to forty days is considered to be within normal limits.

Length of the cycle is important for more than academic reasons. It has been successfully used in the calculation of the most fertile periods in women of lowered sterility, and also for evaluation of the infertile periods for contraceptive reasons.

Stages of the Menstrual Cycle (Histological Changes)

1. *Menstrual Stage*

The menstrual period proper is considered the first stage in the menstrual cycle and its length varies from three to seven days with the day of its onset considered to be the first day of the cycle.

Also termed the dismantling phase, it consists of the sloughing off of the functional layer of the endometrium, leaving the basal layer intact. The entire functional layer is not destroyed over the entire inner uterine surface at any one time; sloughing occuring over small areas. After the functional layer has been shed from one small area, repair of that area from the basal layer begins almost immediately as the sloughing process continues in other areas.

The sloughing occurs as the direct result of the constriction of the spiral arterioles. This constriction is thought to set in twenty-four hours before any bleeding and to continue for several hours. Necrosis results from the arteriolar constriction, the arteriolar walls weaken. As the spiral arterioles relax, blood oozes out of the weakened walls of the arterioles into the necrotic adjacent endometrium and then sloughing occurs.

The menstrual discharge consists of blood (50%-75% of the total volume), mucus, endometrial tissue and desquamated vaginal epithelium. Since menstrual blood does not clot rapidly, it is assumed that some factor is present in the endometrium which will dissolve any clots formed in the uterine cavity.

2. *Proliferative or Estrogenic Stage*

This stage commences when the damage from menstrual stage has been completely repaired (fifth to sixth day) and continues until the fourteenh day of the cycle (for eight or nine days).

During this stage the endometrium grows from less than a millimeter in thickness to two or three millimetres. The epithelial cells composing the glands and covering the endometrium are low columnar at this stage; most of these secrete a watery mucous material, but patches of nonsecretory, ciliated cells are scattered among the others. The glands are narrow and pursue a straight course through the functional layer.

3. *Progestational or Secretory Stage*

Assuming that ovulation occurs on the fourteenth day of the cycle, the progestational phase begins on the fifteenth day and continues until the twenty-eighth day, which is the final day, or until the onset of the next menstrual cycle.

During this stage the endometrium increases to about four to five millimeters in thickness, by the following mechanisms:

(1) mitosis of existant stromal cells
(2) enlargement of stromal cells
(3) accumulation of tissue fluids in intracelluar spaces
(4) accumulation of glandular secretion

The glands remain as in the estrogenic stage in the superficial part of the func-

tional layer, but in the basal layer and the deeper part of the functional layer they become elongated, coiled and dilated. The mucous secretion of the glands becomes thicker. The endometrial cells become high columnar in character and accumulate glycogen particles between their nuclei and free borders.

The endometrium is now fully prepared for the implantation of the fertilized ovum. The ovum had been released normally half way through the cycle, if fertilized, the growing ovum would then be successfully implanted. If fertilization does not occur, the menstrual period commences and the cycle is repeated.

Ovarian and Hormonal Changes During the Cycle

1. *Proliferative Stage*
   (*Fifth to Fourteenth Day*)

During this period, under the stimulation of F.S.H., follicle stimulating hormone secreted by the anterior pituitary gland, a new follicle has developed, is growing, and is stimulated to produce the hormone estrogen. Estrogen acts on the uterus of the mature female, bringing about the proliferative stage. This hormone also keeps the female sex organs in a mature developed state, and initiates at puberty the development of the secondary sex characteristics. By the fourteenth day the blood level of estrogen is such that the anterior pituitary is stimulated to release L.H. or luteinizing hormone which first acts on the follicle, causing its rupture. Regardless of the length of the menstrual cycle, it is now accepted that ovulation occurs about fifteen days before onset of flow. L.H. continues to act on the ovary, causing the development of the corpus luteum in the cavity of the ruptured follicle. The corpus luteum being formed from both the cells of the ruptured follicle and also ovarian stromal cells.

2. *Progestational or Secretory Stage*
   (*Fourteen-Twenty-eight Days*)

The corpus luteum, under stimulation of L.H., secretes progesterone, which acts on the endometrial mucosal and glands, effecting further proliferation of the mucosa and increased secretory activity.

As the progesterone level increases, the production of F.S.H. is inhibited and no further follicular development occurs. If pregnancy occurs, the corpus luteum continue to grow and secrete progesterone. If no fertilization occurs, the corpus luteum eventually ceases to function and becomes the corpus albicans, through the following mechanism: as the progesterone level in the blood rises, the production of L.H. by the anterior pituitary is inhibited, and at the same time the estrogen level has fallen and there is no stimulation for the production of L.H. Since L.H. is essential to the development and continuation of the corpus luteum, when it is no longer produced or when its blood level falls, the corpus luteum is deprived of stimulation, the progresterone level falls, allowing the release of F.S.H., which reiniates the cycle.

3. *Menstrual Cycle*
   (*First to Fifth Day*)

At onset of menstruation a primordial follicle is developing in the ovary and the corpus luteum is degenerating. There are several conflicting theories as to the exact excitant of the menstrual bleeding. They are in brief:

(1) Esrogen deprivation after corpus luteum degeneration when the blood level of estrogen is extremely low. This has been substantiated by experimentally iniating menstrual bleeding after ovariectomy. Conflicting evidence is that menstrual flow can also be initiated by large doses of estrogen.

(2) Progesterone deprivation following corpus luteum degeneration. However, this theory is untenable when it has been

shown that progesterone often evokes bleeding in amenorrheal women.

It is more plausible that it is due to a deprivation of both estrogen and progesterone which initiates and evokes menstrual flow.

(3) Acetyl choline stimulated by the presence of estrogen may bring on hyperaemia and bleeding.

(4) An unknown endometrial bleeding factor has been suggested as being present in human females and in the Rhesus monkey. This factor is postulated as acting upon the spiral arteries of the functional layer to bring about arteriolar spasm and thence bleeding.

Menstrual bleeding is frequently concurrent with various mild physical complaints even in the completely normal woman. Among these are headaches, fatigue, backache, pelvic pain and breast tenderness. Nervousness and depression are not uncommon before actual bleeding begins.

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ENDOMETRIOSIS: THE RESULTS OF CONSERVATIVE SURGERY

D. B. Whitehouse, M.D.M.R.C.O.G. and A. Bates, M.B.D.Obst.R.C.O.G. The Journal of Obstretrics and Gynecology of the British Empire, June 1955, Volume LXII, No. 3.

The treatment of endometriosis during the past 40 years has gone through a complete cycle. When first explained by Sampson in 1921, endometriosis was treated by conservative surgery. The next century found radicle surgery widely practised. The pendulum now swings back to conservative surgery with preservation of reproductive function and relief of symptoms the first principle in the treatment in younger women.

The authors divided the operations into three groups: (a) resection of pelvic endometriosis preserving reproductive function; (b) total hysterectomy conserving ovarian tissue; (c) excision of extra-abdominal endometriomata. 85% of their cases fell into group (a), 54% of these cases were followed up with the following results: 70% were completely cured of symptoms. The pregnancy rate was 45%, the recurrence rate was 33%, but only one-half of these cases required radical treatment.

