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THE ROLE OF THE PRESACRAL NEURECTOMY IN
PRIMARY DYSMENORRHEA

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Introduction

Dysmenorrhea is one of the commonest symptom complexes known to the gynecologist. It is also one of the most difficult to treat, medically or surgically. It has been estimated that about one of four women complain of some type of discomfort during their menstrual period (47). Of these women, 10 to 16 per cent are severely hampered in their activities during this time. The discomfort comes on any time from a few days preceding the onset of the menses to the first day or two after the start of the flow.

The type and severity of discomfort varies from mild aching controlled by mild analgesics to severe pain requiring bed rest and opiates.

If the uterine cramps were the only problem, more could be accomplished with medications. The problem arises with the treatment of the other associated symptoms which include pelvic discomfort, abdominal distention, nausea, vomiting, painful breasts, premenstrual tension, depression, irritability, and symptoms related to almost every organ system of the body.

It is because of these problems and the failure of medical treatment in many of these patients that this paper is being presented. This paper does not advocate the routine use of surgery in treating the patient with dysmenorrhea, but it will present the surgical approach to the problem and show that

there are definite indications for the operative approach to dysmenorrhea. When a careful work-up and a diligent attempt at medical treatment fails to answer the patient's needs, surgery in a selected group of patients done by an experienced surgeon should give almost 100 per cent relief to the patient.

History

The earliest attempts at the surgical relief of pelvic pain date back to the 18th century when Hunter noted symptoms of extreme pain and even death associated with abnormal uterine position. Retroversion and retroflexion of the pregnant uterus and subsequent incarceration were observed by him in many patients in the early part of the second trimester. Hence, when he found this in the nonpregnant patient he felt that this malposition was the cause of the pelvic pain. From this observation stemmed the various attempts at repositioning the uterus in hopes that the pain could be relieved. Doctors of the time used various methods to attempt this repositioning, including the packing of sponges into the uterine canal. It was believed that as the sponges absorbed moisture they would swell and force the uterus up and out of the abnormal position. Simpson's present day uterine sound was originally designed as a uterine elevator. Caustic substances were applied to the posterior lip of the cervix and the adjacent vaginal wall with

the idea that the resultant scar tissue would draw the uterus into its normal position (60).

In the 1840's Alquié, a Frenchman, advocated uterine suspension by exposing the round ligaments in the inguinal canal and shortening them. A Belgium physician, Deneffe, in attempting Alquié's operation failed to even locate the ligaments. From some of the literature one is led to believe that he lost his license because of this failure (60).

In the 1880's, after the introduction of asepsis, Batty attempted the relief of pelvic pain by removing the ovaries. This procedure was not popular for long because of the criticism which arose from the removal of normal tissue. All of these procedures only further emphasized uterine suspensions. In 1925 there were over 110 different types of suspensions in use (60).

Jaboulay, in 1898, was the first to attempt surgery on the nerves to the pelvic organs as a method of relief to women with pelvic pain. His method was to sever the afferent pathway in the sacral sympathetic chain by a posterior approach. His only criteria was pain. It was usually associated with pelvic malignancy. His operation proved to be too difficult and hazardous. For these reasons, the procedure was quickly abandoned. Ruggi, in 1899, following Jaboulay's lead advocated the vaginal approach for sympathectomy and hence relief of

functional disturbances of the female genital organs. In this procedure, Ruggi resected the utero-ovarian plexus by a trans-peritoneal route. This procedure was likewise abandoned because of its high incidence of failure and the hazards involved. Jellet (1900), Veit (1910), Wagner (1922), and Condamin (1927) also used the vaginal approach without success (16).

Fraenkl, in 1909, was the first to use the abdominal approach. He transected the uterosacral ligaments as did Brose (1923) and Molin (1929), all with rather poor results (16). In addition, Fraenkl interposed omental fat between the cut ends of the ligaments in an effort to retard regeneration of the involved nerves.

In 1920 a Romanian physician by the name of Gomoiu performed several operations for the relief of pelvic pain by sectioning the superior hypogastric plexus. His series of cases was small, but he had by far the best results and the fewest complications of those who had preceded him in this type of surgery.

Four years later, in 1924, found Leriche doing a peri-arterial sympathectomy of the internal iliac arteries for the relief of pelvic pain. His indications were, as most of those preceding him, related to pelvic malignancy and the associated pain. After a short but fairly fruitful life, this procedure was abandoned because of the difficulty in doing the operation. It was also very time consuming.

