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Primary dysmenorrhea

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PRIMARY DYSMENORRHEA

by

Edward E. Lindell

Senior Thesis

Presented to the
College of Medicine,
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1938

Foreword

In medicine there are numerous subjects whose etiology and treatment are well supported by experimental evidence. Little or nothing is known of other conditions. Between these two extremes are disease entities of which dysmenorrhea serves as an example. Although certain facts are accepted regarding etiology and treatment, the condition has not been well worked out. As a result an inadequate presentation for a working knowledge is given the medical student.

The writers curiosity was aroused long ago, noting the periodic discomfort suffered by female members of the family and schoolmates.

Since entering the study of medicine the great incidence of pain with the menses has impressed the writer and with this a determination to know the cause and treatment of this so-called minor functional ill.

Therefore this thesis concerns itself with that type of functional menstrual pain where no pathology can be found to explain the mechanism. In the literature this entity is termed primary or essential dysmenorrhea.

As the best way to begin it seems that a brief discussion of the normal menstrual cycle followed by a review of what constitutes pain and pain preception is the most logical.

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Physiology of Menstruation

In order to consider the various theories of dysmenorrhea it should be of advantage to briefly review the changes known to occur in normal menstruation. The related clinical characteristics are also mentioned.

In most human females there is a regular periodic flow ordinarily occurring at regular monthly intervals. The most common interval is twenty eight days. The periodicity of the flow of various individuals is subject to wider variation than ordinarily assumed by the laity.

The amount of menstrual discharge varies greatly. The total usually is between five and ten ounces. This discharge is scanty during the first twelve to twenty four hours. It reaches its height the second day and as a rule ceases gradually on the fourth or fifth day. Clots are not present in the normal discharge.

Psychic and physical depression accompany menstruation in most individuals. Nervousness, headaches, languor, backaches, and abdominal discomfort are usual. Definite pelvic pain is experienced by approximately fifty percent of city dwelling American women. Painful breasts associated with menstruation have become much more frequent during recent years(19).

Endometrial Changes

Hitschman and Adler divided the menstrual cycle of a normal woman into four artificial arbitrary stages.

1. Postmenstrual or Regenerative Stage: This stage begins with the cessation of the menses and lasts from four to ten days. An average is six days. The histological changes are a marked formation of the epithelial cells; the stroma cells return to their normal ellipical shape; the blood in the stroma is absorbed and the endometrial glands are short and straight.

2. Interval or Resting Stage: The stroma shows little change; the endometrium increases some in depth and the glands become more tortous. This stage lasts about six days.

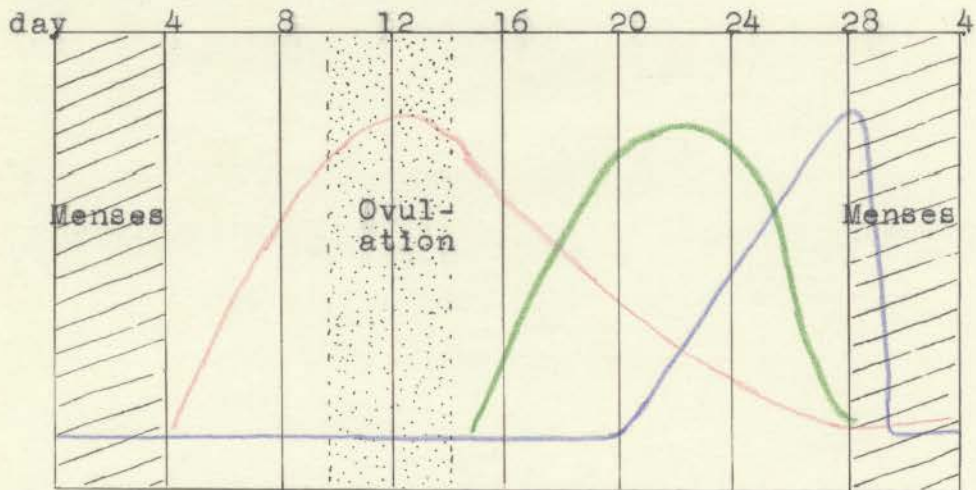
3. Premenstrual Stage: The endometrium thickens, with the glands becoming very tortous and long. Some mucous is contained within their closed cavities. The mucosa differentiates into the layers compactium and spongiosum. Decidual like changes are evident in the stroma cells. This stage lasts about twelve days.

4. Menstrual Stage: During this stage the endometrium breaks down and is cast off. The capillaries and glands rupture with some extravasion of blood into the stroma. The blood, mucous, and endometrial tissue flow into the vagina. The duration of this period averages from four to five days(19).

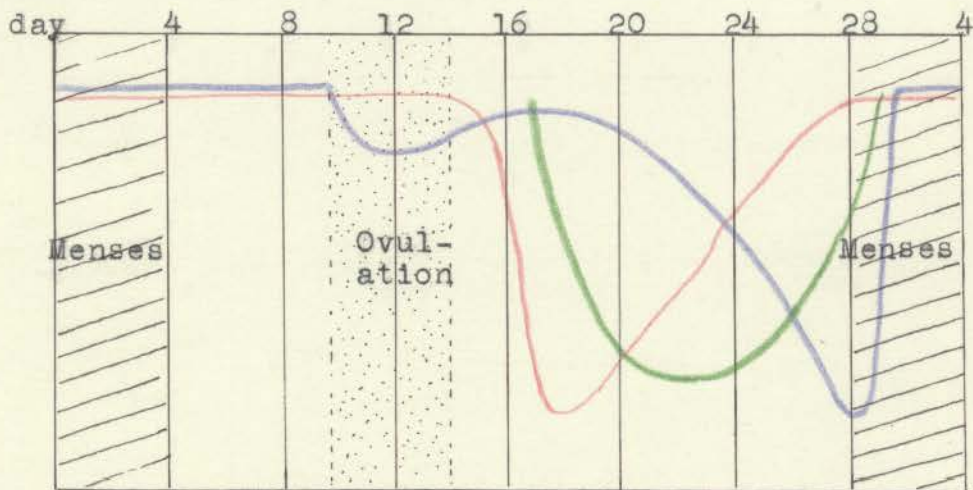
Hormonal Changes

This is best presented by means of the following charts:

Hormone levels in the blood.



Hormone levels in the urine.



Anterior Pituitary Like Substance--Red color

Estrin--Purple color

Progesterin (theoretical)--Green color

Mechanism and Interpretation of Uterine Pain

Anatomy of the Nervous Supply

The nerve supply to the uterus is furnished by that portion of the Abdominal Sympathetic system which lies anterior to the fourth and fifth lumbar vertebra in the interval between the common iliac arteries. It is formed above by the confluence of the intermesenteric nerves. These arise from the solar plexus and the adjacent lumbar ganglia. The presacral nerve descends toward the hollow of the sacrum and each lateral division ends in the pelvic ganglia located on the lateral aspect of the rectum.

The Parasympathetic supply to the pelvic ganglion is furnished by the nervi erigents. These fibers arise from the fourth lumbar segment through the remainder of the cord(10).

From the pelvic ganglion there is a relay of both Sympathetic and Parasympathetic fibers to the uterus, bladder and rectum. The spinal center for the efferent fibers of the Abdominal Sympathetic system extends from the first dorsal to the second lumbar inclusive(10).

Afferent fibers carrying pain stimuli from the uterus travel by way of the presacral nerves. Presacral resection with resulting relief of pain is the basis for this belief(73)(37).

Pain fibers ascend in the spinothalamic tract of the cord and terminate at the thalamus. Between the thalamus and the cerebral cortex there exists a reciprocal connection, of which the cortico-thalamic tract is probably inhibitory in function. In addition, the thalamus is most intimately connected with the afferent head ganglia of the Parasympathetic nervous system situated more basally in the hypothalamus(43).