In this discussion the authors state that each case presents an individual problem. They feel that it is better to err on the conservative side rather than the radical when operating. Medical treatment is discussed and compared. They doubt that medical treatment will give better results and feel that too often after trial medical treatment the disease is too extensive for conservative surgery. For young women and older women, still wanting children, the first principle is to relieve symptoms while preserving the child-bearing. This can best be achieved in a high proportion of cases by conservative surgery.

—Robert Martin, Meds '56

Endocrinology In Gynaecological Practice

Earl R. Plunkett, M.D., Ph.D.

INTRODUCTION

It is the purpose of this communication to discuss in general terms a few of the endocrine or pseudo-endocrine problems which may present in gynaecological practice. Many such conditions are associated with abnormalities of uterine bleeding and it should be clearly understood that all such patients must be given a thorough pelvic investigation.

History taking, as in all branches of medicine, should be thorough, and although the patients now under consideration present with gynaecological symptoms, a gynaecological history alone is by no means sufficient. To assess the status, it is helpful to determine any symptoms referable to each of the glands. For example, opposite thyroid, the state of thermal tolerance, condition of skin, bowel function, etc., is recorded. Details of previous treatment, particularly endocrine treatment, is very important since administered hormones may influence the type of investigation and treatment to be used. Administration of thyroid causes depression of the production of thyroid stimulating hormone from the pituitary with a consequent reduction in intrinsic thyroid activity. This results in a depression of the radioactive iodine pickup, while a B.M.R. may be elevated. In a similar manner, estrogen therapy decreases the production of pituitary gonadotrophin found upon urinary assay and Cortisone lowers the level of adrenocortical function. Furthermore, endocrine therapy for one target gland affects the functions of others via the pituitary. For example, estrogen produces an increase in the plasma protein-bound iodine (Engstrom and Markardt, 1954) and Cortisone causes an increased excretion of pituitary gonadotrophin (Noble and Plunkett, 1955).

Rather obvious but often overlooked, conclusions may be drawn from history and examination which assist in determining the function of the pituitary-ovarian axis. Uterine bleeding indicates adequate follicle stimulating hormone production and an estrogen producing ovary, provided that pelvic pathology is excluded and the very rare feminizing adrenal hyperfunction does not exist. Since gonadotrophin is usually depressed very early in all types of hypopituitarism, uterine bleeding is a very important observation in diagnosis.

In a similar fashion, breast development can only occur in the presence of estrogen and if a patient complains of delay in the menarche, breast growth helps to rule out hypopituitarism and ovarian aplasia.

The nutritional state of the patient is often important in determining the presence of endocrine disease. In spite of the fact that marked cachexia is frequently described as a symptom of hypopituitarism it is very rare that such is the case. Most instances of depressed pituitary function are not absolute and it is possible that only in the presence of gross glandular destruction marked wasting appears. Provided other organic disease is ruled out, most patients with cachexia and evidence

of depressed pituitary function have anorexia nervosa.

Very marked obesity is almost never due to endocrine disease except in rare instances of a lesion involving the hypothalamus. Hypothyroid patients may tend to be overweight but are seldom grossly obese. Obesity is often accompanied by a lowered B.M.R. due to increased skin surface and the low oxygen requirements of fat tissue.

Certain simple endocrine tests may be carried out in the office to determine the adequacy of estrogen production. These particularly apply to patients with amenorrhoea. A sample of mucus from the cervical os may be spread on a glass slide and allowed to dry. The appearance of fern-like crystals is indicative of estrogen production. With practice, the vaginal smear, suitably stained, shows a high percentage of epithelial cells with pyknotic nuclei upon estrogen stimulation. Uterine bleeding following two or three intramuscular injections of 50 mg. Progesterone at forty-eight hour intervals is diagnostic of a good estrogen level; sufficient to maintain the endometrium. This latter test is also used in the early diagnosis of pregnancy, since bleeding does not occur in the presence of a fertilized ovum.

The diagnosis of ovulation may be made by doing an endometrial biopsy with the Novak suction curette. It is best done within the first twelve hours of bleeding and has the advantages of not requiring an anaesthetic, much less cost to the patient than a D and C, easier to arrange with regard to timing in the cycle than a D and C and the patient is able to go home after a short rest. This procedure is often done in the office but in the absence of good sterilizing equipment for the instruments, the Out Patient Department of a hospital is probably the place of choice. Under usual existing circumstances the endometrial biopsy should not be used as a diagnostic method for ruling out local endometrial disease. This latter is the function of the full curettage.

ENDOCRINE DISORDERS OF THE PREPUBERTAL EPOCH

Endocrine disorders of this age group are relatively rare but when they do occur are usually of considerable magnitude. Sexual precocity including uterine bleeding before the age of nine or ten years should be regarded with suspicion. Apart from a few patients with a physiological early puberty occurring in the borderline age, the commonest cause of precocity is the granulosa cell tumour of ovary. Such patients tend to be tall for their age because of the growth promoting effect of the sex steroids, but due to the early closure of the epiphyses, eventual short stature is the rule. Breast development and gynecoid fat distribution is present along with axillary and pubic hair. Uterine bleeding tends to be irregular and if followed over several months no definite cycle is in evidence. Usually recto-abdominal examination, provided the patient is relaxed, will reveal a pelvic tumour 4 to 8 cm. in diameter. Apart from an elevated excretion of estrogen in the urine, hormone assays are of little value in this age group. Theca cell ovarian tumours and estrogen producing adrenal lesions may produce a similar clinical picture but are exceedingly rare.

Polyostotic fibrous dysplasia (Albright's syndrome) consisting of multiple bone cysts, café au lait areas of pigmentation in the skin, and sexual precocity is a rare condition affecting only girls. This may be classified as a true type of precocity since the pituitary actually produces a normal adult level of gonadotrophin resulting in stimulation of the ovaries and consequent development of the secondary sexual characteristics and cyclic ovulatory bleeding. True pituitary precocity may also be caused by pineal tumours, inflammatory disease or injury in the

region of the hypothalamus. In the case of pituitary types of precocity no endocrine therapy is indicated, since glandular function is essentially normal except for time of onset.

In all cases of precocity where the etiological factor is amenable to treatment, efforts should be made at correction. In instances where an ovarian tumour is present, surgery should be carried out as soon as possible.

Delay in puberty with hypoestrogenism will now be considered. Evidence of estrogen production may be seen normally as early as age seven with the beginning of changes in fat distribution. The more obvious evidence of breast development generally does not appear until the years of nine till twelve. Accompanying the signs of sexual maturation is an upsurge in statural growth under the stimulus of the sex steroids. The menarche usually occurs between the ages of ten and sixteen with the average for this country being approximately thirteen years.

Chronic debilitating disease and extreme nutritional deficiencies are capable of delaying sexual maturation. However, in the absence of such conditions, complete failure of pubescence at the age of sixteen or seventeen years warrants a thorough investigation. Two major possibilities exist: failure of the pituitary to produce gonadotrophins or failure of the ovaries to respond to the trophic stimuli. The former may be the result of a craniopharyngioma or other suprasellar tumours, inflammation, infarction of the pituitary, gumma, metastases, xanthomatosis or congenital absence of the pituitary eosinophiles.