In 1924, Cotte, after whom the present day procedure is named, was doing the Leriche operation. In December, 1924, he did the first presacral neurectomy, the technic of which is still used today (8). The greater simplicity of this procedure over the Leriche operation was one of the factors which influenced the abandonment of the latter procedure. Cotte was not the first to do the presacral neurectomy but he was the first to use it as a treatment for primary dysmenorrhoea. Cotte was an exacting man and, as such, set down a strict list of criteria for selection of his patients. By adhering very strictly to these criteria his success in the use of this operation has not been duplicated to this day. In 1927 he published a follow-up on the first 90 patients on whom this operation was done. His follow-up failed to show a single failure. By 1929 Cotte had performed 200 presacral neurectomies. In a twelve year follow-up on these patients he could demonstrate but two failures. One of these patients later developed pain typical of ovarian involvement (lateral pelvic pain) and the other had endometriosis.

Following Cotte's lead, the presacral neurectomy became an often used operation in Europe. Its use in America was much slower to catch hold. Louis G. Herman of Cincinnati General Hospital was the first to do this procedure in America (14). Following him in the early 1930's Keene (7), Counseller and Craig (12), Adson and Masson (13), DeCourcy (35), and Abbott (36)

reported favorable results with the procedure although most of their series were small and the follow-ups incomplete.

Since then almost every gynecologist in the country has used the procedure at least once. However, there have been only a few who have had large enough series of patients or who have adequately followed their patients so that most of the statistics and the conclusions drawn from them are inconclusive, although not necessarily wrong.

Otte, in 1949, made the following statement: "In over 1500 patients seen in a period of twenty-two years I count only 2 per cent who were not cured by the operation." (33) As can be seen from his statement, it is rather difficult to compare small series of 20, 30, or even 60 patients to his large series of 1500. There are also other aspects of the problem to be considered besides mere numbers. One must consider the indications used by the surgeon as well as his experience. There is certainly no one who can match Otte's experience.

Etiology and Pathogenesis

What is dysmenorrhea? Simply and plainly stated, it is pain associated with the menstrual period. From the surface this does not appear to be much of a problem, but when one looks into the etiology of the pain it can soon be seen how complex the problem really is.

So that this paper does not run into infinity trying to list all of the possible causes of dysmenorrhea, it will be broken down into the essential or primary types and the acquired or secondary types. Since this paper is designed to discuss the primary type, it will suffice to say that the secondary type of dysmenorrhea is that type which is associated with any lesion or demonstrable cause for the pain. Most commonly endometriosis and pelvic inflammatory diseases are the associated lesions.

The primary type is much more difficult for which to ascribe a definite cause. Characteristically, its onset is at the time of the menarche or soon after, depending on the time of the start of ovulation. The symptoms vary some with the individual patient. In general, the patient will complain of a dull midline ache in the lower abdomen with or without associated colic. This is often associated with nausea and vomiting if the pain is severe. Many and sundry secondary symptoms may also be present, perhaps related to "premenstrual tension."

The specific etiology of primary dysmenorrhea is not known at the present time. Many theories with supporting evidence can be found throughout the literature. Perhaps the first theory presented was the one associated with cervical obstruction (8). This has been refuted on the basis that cervical stenosis is rarely associated with severe dysmenorrhea. The reason that this was originally introduced as a possible cause was that many of

the workers in the field were doing cervical dilatations and were having success. However, it was found that within six to nine months most of these patients would again have a return of symptoms. Duncan (39) feels that the short term results were due to changes in the endometrium. When the endometrium has returned to its normal configuration and function the symptoms return. He states that this occurs in about six to nine months. Other workers feel that the results were related to alteration in the nerve fibers within the cervical canal and the regeneration of the nerve fibers was associated with the return of symptoms. One might relate this process to the permanent relief that most women have after delivery of one or two children. The reason for this may be that the cervix is dilated many times more in childbirth and there may be an associated destruction of nerves within the cervix rather than a simple stretching of the nerve fibers.

Because of the frequent finding of hypoplasia of the uterus in patients with dysmenorrhea it was felt that this might be a causative factor (12) (30). The reason was related to the possible increase in the amount of fibrous tissue within the uterine wall leading to the production of irregular contractions. This has been more or less disproved because: (a) many of the small uteri are found in women who have never experienced dysmenorrhea, and (b) many of the patients with dysmenorrhea have normal sized

uteri. When this theory was first advocated cervical stem pessaries were used with the hope of increasing the size of the uterus and increasing the musculature of the uterus. This was supposed to allow the contractions of the uterus to become more rhythmic and to decrease the tone of the uterine musculature. This also can be related to the changes that take place in pregnancy.