Pelvic Neuro-Physiology.

In general the Sympathetic and Parasympathetic systems are antagonistic. The functions of the presacral nerves according to Keene(37) are as follows:

1. Transmission of pain impulses. While numerous writers believe this to be true, other writers think the effects gained after presacral resection is more likely due to the removal of the sympathetic influence upon the pelvic blood vessels.
2. Control of uterine function. Wright has shown experimentally the presacral nerve is motor to the circular fibers and inhibitory to the longitudinal fibers of the uterus(74). Cotte(16) observes that presacral resection has no effect on subsequent pregnancies or labor.
3. Control of the bladder. These sympathetic fibers are motor to the internal sphincter, trigone, and ureteral orifices. They are inhibitory to the detrusor

muscles. Pain due to distention or disease in the bladder is transmitted by the presacral nerve.

4. Control of rectal function. The sympathetic fibers are motor to the circular fibers, and inhibitory to the longitudinal fibers of the rectum.

The parasympathetic fibers have exactly the opposite influence on the above organs. Keene(37) says that thermal and tactile impulses along with sensation of fullness are transmitted by way of the pelvic nerves.

Interpretation of Pain.

The central connections between the thalamus, cortex, and hypothalamus serves to explain the intimate relationship between psyche, the autonomic nervous system and pain(43).

Mental states may lead to pain, either through a disturbance of cortico-thalamic inhibition or through projection to a viscus, along the visceral efferent pathway. The algesic counterpart of the disturbed visceral function is then reflected to the central nervous system and registered as pain(43).

McKendree(43) divides pain into two types. The first he calls neural, where there is intrinsic pathology of the pathway. The second type is termed extra-neural, where the nervous system participates essentially as the conductor of pain.

Pain is variable, therefore an individuals statement regarding pain is not reliable. They are prone to be inaccurate and exaggerated (28).

Cannon presents an excellent discussion of pain. He states there is no such thing as imaginary pain; it can never be mistaken for an extra organic reality as is true with hallucinations. Pain may be localized outside the body as seen after occasional limb amputations. Pain obeys the laws of physical causality, while such purely spiritual qualities of mind as willing, knowing, and the like are conditioned but not determined by physical causes. As positive grounds for assuming pain in a physical or quasi-physical reality, Cannon (11) presents the following:

1. Pain is appreciated through a sensory apparatus, the anatomy of which is imperfectly understood.
2. Pain can be localized in space, both somatic and referred pain. Mental phenomena such as emotion and **feeling** cannot be localized.
3. As noted above, the category of physical causality is applicable to pain but not to **feeling** and emotion. Such phenomena depend not on physical stimulus but on the meaning conveyed to our intelligence.
4. Pain is not a quality of inorganic matter. Plants are not known to have pain. Pain is peculiar to sensi-

tive organisms, therefore is not purely physical but is a quasi-physical or psycho-physical reality.

5. Pain, hallucinations of sense, all psycho-physical realities, also the intra-organic realities as hunger, thirst, etc., are so-called tertiary qualities of matter to distinguish them from the secondary and primary qualities. The latter are extra organic, as they are conditioned by organs of sense.

Definition

Dorland(23) defines dysmenorrhea as meaning difficult and painful menstruation. Literally translated it means difficult menstruation and is applied to the condition in which there is pain at the time of the menstrual flow(19). Because of its meaning it is a misnomer and for that reason Massey suggested the term 'menorrhagia'(51). Cannon(11) defines menorrhagia as a symptom of some gross pelvic condition, which is not always possible to recognize clinically. He calls this pseudo-dysmenorrhea.

Medina, a Spanish writer, suggests some other differentiating terms. Algomenorrhea, designating severe menstrual pain in the region of the small pelvis. Exmenorrhea, used to refer to exaggerated discomfort in any other part of the body. Dysmenorrhea means a combination of both algomenorrhea and exmenorrhea(28).

Theobald(68) regards dysmenorrhea as largely, if not entirely, a referred pain in the area of the skin supplied by the first lumbar nerve.

Cannon(11) defines genuine dysmenorrhea as pain intramenstrually, although at times is premenstrual, essentially spasmodic, never a dull ache and occasionally accompanied by spasm of the bladder or rectum.

Historical

Folk lore is full of beliefs about menstruation. These beliefs are found among all groups of peoples, and deal mostly with the uncleanness of the woman during her period, the dangers of contamination by the menstrual flow, or peculiar medicinal properties of the discharge (51).

Pliny states, and it was believed up till the middle of the seventeenth century, that male semen and female menses were the constituents out of which a child was formed (17).

The recognition that pain was associated with the menstrual period dates from early times. Considerable attention was paid to the management of menstrual disturbances as noted in the Ebers papyrus, also in the Brugsch papyrus. These were written about 45 B. C. (5).

Only during the past century has knowledge concerning dysmenorrhea advanced at all. Churchill, 1850 and then Bennett in 1853 described the neuralgic, congestive and mechanical types of pains.

Marion Sims in 1886 insisted that dysmenorrhea was due almost entirely to mechanical causes, and was really only a sign or symptom of some abnormal organic state (5).

During the intervening years much has been written as to the cause and treatment. Considerable debate still exists over the numerous theories.

Incidence of Dysmenorrhea

Although dysmenorrhea is only a symptom, practically it must be considered a disease entity. Menstrual pain is a very common complaint and Curtis(19) lists it present in five to fifty percent of all women. But in a review of the literature many interesting points are found on just what is termed dysmenorrhea.

McKendree(43) states that sixty five to seventy five percent of women suffer from dysmenorrhea and has the following to say of its importance. The disease was considered of minor importance prior to the present activity of women in the business and professional world. It was looked upon as a peculiarity of nature to be endured without audible complaint and treated empirically. Failure meant one to two days in bed and the necessity of devising excuses. In the present era woman cannot jeopardize her economic and social welfare by absences. The problem is to give prompt relief from crippling pain and not to sacrifice her efficiency in doing so, and lastly to offer some hope of a cure.

In a study of University of Michigan students, Bell and Parsons found twelve percent complained of severe dysmenorrhea(28).

Cunningham(18) in a study of fourteen thousand students at the University of California, found that

fifty percent complained of pain with the menstrual period and thirty five percent had severe dysmenorrhea.

Novak(51) in a study of one thousand girls found thirty one percent had menstrual pain with only a little over five percent with severe pain.

Nearly every gynecologist seventy five years ago had some treatment for this condition, so it must have been very common. Wharton(73) thinks that with the more rational and hygienic principles of modern life that the incidence of dysmenorrhea is greatly reduced. He regards an average of ten to twenty percent as given by many authors as high. This view is also held by Clow(13) who reduces the incidence of menstrual pain by seventy five percent by applying principles of good hygiene and exercise.

Barry(3) summarizing a number of studies found that reports of pain with the menses varied from seven to seventy seven percent, and those handicapped by pain varied from two to seventeen percent.

The variation in figures depend largely upon the patients own statements. As stated previously in this paper, pain is variable with different individuals and therefore reliance cannot be placed in an individuals statement(11).

Classification

This is a most difficult matter as there does not seem to be any standard classification beyond that of primary and secondary types.

Novak(5), Hutchinson(35) and others group all primary dysmenorrheas into three types. They are spasmodic, congestive and endocrine.