If the hypopituitarism begins early in childhood or is congenital, the statural growth is usually markedly retarded, the bones are small and development is delayed, the facies remains childish, the hair is fine and silky, pubic and axillary hair growth is scant or absent and the childish appearance of such a patient persists until the third or fourth decade when a very rapid aging takes place. Complete failure of the development of secondary sex characteristics occurs. Usually there is evidence of decreased thyroid function and possibly hypoadrenal function as well. Such patients should have a thorough endocrine investigation with effort being made to establish the etiology of the pituitary deficiency. Since craniopharyngioma is the commonest pituitary or suprasellar tumour in childhood, X-rays of skull may show calcification in the suprasellar area which point to this type of lesion.

The endocrine treatment of hypopituitarism is directed toward replacement of the hormones which the patient is lacking. It should be stressed that even if the deficiency only refers to the gonadal hormones, cyclic estrogen therapy does a great deal to improve the health and general well being of the patient. Full sexual development occurs, though the patient will likely remain sterile, and the negative nitrogen balance which tends to be present is to some extent corrected. Cyclic estrogen therapy should never be carried on indefinitely since occasionally these patients do develop better pituitary function and are able to get along without treatment. Consequently, one or two months rest from treatment should be given after each four or five cycles. As a general rule pituitary tumours are not submitted to surgery unless pressure on the optic chiasm begins to cause blindness.

Failure of ovarian development often presents with the following findings: short stature, webbing of the neck, increased carrying angle of the forearms, other orthopaedic anomalies, complete failure of pubescence, mental retardation and a markedly elevated urinary gonadotrophin excretion. This series of symptoms has been called Turner's syndrome.

Ovarian aplasia, however, can occur without all of the above-described find-

ings. The two consistent observations, however, are a complete failure of sexual development and an elevated urinary gonadotrophin excretion. Treatment consists of replacement estrogen therapy usually given in twenty day cycles with a ten day rest period between. Doses of one to two mg. of Stilboestrol or its equivalent per day bring about rapid establishment of a gynecoid body habitus, a general improvement in health and a feeling of well being. Uterine withdrawal bleeding frequently occurs with this regime but the chances of bleeding may be increased by giving one to three injections of 25 mg. Progesterone during the last week of each cycle. The bleeding itself is of no importance apart from the psychological benefit which may follow.

In simple instances of a delayed menarche, where breast development, pubic and axillary hair growth and gynecoid body habitus has appeared and where no local abnormality exists in the genital tract, three or four cycles of estrogen and Progesterone therapy as described above is usually sufficient to initiate spontaneous periods.

Postpubescent amenorrhoea or secondary amenorrhoea is a relatively common gynaecological symptom. However, differentiation should be made between the patient who is perfectly healthy and has relatively few ovulatory cycles per year. Even if the bleeding only occurs three or four times a year, this patient is probably better left untreated. In order to rule out such patients and also the physiological lapse of pregnancy, Hamblen has defined amenorrhoea as absence of spontaneous uterine bleeding for at least one year. If one reviews the literature with this in mind, it soon becomes apparent that the very good results reported by a wide variety of therapeutic measures is primarily confined to that group of patients having infrequent bleeding, while the results in the treatment of true amenorrhoea are uniformly poor.

The etiological factors which may result in postpubescent amenorrhoea are extremely numerous. Almost any endocrine disease, debilitating diseases of all kinds, undernutrition, ovarian cysts and tumours, tuberculous endometritis, and psychological disturbances may cause cessation of the menses. All such patients require a very thorough medical and endocrine evaluation, including a gonadotrophin assay. The latter is depressed in hypopituitarism, elevated where ovarian failure has occurred and usually within the normal range or at the lower limit of normal in the psychogenic type of problem. Therapy in all instances should be directed at the cause of the problem.

Of particular interest is the amenorrhoea which occurs in the presence of obesity. Since many grossly obese women have cyclic cycles and are fertile, it would appear that fat deposition per se is not at fault. Hamblen (1953) has observed that amenorrhoea is most likely to occur in those patients who suddenly gain a considerable amount of weight in a short period of time. It is very possible that the psychological disturbance which stimulates the patient to eat excessively is actually the cause of the menstrual problem as well. Weight reduction and the improvement in general health which accompanies it frequently results in a return of uterine bleeding.

Amenorrhoea which persists after a difficult or traumatic delivery may be on the basis of a focal necrosis of the pituitary, producing varying degrees of hypopituitarism. This type of pituitary deficiency has been called Sheehan's syndrome. Thorough endocrine investigation is required to evaluate the amount of endocrine deficiency existing. Replacement of gonadal, thyroid and adrenal hormones is then made according to the requirements of the patient. Although the full picture of Sheehan's syndrome is not common, recent studies by Schneeberg et al (1953) in women with a history of postpartum

hemorrhage or shock has demonstrated slight suggestive evidence of decreased adrenocortical function. Persistent lactation, two or more years following a pregnancy or in the absence of any history of pregnancy when accompanied by amenorrhoea poses a difficult diagnostic problem. Since nervous stimuli from the nipples are an important factor in the maintenance of lactation following pregnancy, the history of prolonged breast feeding or mammary masturbation must be considered. Such a combination of symptoms, however, require a careful pituitary investigation since it occurs in a high percentage of chromophobe and mixed pituitary tumours. Such patients should have a periodic examination over a period of three to five years since these tumours may grow very slowly. Endocrine deficiencies may appear four to five years before any neurological or radiological evidence of the pituitary tumour appears.

Functional or psychogenic amenorrhoea may persist for years without evidence of hypoestrogenism. These patients usually have a normal or lower limit of normal follicle stimulating hormone production but are lacking in luteinizing hormone. The endometrium is in a resting estrogenic phase and is capable usually of responding to Progesterone with bleeding. The vaginal mucosa seldom becomes atrophic and breast regression is uncommon. The promotion of withdrawal bleeding by estrogen and Progesterone cyclic treatment is successful in a small percentage of cases in promoting spontaneous cyclic bleeding. Low dosage irradiation therapy of the ovaries and pituitary has been studied over three generations by Kaplan, Rubin and others without encountering genetic effects. Recently Rakoff and Perloff, independently at the Canadian Society for the Study of Fertility meeting in Montreal, 1954, said they found no difference in the results between patients treated with pituitary and ovarian radiation and those receiving ovarian radiation alone. There is no question that this form of therapy is effective and it seems quite justified when used cauiously after other measures have failed. Psychotherapy, carbon dioxide inhalation therapy and electro-shock treatment have been observed to restore bleeding in patients under treatment for severe psychopathies.

Virilizing syndromes are not common but one is frequently faced with the problem of hirsutism. Virilization means more than this, however, and includes voice change, prominence of the larynx, oily skin and acne, increased muscularity, regression of the hair at the temples, breast regression, amenorrhoea and frequently an increase in libido. These changes may be due to a virilizing tumour of the ovary, adrenocortical hyperplasia or adrenal tumour. In all cases there is an increase of Androgen production which is usually reflected in an elevation of the urinary 17 ketosteroids. When the virilizing lesion is situated in the adrenal it is possible to have excessive production of other cortical hormones resulting in Cushing's syndrome. A trial of Cortisone therapy frequently differentiates tumour of ovary or adrenal from adrenocortical hyperplasia by its ability to suppress the excretion of 17 ketosteroids in the latter instance while failing to do so in the presence of tumour. The presence of a mass in pelvis or the upper part of the abdomen, evidence of renal displacement in an intravenous pyelogram and an enlarged adrenal shadow radiologically after presacral air insufflation provides additional diagnostic evidence.