Parsons (5) feels irregular contractions of the uterus, because of contractions of isolated muscle groups (proven by intrauterine balloon studies), are the cause of the pain. He attributes this to spasm of the terminal arterioles of the endometrium, probably because of the rapid regression of the endometrium following withdrawal of the growth stimulus when the corpus luteum degenerates. According to Parsons, this recession causes a disproportion to occur between the endometrium and the length of the coiled arterioles supplying the endometrium. This slows the circulation causing relative stasis. The arteries become compressed and the endometrium becomes necrotic. This necrotic tissue is toxic because of a toxin which results in vasoconstriction. Eventually, bleeding is followed by alternating vasoconstriction and vasodilation. The spasm of the muscle groups results from contractions in the presence of the ischemia.

Novak and Reynolds (12) were the first to support the

theory that endocrine dysfunction might be a possible cause of dysmenorrhea. Novak and Reynolds agreed with Parsons that the pain resulted from spasmodic contraction of the uterine muscles. Novak and Reynolds went on to say that rhythmic uterine contractions exhibit definite variation which are related to the various phases of endocrine activity. The original work by these two men resulted in the conclusion that primary dysmenorrhea had its origin in exaggerated or spasmodic uterine contractions, and that these are the result of decreased action of the corpus luteum and an increased action of the follicular hormone (folliculin) while this is in turn inhibited by the action of progestin (progesterone). Much of this work has since been disproved on the basis that primary dysmenorrhea is absent in the presence of anovulatory bleeding, as is progesterone. From this it has been concluded that progesterone may increase the tone of the uterus and may be the cause of the contractions.

In 1949 Greentree (55) wrote an article which incorporates most of the proven points. He describes the etiology of primary dysmenorrhea as a complex of four factors. He feels that primary dysmenorrhea is dependent on: (1) the amount of menstrual toxin circulating in the blood at any given time, (2) the endocrine, lymphatic, and sympathetic nervous system, (3) the threshold of pain at the nerve endings, and (4) the sensitivity of the cerebral cortex to the impression of pain made on the thalamus.

It has been shown that in the circulating blood of a menstruating patient there is a fibrinolytic toxin in the euglobulin that is identical to the toxin produced by Menkin (55), called necrosin. Menkin and others support the concept that cellular injury from any source will produce and liberate a vascular poison which stimulates the adrenotropic factor of the anterior pituitary. This, in turn, causes adrenal hypertrophy and an increased sympathetico-adrenal discharge. Seyle (55) feels that this noxious stimulus leads to the involution of the lymphoid tissues which in turn is responsible for the production of antibodies. Through these antibodies there is a further increase in the sympathetico-adrenal activity.

Most of the viscera, because of their protection within the body, contain few free nerve endings. Keiffer (55) has shown that the cervix, by contrast, has a rich nerve ending supply whereas the rest of the body of the uterus is almost devoid of nerve endings. These nerve endings respond to both chemical stimuli and distention. The variable present is one of innate sensitivity of the nerve endings to the various stimuli presented to it.

Greentree feels that the sensitivity of the cerebral cortex is related to sodium retention due to progesterone acting like a desoxycosterone and the resultant edema and ischemia of the cerebral cortex.

In summary, the etiology of primary dysmenorrhea can be related to several factors:

- (1) increased vasoconstriction due to (a) increased circulating menstrual toxin and (b) progesterone tending to nullify the vasodilating action of estrogen;
- (2) increased uterine contractions following ovulation and caused by the action of progesterone, not estrogen; and
- (3) increased pain sensitivity within the cerebral cortex related to the sodium retention and the resultant ischemia and edema caused by the progesterone.

In addition to these physiological factors one must consider the psychological make-up of the patient (55).

Anatomy

The presacral nerve of Latarjet is a segment of the sympathetic nervous system extending from the bifurcation of the aorta to the sacral promontory (10) (11). The term "presacral nerve" is a misnomer since the position is prelumbar and the physical nature is more often a plexus than a single nerve. Other names applied to this "nerve" include the superior hypogastric plexus (Plexus hypogastricus superior of Hevelacque),

plexus sous mesenterique (Winslow, 1732), nervus ganglionnaire lamelliforme (Bourgery, 1844), plexus uterinus magnus (Frankenhauser, 1867), plexus interiliaque and nerf presacre (Latarjet and Bonnet, 1913), nerf prelombaire (de Rouville, 1927), and prelumbar nerve of Elaut (29). The two most common terms used are presacral nerve and hypogastric plexus (10) (11) (14) (15) (29).

Preganglionic fibers of this nerve originate from the lowest thoracic and the upper lumbar levels of the intermediolateral columns. The axones are sent out over the lower white rami of the thoracolumbar outflow to the pre-aortic and lumbar ganglia. The postganglionic neurones originate in the sympathetic trunks and the pre-aortic ganglia. These postganglionic fibers join to form a plexus which descends along the abdominal aorta (10) (11).