In reviewing the literature, the primary dysmenorrheas may be classified into the clinical types described by different authors. They are as follows:

1. Mechanical obstruction
 - a. anteflexion
 - b. retroflexion
 - c. stenosis
2. Nervous disturbances
 - a. neurotic
 - b. Autonomic Nervous system unbalanced
3. Congenital Hypoplasia
4. Abnormal separation of menstrual decidua
 - a. membranous dysmenorrhea
5. Allergic
6. Endocrine unbalance
7. Nasal dysmenorrhea
8. Constitutional disturbances
 - a. anemias
 - b. poor physical development
 - c. poor hygiene, masturbation
 - d. low blood sugar, etc.
9. Pelvic congestion

Etiology (Older Theories)

Theory of Mechanical Obstruction

This is one of the oldest theories. It dates from the time of Mackintosh of England who first presented this view in 1832. Marion Sims and Sir James Simpson later strengthened this view, so well in fact that several of the present day textbooks stress this as a factor (51).

The pathology supposedly present was due to an obstruction to the outflow of the blood being brought on by an acutely anteflexed uterus. However, the rate of blood discharged is computed at only two-thirds of a drop per minute (68). Novak observed that at the time of menstrual pain a probe could be passed into the uterine canal without any evidence of obstruction (56). Curtis describes a retrodisplacement dysmenorrhea (19). Bailey (4) also gives support to this theory and Blair-Bell (5) mentions it as a etiological factor.

Contrasted to these opinions are the theories of Novak, Sellers and others. The belief that the theory of mechanical obstruction is unsound is carried out in most of the literature of the past few years and is amply supported by experimental evidence. According to Novak (51) antiflexion was shown by Herman to have no relation to dysmenorrhea.

Neurotic Dysmenorrhea

Menge makes the observation that physiological contractions of the uterus which are not noticed by normal women are felt as labor pains by psychoneurotic women. Another reason for the advancement of this theory according to Menge is the failure to recognize the necessity for a definite nervous apparatus for the production of pain. An idea is entertained that pain can arise in consciousness independent of the afferent impulse which is the ordinary cause. But recent work has shattered this view and has shown that either somatic or visceral pain has its own sensory apparatus and its own appropriate physical stimulus(11).

Aristotle believed the human organism to be an organic synthesis of two fundamental but incomplete substances, namely primal matter and substantial form, which are complementary to each other. The two adapt themselves to one another under the influence of external and internal environment. At a certain level the organism becomes conscious of its own activities and can select its own mechanisms. The mind delegates the execution of many purposes which are carried out blindly and inevitably. This theory of Hylomorphism gives ground that a type of dysmenorrhea may be due to psychogenic causes. Some clinical material suggests this(11).

At the present time the foremost proponents of this theory are two foreign writers, J. Novak and Harnik(57). These men think that all essential dysmenorrheas are neurotic in origin, and that careful investigation will reveal some psychic trauma as a basis for the first attack. This is most usually of a sexual nature. The recurrence of monthly attacks is the result of anxiety and fear, which subconsciously is associated with the menses.

Curtis(19) describes a neurotic dysmenorrhea, with symptoms similar to the congenital type, although the pains are less paroxysmal and more cramplike. Nervous signs are more frequent. Masturbation is suggested as an not infrequent cause.

Novak(51) in one of his more early papers, listed three subdivisions of neurotic dysmenorrhea. The first he called hysterical dysmenorrhea, and should never be diagnosed in the absence of the earmarks of hysteria. A second type, neurasthenic in origin, he subdivided into primary and secondary groups. The primary group resulted from heredity and environmental factors; the secondary group was due to some chronic disease that undermined the whole nervous system. The third type was termed neuralgic dysmenorrhea, the existence of which Novak doubted.

Reynolds and Novak (55) agree that many cases are explained on a psychogenic basis, but that the majority have other causes. Sellers (66) agrees that the neurotic factor is infrequent, but is important. He finds although some women magnify the pain, others suffer so that the nervous system is undermined and a neurosis develops.

Barry (3) speaks of this neurotic type as a fault of heredity or victims of suggestion from mothers who are too solicitous. Stacy and Shoemaker (67) believe nervous instability and a lowered tolerance to pain may be an etiological factor in aesthetic persons.

Kotz and Parker (40) although classifying neurosis as a cause regarded few if any cases as being really due to that condition. Cannon (11) thinks the whole idea is wrong because it is based upon the false theory of psycho-physical parallelism. He suggests that psychogenic factors may aggravate the pain but states there is no direct evidence yet available of a causal relation.

Congenital Hypoplasia

A familiar triad of presenting symptoms are sterility, dysmenorrhea and genital hypoplasia. This clinical entity is characterized by pains, cramplike in character, appearing with the first menstrual period and recurring at each succeeding period. Examination reveals genital hypoplasia. The uterus is of infantile form, the fundus

small and sharply bent at the cervix. The cervical canal is usually tight and may be strictured. Hypoplasia of the ovaries is usual(19). According to Israel(36) Schulz in 1903 first advanced the theory that the dysmenorrhea was the result of the uterine hypoplasia.

Cannon(11) states there is a steadily growing view that hypoplasia is responsible for some dysmenorrhea. But it has been noted that some women with markedly hypoplastic uteri have no pain at the time of menstruation. The chief evidence against this theory is that primary dysmenorrhea is often an acquired disturbance. Novak(51) supports this with an observation of one hundred cases of dysmenorrhea, in which menstrual pain appeared long after puberty.

Little is known of the production of the dysmenorrhea of this type. Novak(51) found such uteri contain a preponderance of connective tissue. Because of the deficiency in muscle tissue, there is a stagnation of blood in the uterine veins and this venous stasis causes pressure on the uterine nerves. Menstruation is usually scanty because there is a physiological as well as an anatomical deficiency in the uterus. But dysmenorrhea is more frequent in milder grades of uterine hypoplasia, while in severe grades amenorrhea is the rule.

Barry (3) speaks of the pain in this type of dysmenorrhea as being due to the obstruction and swelling of the endometrium, which acts as a foreign body. Schulz (62) believes the pain in a hypoplastic uterus is due to the failure of myometrium to empty the uterine veins during the physiological hyperemia accompanying menstruation.

Blair-Bell (5) thinks hypoplasia of the uterus is an important factor in causation of pain. However, they do not think excessive exfoliation gives rise to pain. But when such a condition is present, the pain is due to the association of intrauterine clots with membranes forming a compact mass.

Hutchison (35) simply calls this type of dysmenorrhea the spasmodic form. He finds this more common in nervous women and he thinks it is due to genital hypoplasia, spasm of the internal os and a general hypersensitiveness.

Ehrenfest (28) has this to say. The gynecologist must realize the combination of dysmenorrhea, sterility and uterine hypoplasia does not necessarily indicate a causative relationship between these conditions. The reason being that the patient has a tendency to overemphasize any menstrual anomaly because in her own belief it plays a significant role in her inability to become pregnant.

Nasal Dysmenorrhea

Fleiss(31) described this type of dysmenorrhea in 1893. He described the genital spots in the nose. These consist of erectile tissue situated on the anterior end of the inferior turbinate and on the tubercle of the nasal septum. During menstruation, in some women, these spots become swollen, congested and hypersensitive. Fleiss describes two types of dysmenorrhea, one in which the pain disappeared with the appearance of the menstrual flow, the other in which the pain continued.

Schiff(61) confirmed the above observations and went as far as to state that seventy two percent of all types of dysmenorrhea yielded to the treatment for this type.

Various writers have waxed enthusiastic over nasal dysmenorrhea as an etiological factor, but during the past few years it has been less advocated. Novak(51) mentions that a biological and possibly physiological connection between the reproductive organs and the nose has long been recognized. This relationship is strengthened by the frequent occurrence of nasal bleeding as a vicarious phenomenon at the menstrual epoch. In a later paper(66) the endocrines are suggested as the connecting link to explain nasal dysmenorrhea. As another possibility, the sympathetic nervous system must be considered.

Curtis(19), Barry(3), Mayer, Brettauer, Kuttner and others, regard nasal dysmenorrhea as an existing type. Kuttner is somewhat dubious as he gets the same effect treating the patients with menthol and other substances. He believes the effect is largely due to the psychic influence on the patient(51).