The treatment of either tumour is surgical while the adrenocortical hyperplasia can now be treated successfully by maintenance doses of Cortisone sufficient to keep the 17 ketosteroids within normal limits. The usual daily requirement is 25 to 50 mg. per day in divided doses. Suppression of the adrenal by this therapy via inhibition of ACTH allows the pituitary ovarian function to occur with consequent production of estrogen and

resumption of uterine bleeding. A virilizing process present from birth is usually on the basis of adrenocortical hyperplasia while appearance of the symptoms later in life are more likely to be due to tumour.

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PALLIATIVE TREATMENT OF PROLAPSE OF THE UTERUS

Solomon Gold, Montreal. Canadian Medical Association Journal, 1953.

Prolapse of the uterus usually results from obstetrical injuries and may be aggravated post-menopausally when there is loss of tone of pelvic viscera. Pelvic pressure, central low lumbar or sacral pain, urinary and bowel disturbances, often associated with constitutional and nervous symptoms are common complaints. Cystitis from residual urine may cause frequency, dysuria and even anuria. In complete prolapse, the above symptoms plus difficulty in walking, epidermidization of the cervix, circulatory congestion and hypertrophy of the uterus may be recorded.

Surgery is the treatment of choice. If this treatment is refused or contraindicated, palliative therapy is in order. Mechanical devices such as hard ring pessaries or soft rubber doughnuts afford a certain amount of relief by stretching the vaginal walls and the vault of the vagina. These pessaries bend to roll out with the folds of the vagina when there has been too much stretching.

Five menopausal women were treated by inserting a rubber bulb into the vagina after the uterus was displaced (asepto syringe bulb B.D.). The bulb was inserted while compressed and folded longitudinally by the index finger, the neck of the bulb toward the introitus. This supports the vault and pushes the bladder upward and somewhat anteriorly. If the bulb comes out white stained a larger bulb is used.

Daily cleansing douches and bimonthly removal and reinsertion is advised. Smaller sized bulbs may be resorted to as the introitus becomes smaller. A few months sees an improvement in pelvic tone and also in urinary and emotional disturbances. No damage to vaginal mucosa was observed in this series. This treatment may be of value preoperatively to allow uterine supporting structures to contract.

—T. E. Staples, Meds '56

Dysmenorrhoea

Jocelyn Pearce, Meds '56

INTRODUCTION

Dysmenorrhoea has been defined as difficult menstruation and is generally interpreted as pain related to the menstrual cycle, either preceding the onset, during or immediately following the cessation of flow.

Until recent times there has been little reference to this clinical problem, and this may be partly due to the female reticence in discussing menstrual difficulties with her physician, and partly due to the male assumption that pain connected with menses is normal. Early references contain mention only to dysmenorrhoea as the result of cervical stenosis, "Nulla dysmenorrhoea nisi Obstructive." However, the causes of this symptom are many, and some are put forth in this paper. It is of paramount importance that we remember that dysmenorrhoea is a symptom only, and although palliative symptomatic therapy is necessary, the prime purpose should be to determine the exact etiological cause and then treat that cause.

The term dysmenorrhoea refers to a symptom which is exceedingly common and is important both to the specialist in the field of Gynecology and to the general practitioner. If the word is broken up into its component syllables it will be seen that the literal meaning is as follows: difficult; plus month; plus to flow. The word therefore means pain or discomfort associated with the monthly menstrual flow. Many women experience some discomfort at this time, but normally they consider it as a minor inconvenience if they consider it at all. If a little more pain occurs the patient may take aspirins or some other readily obtainable drug for a day or two each month, but not consider it of sufficient importance to seek a doctor's help. However, there are a large number of women who find it impossible to carry on their regular work for a few days each month; there are others who are forced by the extreme discomfort to remain in bed. It is these patients who come to the doctor for relief from their distress, and this article is primarily aimed at reviewing the various theories of etiology and the treatments which have been and still are being tried in this condition. Quite apart from the humanitarian point of view, dysmenorrhoea is of considerable importance because of the economic wastage which it causes. It is well known by firms that employ large numbers of women that more working days are lost through dysmenorrhoea than because of any other form of ill-health except perhaps the common cold. Efficiency is also lowered appreciably and more accidents occur in factories because of this lowered efficiency and concentration. At the risk of discouraging some of my readers from proceeding further, I would like to stress right at the outset of this article that there is still no ideal treatment for this condition and no certain cure. This, of course, is no excuse for neglect by the physician to examine such a patient thoroughly and attempt conscientiously to alleviate her symptoms.

Types of Dysmenorrhoea

Before one can discuss the treatment of this condition intelligently, something of the etiology must be known. There are two clearly demarcated types of dysmenorrhoea, primary and secondary, or, if you like, essential and acquired. Secondary dysmenorrhoea is due to some demonstrable pathology in either the pelvic or the neighbouring abdominal or-

gans. There may be some degree of constant pain in these instances, which is accentuated at the time of mensruation, or the pain may only be present at the period. It is thought that the increased congestion of the pelvic organs coincident with menstruation increases the pain of the chronic disease. Primary dysmenorrhoea, on the other hand, is defined as pain occurring with the menstrual flow in the absence of any demonstrable pathology in the pelvic organs. Because the cause of this type has not been generally agreed upon, the treatment also varies with the individual physician, but there are several general principles employed by all which I shall discuss later.

Differential Diagnosis

It is immediately obvious that before treatment of any definite nature can begin, the physician must satisfy himself as to the type of dysmenorrhoea present.

Primary Dysmenorrhoea

There are several factors about the pain of the primary type which will help in this differentiation. It is known that anovulatory menstrual cycles are painless menstrual cycles. This explains the common finding that the patient with primary dysmenorrhoea had quite normal periods for a few months to years after the menarche, and that the pain commenced when the patient was in her late teens or early twenties. Characteristically the discomfort commences a day or so before the flow begins, to last until the first day of the flow. In atypical cases the pain may be of only a few minutes duration or it may last throughout the period and even for a few days after the flow has ceased. One commonly finds nausea and vomiting, headache or backache, and a feeling of depression and irritability accompanying the actual pain. Most patients complain of constipation, but diarrhoea may occasionally be the main symptom. The amount of the flow seems to have very little relation to the presence or absence of abnormal pain, but the passage of clots may be painful in some cases. The character of the pain is again typical. It is of a crampy, spasmodic variety, compared by some to miniature labour pains. The spasms may be very severe, and in emotional individuals may be so severe that some patients are reported to have contemplated suicide. The discomfort is sometimes present as an aching and a sensation of pressure in the pelvis, but these feelings are more often due to secondary dysmenorrhoea. The latter also differs from primary in that the discomfort tends to occur during the flow rather than before it, and fewer of the secondary symptoms such as depression and emotionalism are seen.

Secondary Dysmenorrhoea

As can be appreciated, the history of the discomfort is important in the differential diagnosis. Equally important is a thorough general and pelvic examination which will often confirm the physician's impression as to the presence or absence of definite pathology. The commonest pathology one finds in association with this symptom is that of chronic inflammation of the uterus and adnexa, and it will often be found to have commenced with childbirth (whereas primary dysmenorrhoea often disappears with childbirth). It can also be caused by neoplasms of the pelvis, especially ovarian cysts; occasionally by retrodisplacement of the uterus, or by cervical obstruction caused in turn by radiation or pelvic surgery. A history of pain commencing in the mid or late twenties and becoming progressively worse over the years strongly suggests endometriosis. On the other hand, the etiology may be found in extragenital lesions of the bladder, ureter, or sacro-iliac joints. The above list is by no means exhaustive but it gives an indication of the multitude of possible causes for secondary dysmenorrhoea. It must be self-evident that permanent relief from

the pain in these instances depends entirely on the discovery and eradication of the underlying pathology. Until such treatment can be instituted, the pain can be controlled with rest, local heat to the abdomen, and mild analgesics. This temporary control will be discussed further under the treatment of primary dysmenorrhoea.