The presacral nerve is formed by the union of three nerve roots. The lateral roots are branches from the first and second lumbar ganglia and fibers that have their origin from the ganglia in the area of the renal arteries. This bundle of nerves pass downwards to unite with the middle root which is a continuation of the intermesenteric plexus which consists of sympathetic fibers from the celiac, semilunar, splanchnic, and mesenteric ganglia. The nerve is finally completed by the addition of fibers from the third and fourth lumbar

ganglia which occurs laterally before crossing the sacral promontory. It should be noted that the fourth lumbar ganglion sends fibers to the plexus which course under the common iliac arteries and not over the bifurcation of the aorta as does the rest of the plexus (10) (11).

Past the promontory the presacral nerve divides into the right and left hypogastric nerves. These nerves course along the posterior pelvic wall supplying branches to the ureters and enter the lateral rectal space, terminating in the inferior hypogastric plexus. This plexus is located at the upper part of the rectum and bladder in the subserous fascia (59). At this point they are joined by the parasympathetics through the pelvic nerves (*nervi erigentes*) which are branches of the second, third, and fourth sacral nerves. These nerves form the pelvic plexus (16). Leaving the ganglia, the combined fibers form a plexus anterior to the rectum in the region of the uterosacral ligaments and continue along the internal iliac artery and its branches to supply the vagina, bladder, and the lower part of the rectum which also receives fibers from ganglia near the origin of the inferior mesenteric artery. These latter fibers contain postganglionic fibers from the sympathetic trunks and the pre-aortic ganglia. From the plexus on the anterior surface of the rectum the nerves course through the uterosacral ligaments to end in the uterus at its junction

with the uterosacral ligaments to form the great plexi of Frankenhauser (10) (11) (16). The bladder, cervix, fundus, and the proximal ends of the tubes are the main organs supplied from the plexus of Frankenhauser with a few fibers going to the adjacent walls of the vagina (16).

The sympathetic nerve supply to the ovaries arises in the ovarian nerve plexus (2). This is made up of fibers from the renal and mesenteric ganglia. These fibers course down with the ovarian vessels to the infundibulo-pelvic ligament where it divides into two branches, one entering the ovary and the other going to the fallopian tubes. A few fibers pass directly to the uterus and others anastomose with the plexus in the broad ligament (16).

The presacral nerve is in the interiliac space of Elaut which is in the form of a triangle with the bifurcation of the aorta at the tip, the common iliac arteries as the lateral limits, and the base marked by a line drawn through the sacral promontory. On the left side of this space is the inferior mesenteric artery coursing through the base of the mesocolon. This may be bound down across the median line of the triangle making access to the nerve difficult and hazardous although usually the mesocolon can be easily displaced for adequate exposure. Covering this space is the posterior parietal [#]peritoneum. The nerve lies in some fibrous tissue beneath

the peritoneum and may or may not be separated from the peritoneum by some fat. The plexus, in its sheath, crosses but is easily separated from the left common iliac vein which is seen as a broad band filling in the upper part and left side of the triangle. As the nerve nears the base of the triangle, it lies on the median sacral vessels and the prevertebral fascia covering the last intervertebral disc and the fifth lumbar vertebra (10) (11) (16).

Physiology

Most, if not all, of the afferent fibers from the uterus are found within the sympathetic nerves to the spinal cord. These fibers undoubtedly carry the sensation of pain. In addition, it is believed that the relief of pain is obtained not only by the cutting of these sympathetic nerves but in addition through the interruption of the efferent fibers which supply the vascular system of the uterus.

Evidence, at the present time, indicates that contractions of the uterus are completely unaffected by denervation (4). Mueller, Brachet, and Gertsmann (11) have shown that neither the section nor the complete destruction of the sacral part of the spinal cord will prevent childbirth, and Juntz (11) states that under these circumstances, parturition precedes with abnormal rapidity. Gotte (8) states that in 30 pregnancies after presacral excision he had never observed any evidence

of altered uterine contractility.

Resection of the presacral nerve does not alter the normal rhythm of menstruation (12) (34), but bleeding a few days after the operation and an increase in the amount and duration of the menstrual flow may result from the removal of the constrictor influence exerted by the sympathetics upon the blood vessels of the uterus and the broad ligaments (12) (34).

The sympathetic fibers are intimately associated with vasoconstriction and inhibition of the muscles of the sigmoid, rectum, and bladder and cause ejaculation and contraction of the involuntary sphincters of the bladder. The parasympathetics, as throughout the rest of the body, work in opposition by causing vasodilation and release of the various sphincters (12).