An interesting suggestion as to the possible relationship between allergic dysmenorrhea and nasal manifestations has been presented by Schwarz and Smith(63).

Etiology (Modern Theories)

Abnormal Separation of the Menstrual Decidua

Whitehouse(74) observed that abnormal separation of the menstrual decidua was associated with dysmenorrhea. The amount of pain is proportional to the size of the menstrual fragments.

A modification of this theory is offered by Blair-Bell(5). He regards the dysmenorrhea as due to the expulsion of blood clots and that the decidual fragments are only causally related to pain. Cannon(10) points out that blood clots are found in forty percent of women with symptomless menses. Premenstrual and intramenstrual pain suggests that the pain is due to separation of the decidua rather than the expulsion of blood clots. Cannon believes this is fundamentally due to an over activity of the lutenizing hormone which causes an corresponding increase in the activity of the corpus luteum. No experimental evidence is presented to support this view.

The writer suggests that some support is offered by a short consideration of of membranous dysmenorrhea. This is a condition where the complete uterine mucosa or large pieces may be cast off at the monthly period(19). Cramplike pains are usually present, but some patients

pass large membranes without notable discomfort. The condition is not common. Some cases are believed to be on an infectious basis. Cotte(14) thinks the more common etiological factor is mucosal hyperplasia caused by endocrine disturbance.

Endocrine Inbalance

A modern theory favorably accepted has been presented by Novak and Reynolds(55). In certain women there may be an endocrine inbalance between the uterine motility factors, especially the folliculin and progestin. Reynolds(60) has shown in a set of experiments upon female rabbits that folliculin alone is responsible for uterine motility. The immediate cause of primary dysmenorrhea is a spasmodic contraction of uterine musculature which is the result of the withdrawal of progestin activity with an ascendancy of folliculin. Barry(3) states folliculin or theelin as it is often called, reaches its greatest concentration in the blood stream during the premenstrual and early menstrual phase. According to Novak and Reynolds the inhibitory influence of progestin is removed a day or two before the menstrual onset and its withdrawal produces dysmenorrhea in some women. Such predisposing factors as constitutional subnormality

or psychoneurosis may play their part. In other cases there is an actual imbalance between theelin and progestin, either quantitative, chronological or both.

Experiments have not satisfactorily proven this theory but Cannon(10) thinks that the explanation is logical and fits certain cases of dysmenorrhea of a spasmodic type. The therapeutic success of corpus luteum and pituitary hormones only proves the immediate cause of the dysmenorrhea is a spasmodic or disordered action of the uterine muscle. Davis(21) agrees as to the effect of the hormones. He adds that the most likely hormonal imbalance is due to a progestin hyposecretion caused by too rapid and complete degeneration of the corpus luteum. Premature progestin withdrawal could well account for the onset of dysmenorrhea several days before the usual menstruation.

Davis(21) writes that the gonadotropic hormone of the anterior lobe of the pituitary also has an inhibitory effect on uterine contraction. However no changes of this substance in the blood or urine are found in cases of dysmenorrhea. The secretion of the posterior lobe of the pituitary is oxytocic and this possibility as a cause of spasmodic dysmenorrhea must be kept in mind. But again there is no proof of concentration of pituitrin in the blood or spinal fluid. Thyroid and

thymus hormones excite uterine contractions experimentally but are not verified clinically. Parathyroid deficiency has been suggested as a possibility but again no diminution in the serum calcium has been shown.

Witherspoon(75) notes that the pain begins at the theoretical time the lutein body begins to degenerate and the follicular hormone stimulation again becomes dominant. He explains the pain as due to the withdrawal of progestin influence. Kotz and Parker(40) are inclined towards a view that the dysmenorrhea is due to secondary ovarian failure brought on by a hypofunction of the anterior pituitary gland. An excess of theelin and a deficiency of progestin result and this disturbed ratio causes excessive uterine contractions.

Lackner(41) although agreeing with Novak that endocrines play a part in etiology does not believe that the cause of pain is related to uterine contractions. Relief of pain following endocrine therapy does not necessarily mean a lessening in size or intensity of contractions of the uterus.

Arguing that a corpus luteum deficiency would have to express itself in a formation of endometrium, Israel (36) points out that Mazer found in a study of twenty women with typical dysmenorrhea, fourteen showed by microscopical study a typical and normal progestin phase of the endometrium.

Many other investigators and clinicians have reported on endocrines as a cause of dysmenorrhea. Essenson (29) is convinced that all primary dysmenorrheas are due to endocrine disturbances. He bases this belief on the results of treatment with hormonal products. Curtis (19) cites the work of Novak and Reynolds in his new book on gynecology. Hutchinson(35) suggests that the endocrine disturbance may be a hypersecretion or hyposecretion of the ovaries. The former would result in a vagatonia, giving the symptoms of lowered blood pressure, lowered basal metabolic rate, increased sugar tolerance, uterine cramps and increased uterine bleeding. Hyposecretion of the ovaries causes a predominance of sympathetic tone. The symptoms are opposite to the ones mentioned above except for the presence of uterine cramps. Disturbed ovarian function causes a more rapid coagulation of the blood, which causes clots and so results in cramps(35).

Autonomic Nervous System Dysfunction

This theory has been advanced by several authors. It most probably is based upon the work of Cotte(14)(15)(16). Cannon(10) favors this as an etiological factor in that type of dysmenorrhea associated with vesical and rectal disturbances. He regards an overactivity of the

of the abdomino-pelvic sympathetic nerve as the cause of the dysmenorrhea. Support to this view is given by citing cases of intractable dysmenorrhea cured by an appropriate sympathectomy.

Davis(21)(20) suggests that an exaggeration of sympathetic activity might lie in a pathological alteration of the nerves to the uterus, thus modifying the impulses. Davis agrees with Cotte, Cannon and others that spontaneous rhythmic uterine contractions can take place independent of the nervous systems. Nervous influences play an important part in the regulation of uterine activity. The parasympathetic and sympathetic systems are antagonistic and normally in balance, but an unbalance could result in exaggerated uterine contractions. This writer made microscopic studies of the presacral nerves following presacral resection for dysmenorrhea. In seventy percent of these cases alternation of the nerve was found in some degree, in the direction of a subacute or chronic neuritis. Davis regards this neuritis of primary importance in the cause of dysmenorrhea, and that the impulses causing the menstrual pain are initiated or at least exaggerated by these abnormally sensitive nerves. Cotte(14) has shown in a series of sixty presacral resections, twenty two with definite

inflammatory changes in the nerve trunk, fifteen with perineural inflammation, eight with neural sclerosis and two with neuromas. Bakscht(28) has also reported finding inflammatory changes in these tissues. He regards these as a likely etiological factor. Kennedy (38) mentions degenerative changes found in Frankenhauser's ganglion which he thinks are brought about by a deficiency of estrin.

Whitehouse(74) while observing a human pregnant uterus injected novacaine in the spinal canal. Within five minutes the uterus contracts and does not relax until the drug ceases to act. This cuts off the parasympathetic fibers and gives the sympathetics predominance.

Cotte(16) attributes dysmenorrhea associated with rectal and bladder complaints, to a disfunction of the autonomic nervous system. A hyper-excitability is responsible for the symptoms. Some cases can be explained by very slight injury to the genital organs or remote pelvic lesions, which provoking by either reflex or direct stimulation, sensory, motor or vasomotor disturbances in the pelvis and cause pelvic neuralgia, dysmenorrhea, cystalgia or rectal symptoms. Slight anatomic injury of the ovary or endocrine glands connected with it, can initiate by reflex or endocrine stimuli,

vicious circles which continue to persist until they can be interrupted. A primary disturbance of the pelvic sympathetic system or of the nerve centers connected with it can lead to the same results, Keiffer(39) thinks that an abnormal nervous state results in a cervical spasm and so causes pain. Bloss(6) offers some support to this view.