The Etiology of Primary Dysmenorrhoea

For a great many years gynecologists have attempted to discover the cause of this common complaint, but, although many theories have been advanced and some apparently proven at the time, the final answer is not yet known. One of the original concepts concerned the patency of the cervical canal. It was felt that a congenitally small canal would obstruct the flow of blood and that the pain resulted as the uterus attempted to expel this blood. It was demonstrated that the so-called "pin-hole os", or the condition of acute anteflexion of the uterus (which would also narrow the os) did indeed exist in many of these patients. Under this assumption the recommended treatment was dilatation of the cervix. The rationale of this therapy was strengthened by the fact that dysmenorrhoea often ceased after the birth of a baby. Dilatation of the cervix is still recommended in some cases, but unfortunately much damage was done by over-treatment. Scarring due to tears of the cervix often prevented adequate dilatation of the cervix at subsequent labours, thus materially obstructing these labours. The treatment frequently did relieve the distress temporarily, but it usually returned after several months or years, and Novak feels that many of these reported cures may have been due to the psychic element present in the original condition. It is known that many patients have acute anteflexion or the "pin-hole os" mentioned above with no symptoms whatever. However, some authorities still consider that cervical stenosis is worthy of consideration in primary dysmenorrhoea and it should be remembered that no one has yet explained the fact that pregnancy often effects a cure.

Because of the crampy nature of the pains, it has been suggested that the uterus is overly irritable and undergoes accentuated contractions in those patients who experience severe pain. In order to test this theory, rubber bags were placed in the uteri of patients with dysmenorrhoea and the time of the contractions compared with that of the cramps. These two phenomena synchronized remarkably well and the contractions did seem to be stronger than those measured in normal women. This theory seemed to be reasonable, but it did not explain why the contractions were stronger. Since then similarly strong contractions have been observed in persons without any dysmenorrhoea, but this does not entirely exclude this theory. It may be argued that these latter patients have a higher threshold of pain and so do not complain of it as readily.

Congenital hypoplasia of the uterus has been noted in quite a few patients and therefore has in its turn been considered as a causative factor. However, it is not always found and so is not apparently a necessary factor. Also it must be remembered that hypoplasia has been recognized quite frequently in patients who do not complain of menstrual pain at all. This factor has therefore taken its place with the rest as having some place in the causation of the pain in some patients but being by no means a major factor. It is now postulated that even when hypoplasia is present it is only a sign of an underlying endocrine disorder which in turn is the real cause of the dysmenorrhoea.

An interesting theory, and one which ties in with a theory previously mentioned, is that of Chassar Moir. He postulates that the excess uterine contractions may increase the intra-uterine pressure until it exceeds the blood pressure, therefore

preventing the entrance of blood into the uterine vessels. This will cause ischemia of the uterine musculature which in turn will cause pain on the same principle as the pain of myocardial insufficiency. This seems to be a reasonable theory as to why the contractions cause the pain, but of course it does not attempt to explain why the excess contractions are present.

The above theories as to the causation of the pain are not made use of in deciding on the clinical treatment as extensively as are the next three theories to be mentioned.

The psychogenic theory is the first of these. There is no doubt that there is a large subjective factor in a number of these patients, and the dividing line between the normal discomfort coincident with the menstrual flow and dysmenorrhoea is very hard to determine. It must be decided by the patient herself in a large percentage of cases and it is difficult but imperative for the doctor to decide whether she has excess pain or is under the impression that the normal discomfort is abnormal. The patient's education has a great deal to do with this. There may be others in her family who had dysmenorrhoea and she may consider that she should feel ill at her periods. This patient may be entirely cured by psychotherapy alone. She must be encouraged to be up and about during her periods and must be convinced to her own satisfaction that menstruation is a physiological rather than a pathological phenomenon. The patient who is under emotional tension at home or at work is also a candidate for dysmenorrhoea and may be cured by psychotherapy alone if the physician is able to devote sufficient time to discovering the source of her tension and helping her to overcome it.

Another of the important factors is constitutional debility. The patient who is the victim of chronic disease, overwork, or chronic fatigue is more liable to be affected by pain. Her physiological pain threshold is lowered. It may be very difficult to decide how much emphasis should be placed on this aspect and how much on psychogenic factors in any individual patient. One should always begin treatment in these patients by attempting to increase their general health and occasionally this may be all that is necessary.

As is occurring at the present time in almost every field of medicine in which the causation is not definitely known, endocrine aberration is considered to be the basic disorder in dysmenorrhoeas which are not purely psychic in origin. Again there have been many theories concerning the exact type of endocrine disorder which would produce the excess uterine contractions, for the latter are still considered as the cause of the pain. It is agreed that estrogen increases the contractility of the uterine muscle and that probably progesterone decreases it. The pain cannot be due to excess estrogen alone even though this would be a pleasingly simple explanation. The pain does not occur right after puberty and it does not occur in functional bleeding where it is known that there is excess estrogen and a deficiency of progesterone. The cramps are therefore probably due to an imbalance in the amounts of estrogen and progesterone present, but just what this imbalance is and in what way it increases the contractility of the muscle is still under debate. Nevertheless, endocrine treatment is directed empirically towards decreasing this excess contractility, or towards preventing ovulation, for an anovulatory cycle is a painless cycle. The latter can be accomplished by the use of estrogen and the former by progesterone, testosterone, or chorionic hormone.

Treatment of Primary Dysmenorrhoea

The reader is now familiar with the principles of the treatment of dysmenor-

rhoea, but a summary will probably not be amiss before I commence the discussion in detail. There are three divisions in the treatment:

(1) Relief at the time of the attack.

(2) Attempts at permanent relief.

(3) Endocrine therapy.

These treatments are based mainly on the four present theories of causation:

(1) Psychogenic.

(2) Constitutional debility.

(3) Endocrine aberrations.

(4) Cervical stenosis in some cases.

Before I proceed let me stress that the physician's first concern must be to do no harm. In most cases this is an annoying but not a dangerous disorder and it must not be turned into the latter. Habit forming drugs must never be used to relieve the pain and endocrines must be used only where there is a definite indication and then used with discretion.

(1) Relief at the Time of the Attack

This is usually easily obtained. The simplest remedies should be tried first, including hot baths, local heat to the abdomen, and non-habit forming analgesics such as aspirin or phenacetin. If the latter are not strong enough, codeine may be used. There are many commercial remedies on the market, containing some or all of the following ingredients: aspirin, phenacetin, codeine, phenobarbitol and dexedrine. Daprysol is one good example of these, containing analgesics, dexedrine and phenobarbitol. The dexedrine relieves much of the associated depression despite being partially neutralized by the phenobarbitol. May I stress again that habit forming drugs such as morphine or alcohol should never be used, even though the severity of the pain may at times seem to call for them.