Through the section of the presacral nerve one can conceive of a better blood supply to the uterus and release of muscular tone in the body of the uterus and the cervix. However, because of the action of progesterone, this is not a complete release. Because of the lack of atrophy or major disturbance of the motor function of the uterus, an observer can safely conclude that the genital nerves of the sympathetic systems are sensory rather than motor (12) (34).

It is important that one understands the effect that these nerves have on bladder and rectal functions since occasionally

malfunction of either of these organs may be a complication of a presacral neurectomy. The sympathetics carried in the presacral nerve are inhibitory to the detrusor muscles and motor to the internal sphincter, trigone, and the ureteral orifices (4). They also exert a constrictor influence on the blood vessels of the bladder. The parasympathetics, carried in the pelvic nerves, are motor to the detrusor muscles and inhibitory to the internal sphincter. Normal micturation is dependent upon the integrity of the parasympathetic pathways which are unaffected by the division of the presacral nerve. On the other hand, the sensation of fullness and the pain of overdistention or disease of the bladder are transmitted via the sympathetic fibers within the presacral nerve and are altered by a presacral neurectomy. As such, in the first few days following a presacral neurectomy the patient may have to be catheterized but they usually quickly regain the lost sensation (12) (34).

The sympathetic nerves to the rectum, blocked by presacral excision, supply the lower rectum and the internal sphincter of the anus. The external sphincter, which is composed of voluntary muscle, is supplied by the inferior hemorrhoidal branches of the pudental nerves and because of this separate innervation, rectal function is not impaired by presacral neurectomy (4) (12) (34).

In summary, the presacral neurectomy might be expected to have an effect on three different organs: the uterus,

the bladder, and the rectum. The uterus will show a decrease in the vasoconstriction of the blood vessels and a release of some of the muscular tone. The bladder will temporarily lose the ability to express fullness, although this is quickly regained (32), and the rectum is unaffected because of the separate innervation of the external sphincter of the rectum.

Picking of Patients

The criteria laid down by Cotte when he originally started to do this type of surgery still hold true for the most part. However, with the advent of new methods of treatment based on a better understanding of the basic physiology, the number of patients which qualify has diminished greatly. In 1930 Cotte (15) defined the presacral nerve as one that innervates exclusively the pelvic and vaginal regions and that resection of this nerve is a cure for "plexalgia hypogastrica" which included dysmenorrhea, dyspareunia, causalgia of the vagina, pelvic neuralgia, and pruritis vulva (33) (35) (36). He further stated that the presacral neurectomy was of no value in the treatment of lumbar pain (ovarian dysmenorrhea) or for ovarian intramenstrual crisis. From this he inferred that midline pain that was referred from the uterus to the anus, coccyx, and/or bladder could and would be relieved by a presacral neurectomy. Cotte (8) (33) (36) also included

metrorrhagia of ovarian origin and sexual neuroses as important criteria and, in addition, one can find in his writings the concept that hypoplasia of the uterus was a possible etiologic agent in primary dysmenorrhea since he included this also in his criteria IF it was associated with insufficient and painful menstruation. These are the basic criteria that Cotte used. One must admit, if his statement is correct, that they seemed more than adequate since he has seen by far the largest number of patients of any one and reports only two failures in his first 200 cases and only two per cent failures in over 1500 cases (33).

Since his original criteria were published they have been changed and altered to fit both the changing trends in medicine and the whims of the individual surgeons. The operation has been used for secondary dysmenorrhea, pelvic cancer of almost any type—including rectal and bladder types, and neurogenic bladders. The results with most of these cases have been generally disappointing. And yet, probably the most extensive use of the procedure is in the treatment of secondary dysmenorrhea in conjunction with other surgical procedures.

Greenhill (18) uses the following criteria: (1) severe dysmenorrhea uncontrolled by medical management, (2) endometriosis in a young women when conservative surgery is indicated, (3) inoperable carcinoma of the female generative tract, and (4) pelvic pain of unknown origin unsuccessfully

treated by one or more laparotomies.

Fontaine and Herrmann (11) used the following criteria:

(1) no organic lesions to explain the dysmenorrhea, (2) cases with slight pathologic processes in the pelvis which do not react favorably to ordinary gynecological treatment, and (3) known inoperable pathology such as carcinoma.

Marshall and Kennedy (20) seem to feel that this operation should be performed on any patient who is operated on for uterine suspension or on any patient with dysmenorrhea who has a laparotomy for any reason. They do not seem to feel that surgery primarily for dysmenorrhea should be done if the pain can be relieved or controlled satisfactorily until the age of thirty or until childbearing. In addition to the above stated indications these authors feel that surgery is indicated when (1) the pain is so severe, in spite of treatment, that it causes disability each month, (2) severe dysmenorrhea persists after the age of thirty, and (3) severe pain begins in adult life.