Although viewing autonomic imbalance as a possible cause, Israel(36) believes most dysmenorrheas to be a local manifestation of a constitutional disease. Lackner(41) thinks the degenerative changes presented by Kennedy responsible for certain cases of dysmenorrhea. Doderlein(22) suggests a hyperesthesia of the uterine mucosa as the basis for many cases of dysmenorrhea.

Allergic Dysmenorrhea

Allergy has recently been advocated as an etiological factor in primary dysmenorrhea. Schwarz and Smith (63) made a study of thirty five cases. In twenty six a positive history of asthma, hay fever or food sensitivity was obtained. Twenty one of these patients had gastro-intestinal distress with certain foods, and six more gave nasal symptoms. By means of the scratch test the foods most frequently giving positive reactions were determined. These were milk, chocolate, fish, beef,

pork, nuts, beans, pepper, cabbage and cauliflower. By elimination diets twenty of the thirty five patients had complete relief from their dysmenorrhea, and nine partial relief. Four of these nine were relieved by the addition of endocrine therapy.

Rowe(59) confirmed the opinion that menstruation can be disturbed and made painful by food sensitization. Dutta(25) thinks it reasonable to believe that menstruation may precipitate an allergic attack in an allergic diathesis. A certain porportion of cases of dysmenorrhea therefore, are on an allergic basis. But he is undecided whether this is a common or rare cause. The allergic process is helped during the menses by the destruction of the mucosa or by the aggravated bacterial action in an infected uterus. The uterus itself may be looked upon as a possible site of allergic spasm producing dysmenorrhea. Dutta likens this to the spasm seen in bronchial asthma.

In a recent book on clinical allergy, Tuft(70) writes that the cause of dysmenorrhea may be allergy, but only in exceptional cases will this be true. In intractable cases allergy must always be considered. This view of the subject is strongly endorsed by Kotz and Parker(40).

Constitutional Factors Producing Dysmenorrhea

1. Secondary Anemia

This is listed as a cause by Curtis(19) and Sellers and Saunders(66). In these cases the red blood count is between three and four and one half millions. The hemoglobin varies from sixty five to eighty five percent.

2. Low Blood Sugar.

Tedstrom and Wilson(28) observed that in every case where the fasting blood sugar was below eighty milligrams percent, the patient complained of menstrual pain or nervousness a few days before the flow. By increasing the carbohydrate in the diet a week before the menses eighty percent of these patients were relieved. These writers also claim that this same food regime relieves women with normal blood sugar levels.

Interesting is a report by Altschul(1) who has treated a limited number of patients with insulin. He reports that eighty percent obtained relief within an hour following treatment.

3. Poor Posture. Lack of Physical Developement.

Adams(28) is a believer that faulty posture, with marked lumbar lordosis and thoracic kyphosis, flattening of the abdomen and enteroptosis probably cause pelvic

venous congestion. This results in a dysmenorrhea. He suggests correction of the posture for relief. Bailey (4) regards the pelvic congestion resulting from poor posture as due to lack of intra-abdominal pressure which normally is great enough to support the thin walled valveless veins. Miller(48) in a four year study of young college^{women} concluded that there was absolutely no relationship between posture and the incidence of dysmenorrhea.

4. Exercise

Clow(13) regarded most dysmenorrhea as due to improper exercise and hygiene. By correction of these factors she reduced the incidence of dysmenorrhea in a series of cases by seventy five percent.

Miller(46) was impressed by the result of directed physical exercise. among a group of young college women. He believed the reduction of dysmenorrhea to be more than a mere coincidence.

5. Miscellaneous

Hutthinson(35) calls this general group a congestive type of dysmenorrhea, which he says results from sedentary living, constipation, masturbation, coitus interruptus, psychosexual excitement, exposure to cold, etc.

Chronic constipation was given as a cause for some cases of dysmenorrhea by Sellers and Saunders(66).

Boynton and Hartley(7) believe that a calcium deficiency has some relationship, or is responsible for dysmenorrhea. But in a previous paragraph mention was given to the fact that serum calcium diminution has not been found in cases of dysmenorrhea(21).

Cunningham(18) makes the observation that the women who have menstrual pain are usually underweight, have a low blood pressure and are farsighted.

Pathology

Not very much reliable material is found in the literature.

Cotte(14) summarizing the pelvic findings in one hundred cases where he performed presacral found the following:

Ovaries, sclerocystic	44
Normal pelvis	19
Infantile uterus	11
Retroflexed uterus	10
Large congested uterus	8
Congested pelvis	8
Anteflexed uterus	6
Serous fluid in pelvis	5
Large cystic ovaries	4
Hematoma or prolapsed ovaries	4

In general the uterus was smaller than normal. It was retroflexed as often as anteflexed.

Davis and Moir(21) points out that during normal menstruation the blood seeps gently through the cervix in an even continuous flow, while in dysmenorrhea it escapes in diminutive gushes, literally being pushed out by each uterine contraction.

Definite inflammatory changes within or surrounding the presacral nerve has been shown by Cotte(15)(16) and Bakscht(28). The degenerative changes in the ganglion of Frankenhauser found by Kennedy(38) have been previously referred to in this paper.

Cause of the Pain

The general agreement is that excessive muscular contractions produce spasmodic visceral pain. This suggests spasmodic or disordered contractions of the uterine musculature as the cause of menstrual pain(11). Moir(21) cites experiments performed upon women, proving an exaggeration of uterine contractions are coincident with individual spasms of pain. Other investigators have shown the same contractions to be present in laboratory animals.

Theobald(68) thinks that the pain is produced by stimulation of the autonomic nerve endings in the cervix, and this causes a referred pain in that area of the skin supplied by the first lumbar nerve. He produced such a pain by the application of a silver nitrate stick into the cervix of dysmenorrhoeic patients, and states that the pain was identical to that experienced during menstruation. This pain could be relieved by local anaesthesia.

Kennedy(38) cites authorities and proofs that the uterine nerves are under control of the ovarian hormones. He reasons that the removal of ovarian influences suppresses uterine activity through degeneration of the ganglia, then a perversion of the ovarian hormones would adversely affect the ganglia and through them the uterus

and so result in pain.

Moir(21) suggests that the pain might be due to a relative ischemia of the uterus, similar to anginal pains of the heart or intermittent claudication of skeletal muscle. He found the greatest pain at the height of each contraction of the uterus, and a disappearance of arterial pulsation at the peak. Lackner et. al.(41) was unable to confirm these observations

Fekete(30) thinks that the pain is more likely due to compression of engorged endometrium within the rigid walls of a small uterus. In a similar manner pain is produced by an internal uterine endometrosis through distention of endometrial glands which are lying deeper in an otherwise normal uterine wall. Pain will also appear as a result of compression of the engorged endometrium in cases where the uterine muscle is in a state of high tonicity due to increased irritability both of the muscle and of the blood vessels as a result of disturbed hormonal balance.

Symptoms

Spasmodic Group

This entity is characterized by severe cramp-like pain, comparable to labor pains, appearing with the first menstruation at puberty and recurring at each succeeding period. The cramps begin several hours to a day previous to the onset of menstruation. The intensity of the distress varies considerably. In typical cases the flow is scanty. The cramps come in paroxysms lasting up to two or three minutes. Headache, general malaise, irritability, back pain, etc., usually accompany the other symptoms to a variable degree. Often associated complaints are sterility and congenital hypoplasia(35)(36). Novak(51) regards this type most common among primary dysmenorrheas.