(2) Attempts at Permanent Relief

After a physical examination has been done to rule out any possible pathology, a careful interview is necessary in order to determine what role, if any, psychogenic factors play in the particular case under treatment. If one decides that there are not any underlying emotional factors, then one can proceed with other definite treatments. However, if tension is seen to be a major factor then the physician is wise to start his treatment along these lines. The patient may not need anything but psychotherapy, or it may at least produce a marked improvement.

General supportive measures may be instituted next. It is of course sensible that if the patient is grossly over or under weight this should be corrected, and any anemia or other chronic debilitating disease must be treated. The patient should be encouraged to take regular exercise, preferably outdoor sports if this is possible, and to remain up and around during her menstrual period unless the pain is very severe. It is a mistake to advise her to remain in bed at the least suggestion of discomfort. It is known that mild exercise tends to relieve the cramps and it certainly helps to keep the patient's attention from centering on the pain. The Billig exercises have been especially designed for the patient who is unable to get regular exercise in any other way. I will not describe them here but a good discussion can be found in the text *"Medical Treatment in Obstetrics and Gynecology"*, by C. F. Fluhmann. Though none of these things can be classed as definite treatment, they will be found to help many patients whose symptoms are not too severe. Whether the results are purely due to psychogenic factors is a debatable point which I shall not attempt to answer.

Dilatation of the cervix has been referred to before in this article. It is considered by some to have great usefulness in instances in which the above treatments

have failed to produce any remission of symptoms. As long as the operator does not tear the cervix in any way this may be a good treatment. A single dilatation of the cervix followed by packing in the uterus for three to four days may be sufficient. Some recommend dilatation with Hagar dilators, repeated at three day intervals for ten days before the period, this treatment to be repeated on three successive months. This treatment is rarely permanent, though it may be in some instances and so is worth an attempt. In most cases it will alleviate the symptoms for several months, which may be highly desirable from a psychological point of view. By breaking the cycle of pain each month it will relieve much of the patient's apprehension.

There is one further surgical treatment which should be used only as a last resort. If all the other methods of treatment have failed and the patient is in such severe pain each month that something obviously must be done about it, then this surgical procedure may be used. The procedure I refer to is a presacral neurectomy. This is quite effective when it is done and there are few undesirable after-effects. It must be remembered that the sensory nerves to the bladder have also been cut and the patient should be warned not to wait for painful sensations before emptying her bladder. A complete loss of bladder control is a rare sequel to this operation. It must also be remembered, however, that this is a major abdominal operation and so not to be undertaken without adequate indication.

(3) Endocrine Therapy

This is the newest form of treatment. Almost all the endocrine preparations have been tried at one time or another but there are only two in general use at the present time and one other which is used occasionally. Shot-gun therapy with two or more of these drugs at one time is most undesirable. The patient may receive some benefit from such treatment but the physician will never know which drug effected the cure, he will be putting the patient to unnecessary expense, and he will be exposing her to the undesirable side effects of all the drugs when one would have been sufficient.

The endocrine preparation in commonest use perhaps is dessicated thyroid extract. It has been found that a considerable number of patients with severe dysmenorrhoea have a low B.M.R., but even if the B.M.R. is normal it has been observed empirically that thyroid extract is often helpful. It can be given in the following dosage: ½-1 gr. b.i.d. for a few days, increasing gradually to 2 gr. b.i.d. if necessary. A definite systemic response to the thyroid extract indicates that the optimum dosage has been reached, this dose to be decided for each patient individually. This treatment may be continued for two to three months, but not indefinitely.

The other commonly used endocrine preparation is some form of estrogen. It must be stated that estrogens should not be used routinely but only if an underdeveloped uterus can be definitely demonstrated. It can sometimes be of considerable help in these cases and should be given in the first half of the menstrual cycle. Small doses are used in the hope of stimulating growth of the underdeveloped uterus but larger doses are used to stop ovulation and so produce a painless period. There are several preparations of estrogen which can be used. Some of the commoner ones are:

(1) Diethylstilboestrol, which is given in the dosage of 1 mgm. per day for the first fifteen days of the cycle.

(2) Estradiol benzoate, which is given as 1.66 mgm. I.M. beginning on the sixth day of the cycle and repeated every three days for six doses.

(3) Estinyl and premarin are possible oral preparations.

The above dosages are those required to stop ovulation. There are of course many variations of the above treatment as to details of dosage and time of administration, but the above are quoted as examples. There are side effects such as nausea and vomiting and abdominal pain, and there are unwanted after-effects such as a profuse flow during the period after that in which treatment was used. Estrogen must never be administered continuously but only for two or three consecutive periods. The treatment is purely temporary and the pain begins again when it is discontinued, but the relief for several months will be a boon to the patient and if there was a large psychogenic aspect there is again the chance of curing the patient by breaking the cycle. The estrogen also stimulates the under-developed uterus and so may on occasion effect a permanent cure in this manner.

Testosterone propionate and progesterone have both been used in the past and are occasionally used now. These are quite costly and again only temporary, so there is little need to use them in preference to estrogen.

Summary

In this article I have attempted to bring to the reader's attention the importance of dysmenorrhoea, particularly the primary type, and the essential simplicity of its management if the physician keeps the probable etiology in his mind at all times and takes as his primary rule that he will do no harm to the patient. He must start with the simpler forms of management, he must use estrogen only if there is definite evidence of underdevelopment of the uterus, and he must reserve the surgical treatment for severe cases refractory to all other measures. If these general principles are remembered and followed, the practitioner should have a large percentage of successes in the treatment of dysmenorrhoea.

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Diagnostic Quiz

Don Bondy, Meds '56

Many Journals feature quizzes of this nature but rarely are they devoted entirely to Gynecological disorders.

We have selected six classical cases and presented the minimum essential clinical evidence required for a diagnosis. The diagnoses are printed at the bottom of the page.

CASE #1—

A 28 year white female, three months pregnant, presents herself complaining of hypogastric pain, nausea and urinary retention. Examination revealed a hypogastric swelling larger than that compatible with a three-month pregnancy. Catheterization failed to reduce this swelling.

CASE #2—

A 32 year white female in the 16th week of her first pregnancy phones her obsetrician complaining of lumbar backache, hypogastric pain and slight uterine bleeding.

CASE #3—

A 24 year white female, four months pregnant, complains of a sudden rise in temperature (103 degrees), rigors and a feeling of chilliness, and abdominal pain. Examination revealed her to have tenderness in the right loin under the costal margin, costovertebral tenderness on the right side. Urinalysis showed turbidity, albuminuria, pus cells and on culture E. coli.

CASE #4—

A 19 year white female is admitted to hospital with a sudden acute attack of hypogastric pain. She had been vomiting since the onset of the pain. Palpation of the abdomen revealed a definite round tender swelling in the hypogastrium. This swelling was also palpable by vaginal examination.

CASE #5—

A 26 year white female, in her sixteenth week of her second pregnancy, complains of a sudden abdominal pain and vomiting. Her abdomen is tender and swollen. She also complains of vertigo and faintness. Physical examination also shows the presence of free fluid in the abdomen and tenderness on digital pressure over the pouch of Douglas. Serial haemoglobin determinations show a rapidly progressing anemia. Further questioning revealed that she had had some "spotting" for the past two weeks.

CASE #6—

A 68 year white female, who is underweight and in state of poor nourishment complains of a bloody, foul smelling discharge. Examination and culture of this discharge failed to reveal Trichomonas organisms. Vaginal smear, using the Panpanicoleou technique, was negative.