In addition to the information gained from the physical and history, important tests are available to help in the picking of these patients. One of these tests consists of the production of an anovulatory cycle with estrogen, since it has been shown that in the absence of progesterone there is physiologically no reason for pain. If pain is present in an

individual in whom an anovulatory cycle has been produced one can safely interpret that the patient is psychoneurotic and will not be helped by a presacral neurectomy (16) (26). This test is invaluable in this aspect since, with a better understanding of the background of these neurotic women, psychotherapy is much more successful and easier.

A test used by Meigs (19) was first introduced into this field of medicine several years before the writing of his article. This test consists of the injection of 2% precaine into the region of the plexus. The second and third lumbar ganglia are injected. By doing this bilaterally one should get complete anesthesia of the presacral nerve. This test gives the same results, mechanically, as the operation itself and if the pain is relieved, one should anticipate complete relief of pain with the operation within the limitation of the capabilities of the surgeon.

Another test that is useful is a placebo test. In this test the doctor substitutes a placebo for a narcotic and gives it to the patient at the height of her pain, telling her it is a narcotic. A thirty minute observation of the patient is sufficient to evaluate her response. If she gets relief she is obviously neurotic and is no longer a candidate for surgery. If she continues to have pain after the administration of the placebo, the narcotic is then given. This test is

repeated at a later date to verify the original results (60).

It would then seem that in order to pick the patients who are most likely to get good results from this operation one must first of all do a very complete history being particularly interested in the menstrual history. If the suspicion arises, the investigator should go into the patient's feelings about menstruation and the family background since this is the first area in which the doctor has the opportunity to eliminate the neurotic patients. Next, a general physical examination is in order. Emphasis here is placed on pelvic examinations so that one can definitely rule out a pathologic lesion. If there is any demonstrable lesion then the patient is not a candidate for presacral neurectomy since, by definition, this procedure is limited to primary dysmenorrhea, although in practice this is not the case.

In treating the patient with primary dysmenorrhea the next consideration is whether or not the patient is a candidate for surgery. In almost every case the patient will not be, the exception being the patient who has been treated for a long time with accepted medical management without demonstrable improvement. With the advent of new drugs and a better understanding of the hormone systems involved in this problem many of the patients that once would have qualified as surgical cases are now more than adequately controlled. Phaneuf (17)

makes the following statement which emphasizes this point:

"This operation should, however, be resorted to only in those cases in which all other simpler methods have failed, and should be reserved for the so-called spastic or uterine form of dysmenorrhea, a condition in which the patient experiences severe cramps." If after a long and diligent attempt at conservative treatment has failed, and some feel that this should include childbirth when feasible, then one should consider a presacral neurectomy.

One aspect of the problem which has not been considered as yet is the patient's feelings. If she is primarily concerned with ridding herself of the pain and is definitely not interested in having children one can resort to a more absolute cure, an oophorectomy or a hysterectomy (16). This seems highly unsuitable for obvious reasons, since most of these patients are early in the child-bearing age (17) (20) (23) (27) (36) (47) (57). In this group conservative surgery is indicated since it will not interfere with either the prenatal period of a pregnancy or the actual delivery and, in fact, makes delivery much easier in most of the patients (8) (11) (26) (34) (40) (59).

If the patient is truly incapacitated with dysmenorrhea, the evaluation of which by now should be relatively easy and complete, and she is willing to submit to the operation and the associated hazards which are the same as with a laparotomy in

the hands of a capable surgeon, then the presacral neurectomy is in order since one can anticipate improvement or cure in over 90 per cent of the patients operated on (Table I). However, at no time should the patient be given a guarantee of the success of the operation.

Technic of the Presacral Neurectomy

Good preoperative preparation, good anesthesia, and good postoperative care need only be mentioned since they apply in all operative procedures. Cotte recommended a midline incision 10-12 cm. in length extending from the pubis to the umbilicus. Cannaday (29) and others feel that a Pfannenstiel incision can be equally well used as long as the bleeding is well controlled. The Pfannenstiel incision gives a much better cosmetic result. The patient is then put in a Trendelenburg position. Adequate exposure, which is of paramount importance, is obtained by packing of the small bowel off to the right side and pushing the sigmoid and rectum to the left. The landmarks then exposed are the bifurcation of the aorta, the common iliac arteries, and the promontory of the sacrum. On the left of the space is the inferior mesenteric artery coursing through the base of the mesocolon. If the mesocolon extends over the interiliac space, the peritoneum is incised well to the right of the midline in order to avoid the left ureter. Otherwise, a midline incision