Congestive Group

An increase in the usual discomfort of the period begins several days before the onset of the flow. This continues throughout the period or may be relieved by the establishment of menstruation. The pain is dull, dragging in character, extending to the back, sides and down the thighs. Often the pain is accompanied by headache, lassitude and nervous excitability(35)(51). This type is more common in the secondary dysmenorrheas (51).

Treatment

In as much as there apparently is more than one cause of dysmenorrhea, a good routine of study is necessary for the purpose of diagnosis and classification of the case. Kotz and Parker(40) use the following outline.

1. Complete history and physical examination.
2. Vaginal, or in the virgin a rectal examination.
3. Urinalysis.
4. Hemogram
5. Basal Metabolic Rate.
6. Hormone study.
7. Blood chemistry in selected cases.
8. Radiograph of the sella turcica in selected cases.

With the establishment of the suspected cause an intelligent approach may then be made therapeutically.

Prophylaxis and Hygiene

1. Pregnancy and Sex Relations.

A general belief among many medical men is that a full time pregnancy will cure most cases of dysmenorrhea. Novak(51) writes that the marriage relation stimulates the development of the uterus and as a result the dysmenorrhea ends. But Curtis(19) makes the observation that the onset of dysmenorrhea may date from the first menstrual period after the birth of a child. Sellers (65) and Emge(27) both question whether marriage or the sex relation gives any relief from dysmenorrhea. Blair-

Bell(5) states that child bearing has less influence on dysmenorrhea than is so generally believed.

2. Hygiene

A general accord of opinion is that good habits of living, namely sleep, proper diet, and a definite amount of physical exercise are beneficial in most cases of dysmenorrhea.

Clow(13) reduced the incidence of dysmenorrhea by seventy five percent by the following method of treatment.

1. Examination to find there was no evidence of disease or anomaly.
2. A bath, at the beginning of the period, hot enough to flush the skin and continued daily throughout the period. This is more efficacious than hot water bottles.
3. Daily exercise begun some days before and continued throughout the period. An exercise that diverts the mind is good. This must be vigorous enough for a healthy glow.
4. A mild analgesic was prescribed during the first three periods for nervous women.

Sellers(66) agrees that outdoor sports, exercises and special exercises are beneficial but states that in certain groups such as students and teachers at the end of a school year, they are definitely contraindicated.

Barry(3) suggests the careful regulation of the bowels three to seven days before the onset of the menses is valuable. A change in occupation may also be beneficial in certain cases.

Curtis(19), Bailey(4) and other writers emphasize the value of hygienic measures, irrespective of the type of dysmenorrhea. Avoidance of mental and physical fatigue, an abstemious diet several days prior to menstruation, with careful avoidance of constipation, are the measures advocated by Curtis. He believes these tend to decrease pelvic congestion.

3. Heat

The beneficial effects of heat and rest are well known and widely used. The hot water bottle is the most common method of applying heat. Hot sitz baths must be employed judiciously. Gin and alcohol should not be used(19).

Medicinal Treatment

This subject will be considered under a general heading of the types of drugs most widely used.

1. Antispasmodics

a. Atropine: Novak(51)(50) first advocated this method of treatment. He suggested its use in young unmarried women with the spasmodic type of dysmenorrhea. Atropine sulphate is used, the dose to be varied with the patient. The ordinary dose was 1/100 grain t.i.d., and pushed to the point of saturation. Medication was

started two days before the onset of menstruation and continued until the second or third day, depending upon the pain. Analgesic drugs may be used also. In cases with considerable nausea and vomiting, the drugs may be given rectally. Cannon(11) regards this as a satisfactory form of medical treatment. His only variation from Novak's regime is an increase in dosage to 1/120 grain every four hours. Bailey(4) uses the treatment outlined by Novak, which he augments by bed rest and heat.

b. Benzyl Benzoate: Litzenberg(42) in 1919 reported better than sixty percent success using this drug. He used an emulsion with the doses ranging from two to eight c.c. every four hours. The emulsion is made up as follows:

Benzyl benzoate	10.0
Mucilage of acacia	5.0
Aromatic elixir of eriodictyon	35.0

Macht states the advantage of this antispasmodic is its non-toxicity. Barry(3) believes the drug to be worthy of consideration. Stacy and Shoemaker(67) writes this emulsion will give relief in fifty percent of the cases. Novak(51) describes this form of treatment.

c. Humulus Lupulus Compound: In an enthusiastic article McKendree et. al.(43) reports on the action of

of this substance. Axinn of Vienna observed that beer had a tendency to lessen menstrual pain. The active principle of the beer is the humulus lupulus in the hops. According to McKendree et. al., this drug has a selective sedative effect on the uterine musculature and is to be preferred to an analgesic which depresses the impulse somewhere along the tract or in the brain cortex. This sedative effect causes a relaxation of the spastic uterus with a resulting relief of pain. These authors claim the following advantages:

1. A selective and sedative action on the uterus.
2. It does not lower the patients resistance.
3. There is a wide margin of safety in dosage.
4. The patient may go about her duties or social engagements without interruption.
5. The drug gives prompt relief.

The drug should be given the patient several days prior to the onset of menstruation.

2. Analgesics

Many forms of sedation are used as a palliative measure, usually in conjunction with heat and rest. Only a few of the more commonly used drugs will be considered.

a. Bromides: These drugs are recommended by Curtis (19). Myers(45) likes this form of medication for the milder types of pain. He uses five grains t.i.d., of sodium bromide combined with the same dosage of potassium

iodide. For more severe pains Myers suggests the use of veramon, six to ten grain doses at four to six hour intervals.

b. Barbiturates: Curtis(19) suggests the use of an alcoholic solution of phenolbarbital three days before the onset of pain. Sellers and Saunders(66) use barbiturates combined with codeine. Barry(3) reports excellent results using one and one half to three grains of sodium amytal every six hours. He also likes a combination of three grains of antipyrine and ten grains of aspirin every four hours. Novak(51) likes the following :

Acetphenetidn	4.0
Caffeine	0.4
Sodium bicarbonate qs a.d.	60.0

M.et. divide in chart. No.XII

Acetanlid and antipyrine are also of value according to Novak.

c. Codeine: This drug is lauded by Curtis(19) who wonders why it is not more widely used for dysmenorrhea. He states there is no danger of habit formation. Sellers and Saunders(66) regard it as very useful. Novak(51) believes it permissible in very severe conditions.

d. Opiates: Opium is effacious but should rarely be used(19). The drug is too dangerous from the standpoint of habit formation(19)(51)(66)(3)(4).

e. Miscellaneous: Many other drugs are used of a

sedative nature. Among them are dilaudid, guaiacum, belladonnae, apiol, elixir viburnum, hydrastis, cannabis indica, pulsatella, gelsemium, piscidis erythema and many others(51).

3. Miscellaneous Medical Measures

A high carbohydrate diet for a week before the onset of the flow is advocated by Tedstrom and Wilson. These authors feel that a low blood sugar is connected to the etiology(28).

Altschul(1) treats his patients with insulin. This is given three to seven days before the onset of the menstrual period. He reports relief in eighty percent of his cases.

Boynton and Hartley(7) use calcium therapy. Their routine consists of giving sixty grains of calcium gluconate daily during the ten or fourteen days preceding the onset of pain, and continued throughout the first two days of the period. In addition, viosterol was given to some patients. The dosage was thirty drops daily. The symptom of bruising easily seems to indicate a more favorable response to this type of therapy.

According to Curtis(19) potassium iodide is an old and time honored remedy.

Surgical Treatment

1. Cervical Dilation.

Mackintosh in 1832, basing his therapy on the theory of mechanical obstruction, dilated the uterine canal in a number of cases and cured their dysmenorrhea. This method of treatment was strengthened by Sims and Simpson. It is still one of the most popular forms of treatment, usually only giving temporary relief. An occasional permanent cure is reported(51).