ANSWERS TO DIAGNOSTIC QUIZ—

1. Retroverted uterus.
2. Threatened Abortion.
3. Pyelitis.
4. Ovarian Cyst with twisted pedicle
5. Rupture of Tubal Pregnancy.
6. Carcinoma of Cervix (endocervix).

Amenorrhea

Ellen Martin, Meds '56

INTRODUCTION

Amenorrhoea is a symptom only and indicates some interference with the menstrual cycle. The diagnostic problem is to determine at which site the interference occurs. The normal cycle depends upon the harmonious correlation of the hormonal function of the anterior pituitary and of the ovary, and the uterus, any defect either functional or structural of these organs will cause amenorrhoea. It is important to consider the role of the hypothalamus in the control of the pituitary, and also the interrelationship of all the endocrine organs, because a defect in one, may secondarily affect the others.

Amenorrhoea is by definition the absence of menstruation for one or more periods between puberty and menopause.

It is not a disease but merely a symptom. It may be primary, in which case menstruation has never occurred or it may be secondary, in which case menstruation has been established and then arrested due to a variety of causes. A classification according to etiology is very helpful.

Classification of Amenorrhoea

I. *Apparent*:

1. Congenital
   - —imperforate vagina
   - —absence of vagina
   - —imperforate cervix
2. Acquired
   - (a) Closure of vagina
     - —due to specific fevers
     - —due to injury
   - (b) Closure of cervix
     - —due to injury
     - —following operations

II. *Real*:

1. Physiological
   - (a) During pregnancy
   - (b) During lactation
2. Pathological
   - (a) Generative System
     - —absence of essential organs
     - —infantile uterus
     - —small adult type of uterus
     - —destruction of both ovaries
   - (b) Circulatory system
     - —anemia
     - —leukemia
     - —Hodgkin's disease
   - (c) Wasting diseases
     - —malignant growths
     - —tuberculosis
     - —prolonged suppuration
     - —diabetes
     - —late stages of nephritis
   - (d) Toxic
     - —after specific fevers
     - —chronic poisoning from lead, mercury morphine
   - (e) Diet
     - —alcohol
     - —ketogenic diet
   - (f) Altered Internal Secretion
     - —anterior pituitary disturbances; panhyopituitarism; chromophobe, acidophilic, basophilic adenoma
     - —myxedema
     - —Exopthalmic goitre
     - —Addison's Disease
     - —obesity
     - —change of habits or environment

(g) Nervous System
—various forms of insanity
—cold just before the onset of flow, or during flow
—suggestion (fear of pregnancy)
—Anorexia nervosa

Such a classification is useful in the assessment of the patient whose presenting and often only complaint is amenorrhoea.

Investigation

History—The diagnosis of the cause may occasionally be made on the basis of history alone. In primary amenorrhoea some constitutional or congenital abnormality should be sought for. Injuries at birth and childhood illnesses should be noted. The patient should be questioned about the onset of menstruation and cycles of the other members of her own family.

In secondary amenorrhoea, one should investigate the mode and the time of onset of menses, psychological effects of menstruation, changes in environment and emotional disturbances which may have some effect on the menstrual cycle. Former diseases and chronic dehabilitating illnesses are also important.

Physical Examination—The physical examination should include: (a) a complete physical, at which time general body build, hair distribution, secondary sex characteristics should be noted; (b) a pelvic examination.

Special Examinations—Special techniques which may be useful are endometrial biopsy, X-ray of the sella tursica, basal metabolic rate, and if indicated, hormone analysis of the blood and urine.

Differential Diagnosis

The physician's first concern, when a patient presents with amenorrhoea, is to determine if it is primary or secondary. This can usually be determined by an accurate history. Primary amenorrhoea may be due to congenital absence or malformation of the genital organs, disease or disturbance of nuitrition or disturbances of the endocrine system.

The next step in the differential diagnosis is to determine if amenorrhoea is apparent or real. In apparent amenorrhoea, there is an absence of external bleeding but the menstrual cycle is normal there being some form of obstruction or mechanical abnormality of the genital tract. If this condition continues, haematocolpos and haematosalpinx may occur. There is usually a history of periodic cyclic discomfort similar to that experienced with normal menstruation but without external bleeding. As time goes on, and the blood continues to accumulate behind the obstruction, the pain may become almost continuous due to the accumulation of blood in the uterus, tubes and occasionally the peritoneal cavity.

The causes of this condition may be congenital or acquired. Congenital causes include congenital absence and imperforate vagina, imperforate hymen and imperforate cervix and double uterus with retention. Acquired causes may be due to closure of the vagina or cervix from injury following surgery or specific fevers. Vaginal examination of these cases will reveal an obstruction above where one usually feels the distended vagina or uterus. Of course in the case of imperforate hymen no vaginal examination is possible, but the hymen may be distended and bulging with blood. Rectal examination may give some indication of the volume of blood behind the obstruction. In severe cases a mass is easily palpated abdominally. The treatment in these cases is the relief of the obstruction by appropriate surgery.

Real Amenorrhoea

(1) *Physiological Causes*

Real amenorrhoea will now be considered. Real amenorrhoea may be either physiological or pathological. Physiological causes are by far the most common causes.

By definition, amenorrhoea is the absence of menstruation for one or more periods between puberty and the menopause, so we have already excluded amenorrhoea which occurs before the onset of puberty and that which occurs after the menopause. However, after the menopause has onsetted, there may be scanty, irregular periods for a time before the complete cessation of menstruation. Pregnancy should always be excluded by the usual and routine obstetrical examinations. During lactation there is usually no external manifestations of the menstrual cycle, although ovulation does occur during the period of lactation without the resumption of the menstrual flow.

(2) *Pathological Causes*

The pathological causes of amenorrhoea are diverse and require special attention.

(a) Pathology of Generative System:

The uterus may be congenitally absent, hypoplastic or refractory to the stimulation of the sex hormones. Congenital absence of the uterus is a rare anomaly, but hysterectomy is becoming quite common and has been overlooked in the past as a cause of amenorrhoea. Hypoplasia of the uterus may be either a primary or a secondary cause. Uterine hypoplasia, when causing a primary amenorrhoea, is usually the result of decreased ovarian and pituitary function. In this case the signs of hypopituitrism are also present. Sometimes uterine hypoplasia is associated with small external genitalia, but the breasts, secondary sex characteristics, and growth are usually normal. It is also of interest that in some, excretion of gonadotrophic hormones and estrogen is normal. This suggests that the genital duct system may be refractory to hormonal stimulus. Secondary amenorrhoea due to uterine hypoplasia can usually be corrected by estrogen, although untreated cases of long standing may be refractory.

Congenital absence of the ovary is rare but may occur. Peritonitis in childhood may destroy ovarian tissue, leading to non-functioning of the ovary. In adult women follicular cysts may cause short periods of amenorrhoea, as may also corpus luteum cysts. Sclerotic disease of the ovary in which there is increased fibrosis of the ovaries is believed occasionally to cause amenorrhoea by decreasing estrogen formation.