is made in the parietal peritoneum, extending from above the bifurcation of the aorta to below the promontory of the sacrum. Blunt dissection elevates the flaps of the peritoneum. When this is complete, the sheet of fibrous connective tissue which includes the plexus should lie completely exposed. Separation of the nerve and the fibrous tissue sheath is made en masse, beginning over the right common iliac artery near the bifurcation of the aorta. The dissection is continued over the left iliac vein to the left common iliac artery (33). The underlying loose areolar tissue is elevated and the dissection is continued down, avoiding the median sacral vessels, until the division of the plexus into the hypogastric nerves is seen. Here, Cotte (33) recommends that each of the hypogastric nerves be separated for a length of 2 or 3 cm. and be included in the removal. However, the surgeon must not dissect down too far along these nerves lest he interfere with the inferior hypogastric plexus which is involved in the act of defecation. Cotte also warns that along the course of the plexus there are nerve filaments extending laterally which must be included in the dissection. The two ends of the dissected nerve are then ligated and severed.

Various technics are used to prevent regeneration of the nerves, such as the use of small silver clamps or simply folding under the nerve ends. The peritoneum is closed with a continuous suture. If this procedure is being used in conjunction with

some other procedure the latter should always follow the neurectomy to prevent contamination of the retroperitoneal space. Closure of the abdominal wall is made in the usual manner.

Review of Articles in English Literature

The following section will deal with the presentation of the results of surgeons who advocate and use this procedure, some more extensively than others. A majority of the material is obtained from American journals. Though the number of publications is small, they have been fairly well distributed over the last twenty-five years.

In trying to evaluate the papers that are available it becomes evident that there is no universal method of grading the results. However, it does seem that most of the results can be conveniently included in three categories, i. e., cured, improved, and failure. Neither is the type of follow-up the same in all cases. Some of the authors go into the results of the operation in much more detail, while some merely mention their results. Many authors worked only with secondary dysmenorrhea and because of this are not included in this paper. Others have material concerning both primary and secondary dysmenorrhea, and in these cases only the material pertinent to primary dysmenorrhea will be presented.

In Table I all of the figures have been compiled. Most of

these authors have published articles which were available for more detailed review. Some have been published in smaller journals not available in our library.

In 1935 Pemberton (27) published what was at that time the largest series in this country. Included in his series were fifteen primary dysmenorrheas and eighteen secondary dysmenorrheas. Also included were an additional six patients with primary dysmenorrhea which had been operated on by James White (unpublished data). In the primary group the average age was 26 with a range of 16-37. Only one of the patients was under 21. Fifteen had nothing more than underdevelopment of the pelvic organs. None had been pregnant. Of the total group of fifteen, fourteen had a dilatation, a suspension of the uterus by the Olhausen method, and an incidental appendectomy as well as the neurectomy. The suspension was done because the author thought that it would help to relieve the dysmenorrhea although no mention is made of the degree of retroflexion present. One of the patients had only a dilatation and a neurectomy. The time elapse for the follow-up varied from 2-36 months with an average of 15 months. James White, whose material was included in this series did only a presacral neurectomy leaving a retroverted uterus. In his limited series of six he reported no failures. The data can be found in Table I.

In 1936 Cannaday (29) published a report on a group of 78

TABLE I

Composite summary of patients with primary dysmenorrhea treated with presacral neurectomy either alone or in combination with some other operative procedure.

Author	Number	Relief		
		Complete	Partial	None
Pemberton	15	12	1	2
White	6	6	0	0
Canaday	41	23	11	7
Abbott	6	6	0	0
Masson et al	11	3	4	4
Meigs	20	14	3	3
Colecock	35	28	6	1
Hendrich	18	15	3	0
Rutherford	20	16	4	0
Marshall et al	80	72	7	1
Duncan	53	46	3	4
Tucker	157	107	15	15
Phaneuf	68	40	19	9
Ingersoll et al	89	73	4	12
Fertitta et al	105	92	9	4
Hoge	78	-----56-----		7
Black	50	31	15	4

patients that were treated with a presacral neurectomy, either used alone (41 patients) or in combination with other procedures. His criteria included pelvic pain, dysmenorrhea and pelvic pain, dysmenorrhea alone, pelvic pain and constipation and dyspareunia with pelvic pain. Only the results of the forty-one patients treated by neurectomy alone are included in this report. See Table I. It is interesting to note that Cannaday broke down the results of the surgery according to the symptoms for which they were operated on. The results are as follows:

	Number	Complete	Improved	Unimproved
Pelvic pain	33	21	7	4
Dysmenorrhea and pelvic pain	11	5	5	1
Dysmenorrhea alone	21	13	5	3
Pelvic pain and constipation	3	2	1	0
Dyspareunia and pelvic pain	4	1	2	1

If the author had but discussed the pelvic pain in more detail and described its character these figures would have meant much more to the reader.