Curtis(19) in his new textbook, states that occasional office dilation will relieve those patients with congenital anteflexion who have moderate dysmenorrhea. More severe cases require dilation before each menstrual period.

Cannon(11) is a bit more skeptical. He agrees that many are cured, a porportion are relieved, but that in a good many cases no relief is obtained. The Hegar dilator is used and the cervix dilated up to about number fourteen.

A. Davis and C. Moir, et. al. (21) regard simple cervical dilation combined with alcohol injection as the best form of minor curative procedures.

Excellent results were reported by Barry (3) who used the following technique. Preliminary dilation with the Hegar dilator, multiple longitudinal incisions

through the internal sphincter, then packing the cervix with iodoform gauze for five days. There is very little chance of infection if the uterine cavity is not curetted. According to Barry, Dewees in 1826 performed this operation as a cure for dysmenorrhea.

Miller(49) thinks this method gives the highest percent of cures. He states that thirty percent receive temporary cures and forty percent are permanently cured. This treatment is indicated in cases where the uterus is the typical anteflexed hypoplastic type, and where there is no other pelvic pathology present. Bailey(4) regards dilation only as a method of temporary respite.

Blair-Bell(5) states that dilation is not recognized as the proper treatment for intrinsic dysmenorrhea in the present state of knowledge, but is of value in the congenital retro-displaced uterus. Cotte(16) thinks that clinicians are not justified in assuming results from dilation of the cervix. He states that one may dilate the anus with as good or better results.

3. Stem Pessaries.

Curtis(19) favors dilation, then the insertion of a cervical stem pessary. This should remain in position for at least six weeks. The possibility of infection is mentioned. Myers(23) uses the cervical pessary in

in cases of acute anteflexion. Sellers(65) regards stenosis and flexion as a cause of dysmenorrhea, and treats these cases with prolonged dilation by the use of a pessary or a uterine pack.

The danger of infection has led Novak(51), Barry(3) Christopher(12) and others to object to the use of these devices. Christopher is very strong in condemnation of the use of cervical pessaries "as a beneficial measure to dysmenorrhea patients". Infection too commonly arises about these stems.

4. Alcohol Injection.

Blos(6) first advocated this method of treatment. Recently it has been brought to the fore by Davis(20). Curtis(19), Emge(27), and others feel that this treatment is logical, although several point out the danger of necrosis and infection. Davis(20) regards the danger of necrosis a remote possibility although he admits larger amounts of alcohol might damage the motor nerve to the bladder. Bailey(4) strongly condemns this measure as a method of general treatment.

The object of this treatment is to block the nerve pathway to the uterus at the nearest accessible point, namely the plexus of Lee Frankenhauser, where the uterine nerve supply is most concentrated. The technique is

as follows:

The patient is placed in the lithotomy position, then under Evipan anaesthesia the cervix is pulled to the left and the right fornix is exposed with a flat retractor. Pass a long graduated Gasserian needle into the side of the cervix longitudinally one half centimeter then remove the retractor. The needle is then passed backwards and outward one and one half centimeters at an forty five degree angle to both the sagittal and coronal planes. The needle is guided by the finger in the rectum. One cubic centimeter of eighty five percent alcohol is injected. The procedure is repeated on the other side.

Blos reports ninety percent success with this method of treatment.

5. Presacral Resection.

Cotte (16) (14) (15) attributes dysmenorrhea to a disfunction of the autonomic nervous system and a hypercitibility is responsible for the associated symptoms. Resection of the hypogastric nerves is performed to correct this condition. This operation is indicated only in those cases of severe dysmenorrhea not relieved by more moderate measures and where there is no demonstrable pelvic pathology. Cotte gives his mortality

rate as one out of one hundred. Out of three hundred cases which he has operated on, only two received no relief andⁱⁿ both cases there was other pathology present which could account for the pain. Reasons for the effectiveness are not given but the theories are (1) suppression and interruption of abnormal sensory motor reflex (2) modification of the pelvic circulation. The operation does not interfere in any way with subsequent pregnancies.

Cotte thinks the cases responding most favorably to this treatment are those where the pain is located in regions corresponding to the final distribution of the presacral nerve. The spasmodic pain comes on a few days before or with the onset of menstruation. Its point of maximum intensity is in the uterus, radiating to the sacrum or coccyx and often accompanied by bladder irritability. It is important to differentiate from dysmenorrhea indicating involvement of the ovarian sympathetic plexus. This type is where the pain is located in one or both sides of the pelvis, radiating to the lumbar region.

Curtis (19) gives a general description of the technique. After the abdominal incision and pelvic exposure the sigmoid bowel is drawn to the left. A midline incision of the peritoneum is made over the

fifth lumbar vertebra, the nerve is located in the retroperitoneal areolar tissue and is isolated by passing an aneurysm needle beneath the nerve. Bifurcation of the nerve occurs as it passes over the promontory. About one half inch or more is resected and the incisions are then closed.

Keene (37) warns that occasionally the root of the mesocolon may extend across the interiliac space and here the incision is made to the right of the midline. The exposure of the nerve is rather difficult and hazardous in these cases because of the vascularity.

Cannon (11) and Davis and Moir (21) feel that it is the most certain therapeutic measure that we have, but that in general it should be used only as a last resort. Other authors reporting favorably on this method of treatment include Keene (37), Ehrenfest (28), Sellers and Saunders (66), Adson and Masson of the Mayo Clinic, Fontaine and Herman, Barry (3), Schaul, Wetherell, Baly and Cannady, Counsellor and Winchell and others.

Endocrine Therapy

This is the more modern trend based on the work of Novak and Reynolds. Here we have innumerable reports of various methods using many endocrine products. The general trend in the literature is that there is some value in this form of treatment in certain cases, but that it has not been very well worked out.

In an article on the uses and abuses of glandular products in gynecological disorders, Novak (54) states the probable important factor in some cases is hormonal. But he warns that the giving of any ovarian preparation seems illogical, because it is impossible to figure theoretically how it might help. They may even aggravate the pain, because research has seemingly proved estrin increases the contractibility of the uterus (60).

1. Corpus Luteum Hormone

Cannon (11) feels the therapeutic results of this hormone are poor. However the time of medication may be an important factor. Ehrenfest (28) points out that most of the marketed preparations suggest by their name that they contain corpus luteum hormone, when in reality they contain the anterior pituitary luteinizing factor. This substance was used by Witherspoon (75) (76). He gave 1 cc. of Follutein three to four days before the beginning of the flow and continued the first two days of the flow. Seventy per cent showed a satisfactory response.

Proluton and progestin is used by Kotz and Parker (40). This may be used as a temporary measure and give relief but will not be a permanent cure. They administer one international unit every other day, beginning about two weeks before the expected period. In severe cases they used five units and continued throughout the period.

The schedule was repeated for three months. The patient cannot expect results until the third period after the starting of treatment.

Lackner et al. (41) thinks there are three types of dysmenorrhea patients. First, those who will receive benefit from corpus luteum therapy are patients with well developed uteri having large to moderate contractions. The second type will not respond to hormonal therapy. Estrin is used on the third type who have small hypoplastic uteri.

Campbell and Hisaw (9) like corporin, and state it can be used in selected cases with excellent results. Elden and Wilson (26) using progesterone on seventeen patients obtained complete relief in fifty per cent of their cases. However, they state that in two cases the dysmenorrhea became worse, which would suggest the cause was a deficiency of theelin rather than progesterone.

Israel (36) argues against the use of corpus luteum therapy. He suggests that the indication for it would be a deficient formation of endometrium.

Another endocrine preparation used is that made from the anterior lobe of the pituitary gland. This most commonly goes under the name of Antituitin S. Essenson (29) uses this product combined with estrin and feels that he gets good results. Barry (3) also

favors the use of Antituitin S, which he feels is the most logical form of endocrine therapy. But he does not believe that his form of therapy is altogether satisfactory.