Tumours of the ovary which cause destruction of both ovaries may rarely cause amenorrhoea. The arrhenoblastoma, by production of androgenic hormone arrests follicular maturation, ovulation and luteum formation and leads to cessation of menstruation. Inflammatory disease of the pelvis, and irradiation also destroy ovarian tissue and amenorrhoea ensues. Of unknown cause is a premature aplasia of the ovaries which in essence is an early menopause. The aplasia of the ovaries is unaffected by pituitary stimulation. This occurs in the menopause also. In both, gonadotrophic hormone is up and the hypophysis becomes hyperplastic.

(b) Systemic Disorders:

Amenorrhoea may occur in conjunction with other diseases. Various wasting diseases and circulatory disorders which are commonly associated with amenorrhoea are listed in the classification above. Dietetic and toxic factors may be concerned. Protein and the B vitamins are necessary for the proper function of the pituitary.

(c) Pathology of the Endocrine Glands:

(i) Anterior Pituitary—

The anterior pituitary contains three types of cells. The chromophobes do not produce any known hormone. The acidophils produce growth hormone and the basophils produce gonadotrophins.

Panhypopituitrism is a state in which all the gland is underfunctioning. There is consequently inadequate stimulation of the ovaries and consequent amenorrhoea. Here the general appearance of the patient will point to the diagnosis. 17-ketosteroid estimation in these cases is practically zero. Included in this group of diseases are Simmond's disease, pituitary dwarfism and Sheehan's syndrome. Sheehan's syndrome may follow difficult delivery, especially if there has been severe post partum hemorrhage. It is believed to be caused by ischemia of the pituitary due to thrombosis of its vessels. Destruction of 75% of the gland causes moderate symptoms according to Sheehan.

Chromophobe adenomas and craniopharyngioma may cause hypopituitrism by pressure on the other cells of the pituitary. X-rays of the sella-tursica are helpful in diagnosis. If a tumour of the acidophilic cells is large enough to cause considerable destruction of the pituitary, ovarian function may cease with amenorrhoea following. The signs and symptoms in an acidophilic tumour are those of acromegaly.

Basophil tumours result in Cushing's syndrome with amenorrhoea. They present a picture similar to those with adrenal tumour, which will be mentioned later.

Lesions of the hypothalamic area may upset pituitary function, leading to dystrophia adiposo-genitalis (Froehlick's syndrome). There is genital hypoplasia, amenorrhoea and a girdle type of obesity.

(ii) Adrenal Cortex—

In hyperplasia and tumours of the adrenal cortex there is increased production of adrenal androgens. This leads to virilism and amenorrhoea. 17-ketosteroid excretion is high. In Addison's disease the adrenal hormone production is decreased and 17-ketosteroid values fall to almost zero. However, ovarian function may be normal until debility is so great that pituitary function is disturbed. Amenorrhoea in these patients often disappears when malnutrition is corrected by adrenal cortical hormone replacement.

(iii) Thyroid—

Both hypothyroidism and hyperthyroidism may lead to amenorrhoea. The hypothyroid amenorrhoea is thought to be due to poor cellular activity of the whole body, including the cells of the pituitary and ovary. In hyperthyroidism amenorrhoea may be caused by toxemia which also affects the ovaries. In some cases the amenorrhoea is attributed to rapid excretion of estrogens which prevents its concentration in the uterine mucosa, thereby effecting amenorrhoea.

Functional Amenorrhoea

It has been suggested that sixty percent of cases of amenorrhoea are functional, of these cases fifty percent are also obese. This suggests that the hypothalamus may be involved in this problem. Psychological stimulus may affect the hypothalamus directly from the cerebral cortex. It is known that shock, anxiety, sexual disharmony, depression, change in occupation or change in environment cause menstrual disorders, but the exact mechanism is unknown.

Anorexia nervosa represents a severe reaction to trauma. It generally occurs in adolescent girls. Extreme wasting and emanciation may occur. These cases must not be confused with Simmond's disease, which is usually a disease of older women, onsetting after a pregnancy. In anorexia nervosa, there is no breast atrophy, no loss of pubic or axillary hair and there is no decrease in the urinary level of 17-ketosteroids.

The treatment of functional amenorrhoea is to determine the abnormal psychological stimulus and eliminate it. How-

ever, this often requires a prolonged period of psychotherapy. The practitioner should be on the lookout for this type since they make up a large proportion of all cases of amenorrhoea.

Conclusion

The underlying cause of amenorrhoea should always be determined. Treatment should not be too long delayed because certain cases become refractory to treatment if neglected too long. The aim is to treat the underlying disease wherever possible rather than merely symptomatic treatment.

SOME PROBLEMS OF CURRENT INTEREST RELATING TO CLASSIFICATION AND TREATMENT OF UTERINE CARCINOMA

James Heyman, M.D., Stockholm, Sweden. American Journal of Obstetrics and Gynecology, March, 1955.

Heyman states at the outset that the value of different methods of treatment can be established only by comparing results presented in uniform and comparable therapeutic statistics. Uniformity of statistics requires precise definitions on the varieties of the disease. The two conventional groups are carcinoma of the cervix and carcinoma of the corpus. He believes that it is not justifiable to compare statistics based on the surgeon's classificaion against those cases based on clinical classification.

Carcinoma of the Cervix

Classification:

Stage O must be more precisely defined.

Treatment:

Intracavitory radium applications at present is the most effective treatment in the control of cervical carcinoma. Surgery is used in cases of failure or local recurrence following radiotherapy.

Five Year Recovery Rate:

(1936-1948 at Radium liemmet)

Total number of cases treated—3704.

Relative Recovery Rate—42.2%.

Stage I —12.2% total number cases; recovery 71.0%.

Stage II —51.4% total number cases; recovery 50.3%.

Stage III—28.1% total number cases; recovery 24.7%.

Stage IV— 8.3% total number cases; recovery 9.1%.

The figures show that the recovery rate in Stage I is 62.5% at institutions in favour of primary surgery, and 65.3% at those in favour of primary radiotherapy. However, the value of radiotherapy is ascertained by statistical proof, whereas conclusive evidence of the value of primary surgary is not yet available. The author feels that there is no urgent need for primary surgery in cervical carcinoma in centres where adequate radiotherapy is available.

Carcinoma of the Corpus

Classification:

For clinical purposes only two stages are used: Stage I to include cases in which the growth is confined to the uterus, and Stage II to include cases in which the growth has spread outside the uterus.

Treatment:

Heyman states that in the treatment one must choose among three methods: (1) hysterectomy plus post-operative irradiation; (2) preoperative intracavitary radium followed by hysterectomy; (3) primary intracavitary radium followed by hysterectomy in cases of failure.

Five Year Results:

hysterectomy and post-operative radiation—73.5%.

pre-operative radium and hysterectomy —70.0%.

primary radiotherapy—65.5%.

These figures indicate that hysterectomy followed by post-operative radium is the best treatment, but this series was done on a selected number of cases. The author believes that primary radiotherapy is justifiable where the operators are well qualified.

—Robin Waite, Meds '56

Recent Acquisitions in the Library

The library of the University of Western Ontario contains approximately 49,000 volumes, all fully catalogued, and is regarded as one of the best working collections of its kind in Canada. It is particularly rich in complete files of the more important medical journals. The total number of titles of periodicals, complete or incomplete is nearly 2200, while 740 periodicals and 71 annuals are received currently.

All the books are on open shelves and the students have access to all books and current periodicals. Recent aquisitions by the library are placed on the new-book shelves where they are left for staff and student appraisal and consideration. Of these new books, we have selected the most recent and the most outstanding for your immediate attention.

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