Novak (52) recommends for temporary relief, one hundred rat units of Antituitin S intramuscularly. This is given one to two days before the onset of menstruation. He uses Parke Davis and Companies product. Theelin is used for permanent relief. Bailey (4) follows Novaks line of treatment. He regards this as the most promising therapy we have at the present.

Kotz and Parker (40) question the value of any anterior pituitary product now on the market. They are more in favor of stimulating the uterus by means of roentegen therapy.

2. Emmenin

Emmenin is an extract of placental tissues whose chief active principle is a complex of theelol (61). This was first made by Collip.

Campbell (8) highly favors this drug. He states that his results were far beyond expectation, the drug is perfectly safe and does not prevent impregnation or gestation. He treated the patient through three menstrual cycles, gradually reducing the dose. Only in a few cases should recurrence of dysmenorrhea be anticipated.

Watson (71) found results with emmenin were good in the patient who had severe uterine contractions. Goldberg and Lesser (34) treated forty patients and reported sixty percent received relief. Stacy and Shoemaker (67) used emmenin and estrogenic substances in cases of dysmenorrhea associated with irregularity of the menstrual cycle and obtained relief in fifty percent of these patients.

3. Theelin

This ovarian substance is formed by the granulosa cells and is called the female sex hormone. Another common name is estrin. The main function of this hormone is maintenance of the secondary female characteristics, preparing the endometrium for action of the corpus luteum hormone, sensitizing the myometrium to the action of the posterior pituitary.

Novak (52) uses theelin for permanent relief of dysmenorrhea. Fifty rat units are given on alternate days for six days, beginning the injections one day after the finish of the menstrual period. In a later paper (54) he is inclined to change his belief as to the effect of estrin. He thinks it is even possible that it may increase the pain.

Cannon (11) thinks estrin is of value in dysmenorrhea caused by an overactivity of the luteinizing hormone from the pituitary. Here estrin will override

the action of the corpus luteum hormone or have an indirect inhibition on the pituitary.

Lackner et. al.(41) favors this treatment in dysmenorrhoea where there is an increase of progesterone. They think the patient most likely to be this type is the one with the small hypoplastic uterus with poor contractions.

Kennedy(38) treating fifty cases, reported improvement in all but ten cases. However only ten patients were completely relieved. Tunis, a German writer, shows better results. Of seventy five patients, sixty five were cured and eight improved. He uses six injections, each of one thousand mouse units of estrin(28).

The definite statement that theelin is not indicated is made by Kotz and Parker(40).

4. Thyroid Extract.

Thyroid is indicated in those patients with a low basal metabolic rate and is definitely helpful according to Kotz and Parker(40). Curtis(19) agrees that here its use is beneficial as an adjunct and may even be the necessary factor for correction.

Roentgen Therapy

Trostler(69) states that the modern radiologist advocates irradiation in all cases of dysmenorrhea not frankly surgical or presenting surgical pathology. Primarily roentgen radiation in small doses is analgesic, the rays always depress pain, never stimulate it. It has a selective action on gland tissue and this author thinks that radiation depresses the overactivity of the pituitary, and so indirectly regulates the hormones forming the menstrual cycle. However, the author admits that this explanation is theoretical and so the therapy is largely empirical, but that it works.

Trostler uses twenty five to forty r. units to each area treated. This is far short of a castration dose so there can be very little danger of producing a permanent menopause. Irradiation of the pituitary alone has produced satisfactory results in cases of dysmenorrhea.

The following reasons are given for preferring this form of therapy. It is safer and more pleasant to take; it is applicable to patients who are poor surgical risks, and it does not necessitate anesthesia or absence from work. It should be administered only by doctors, never by technicians.

Rubin used Xray in cases of functional sterility according to Kotz and Parker(40). Newell and Petit(58)

reported seventy to eighty percent of their patients obtained relief by pituitary irradiation. Stacy and Shoemaker(67) recommend this method in those cases where other methods have failed.

Drips and Ford(24) in 1928 recommended irradiation of the ovaries and pituitary for dysmenorrhea, using five percent of an erythema dose well filtered. Ford (88) reported relief in eighteen out of twenty nine cases treated by irradiation.

Mazer and Spitz(44) observed that irradiation often relieved those patients with genital hypoplasia and dysmenorrhea. Irradiation did not affect the menstrual rhythm. However, they state that the margin between a safe and harmful dose is probably limited.

Kotz and Parker(40) thinks this therapy is more beneficial than any of the endocrine measures now available and recommend the technique of Christie.

20 millamperes, 200 K.V.P., at 60 inches, for five minutes using a 1 m.m. Copper and Aluminum filter. This dosage 170 r. units, is directed to the right and left sides of the pituitary gland every three weeks for four treatments. At the end of this the patient may return in two months and another series be given if necessary.

Treatment of Special Types of Dysmenorrhea

1. Membranous Dysmenorrhea

Novak(51) reports that treatment in this type is

very unsatisfactory. Cotte(16) reports relief of pain by presacral resection, but that the patient still continues to pass casts.

2. Allergic Dysmenorrhea

Schwarz and Smith(63) treated this type by elimination diets and then omitting the foods the patient was sensitized to. Thyroid extract and emmenin was used on a few patients. Dutta(25) determined the offending substance by history and skin tests. Treatment was either by desensitization with the substance, nonspecific proteins or by the avoidance of allergens. Attacks were always relieved by adrenalin.

3. Nasal Dysmenorrhea

Novak(51) gives the following technique as the Proper method of treatment. (1) Anaesthetize the area with twenty percent cocaine on cotton. (2) Touch both the tuberculum septi and the anterior end of the inferior turbinate with a crystal of trichloroacetic acid on the end of a probe. (3) Repeat the procedure in the other nasal cavity. A slough appears which disappears in about five days. (4) Repeat the treatment four times. If not relieved, repeat the same course of treatment the second time.

Conclusions.

1. The presence of afferent fibers conducting pain impulses from the uterus have not been proven to be located in the presacral nerve.
2. The incidence of dysmenorrhea depends upon clinical interpretation, and will therefore show variation. A average would show fifty percent of women with menstrual pain, fifteen to thirty five percent who suffer severe pain.
3. There are probably several etiological factors that may cause primary dysmenorrhea. Autonomic nervous system disfunction or endocrine inbalance are the most plausible explanations. Allergy or constitutional abnormalities may be responsible for an occasional case.
4. A neurosis may influence a dysmenorrhea or result from it, but in itself is rarely if ever the cause of the pain.
5. Experiments favor excessive uterine contractions as the immediate cause of the pain.
6. Mechanical obstruction does not appear to be a logical explanation of primary dysmenorrhea.
7. Congenital hypoplasia is probably a symptom associated

with dysmenorrhea rather than an etiological factor. An endocrine deficiency seems a more likely cause of both conditions.

8. Treatment must be varied and is largely empirical, depending on the individual case.

9. Good hygiene, proper exercise and good habits of living are indicated in the treatment of this condition.

10. The non-specific medical management offering the best result is the use of antispasmodics, analgesics, heat and rest, either singly or in different combinations.

11. A high carbohydrate diet for the week preceding the onset of the menses, is worth trying.

12. Alcohol injection should be considered but is probably somewhat hazardous for the average medical man.

13. Presacral resection is indicated in cases of intractable dysmenorrhea.

14. Results with hormonal therapy are disappointing. About the same degree of success is reported with the various preparations. More work is needed in this field.

15. Roentgen therapy although not used extensively as yet, offers good results along with some decided advantages as a therapeutic agent. Irradiation associated with hormonal therapy might increase the percentage of success above that of either treatment when used alone.

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