Also in 1936, Abbott (36) published data on eight patients. The age range of his patients was 20-34. He apparently grouped his patients at the operating table. He found six patients without demonstrable pathology at the time of the neurectomy. Of the other two, one had a low grade endometriosis and one had a cyst of the right ovary which was adherent to the sigmoid. All eight of his cases were completely relieved. The results are in Table I.

In 1938 Masson and Shoemaker (31) reported a review of all

of the cases at the Mayo Clinic from 1931 to 1934. There was a total of 682 patients with some form of dysmenorrhea. Of this group 112 did not require treatment. Four hundred five were handled medically. The remaining 165 were advised to have surgery. Thirty-seven of these refused. Of this entire group only fifteen had a neurectomy, either alone or in combination with some other procedure. The results were as follows:

	Total	Complete	Improved	None
Resection of the nerve --				
alone	8	2	3	3
with resection of endometrial implants	3	2	1	0
with uterine suspension	3	1	1	1
with myomectomy	1	1	1	0

In 1939 Meigs (19) published what appears to have been the first series dealing only with primary dysmenorrhea. He had a series of twenty patients. He did only a neurectomy on these patients. He graded the results in percentages as follows: four, 100%; five, 90%; one, about 80%; four, 70%; two, 50%; one, 30%; and three failures. The results are tabulated in Table I.

In 1941 Colcock (23) reviewed a series of thirty-five patients on which a presacral neurectomy was done for primary dysmenorrhea. All of these patients had a normal appearing pelvis at the time of surgery. All of these patients were followed for at least six months or more. Their ages ranged from 17 to 45 with more than 70% under thirty. Twelve of the

patients were married and had had children. The duration of symptoms varied from five months to nineteen years. In twenty-six the dysmenorrhea had started before the age of eighteen. All had had some form of conservative treatment including analgesics, hormones, dilatations, uterine suspensions, and one had had an oophorectomy, all without relief. In eighteen of the patients retroversion was noted. Five had cystic ovaries. In eight of these patients only a neurectomy was done. No mention is made concerning the results on these eight patients specifically. In twenty-seven a uterine suspension was done in addition to the neurectomy. He noted no post-operative complications. His results are in Table I. In the seven who got only partial or no relief two had only mild dysmenorrhea not requiring further treatment, one (45 years old) was 50% relieved and later was completely relieved with a hysterectomy. One had two to three hours of rectal discomfort. In these four the author questioned the complete excision of the nerve. The other three were poorly picked. All of these patients had dysmenorrhea which was improved but other symptoms of constipation, abdominal cramps, and those referable to ulcerative colitis were not relieved.

Hendrich (57) published an article in 1941 in which he reviewed the results of a presacral neurectomy on 18 patients with primary dysmenorrhea and 52 with secondary dysmenorrhea. In the primary group the range in age was 16 to 34 with an

average of 23. The results can be seen in Table I. In this group he reported six normal pregnancies. In the secondary group the age range was from 18 to 36 with an average age of 24. In this group he placed patients on whom he did such additional surgery as dilatation and curettage, cauterization of the cervix, removal of ovarian cysts, and Gillian's suspensions. Unlike many of the authors, Hendrich apparently did not include suspension of the uterus and dilatation and curettage as a routine along with the neurectomy.

Rutherford (15), in 1942, reported on 23 cases of dysmenorrhea of which three were of the secondary type. In his report he stated that all had 50% or better relief. His results are included in Table I. Only one of the patients reported that the operation was not worthwhile. The curious aspect about this is that she was one of the twelve who got 100% relief. In his follow-up he reported eight normal pregnancies without complications.

In 1945 Marshall and Kennedy (20) reported a series of 100 consecutive cases of which 80 were for primary dysmenorrhea. Their results are in Table I. In all of these cases, spinal anesthesia was used. In addition, all were subjected to dilatation and curettage. He reports that all were subjected to long periods of medical treatment. General hygiene was improved, antispasmodics and hormones were administered, and psychotherapy was applied where needed. In the group of 100, 69 had suspension of the

uterus for "malposition." Ages ranged from 13 to 39 with an average of 26.7 years.

In 1946 Duncan (39) published an article in which he reviewed 53 cases of primary dysmenorrhea observed between 1941 and 1945. There were also 33 cases of secondary dysmenorrhea reviewed in this paper. The results can be seen in Table I. Duncan did note that those patients who complained of true mid-line uterine colic were completely relieved. He also noted that none of the four failures in the primary group had the typical description of uterine colic. In the essential (primary) group the treatment consisted of combining a dilatation and curettage and a suspension with the neurectomy where indicated. He apparently used this combination in all of the patients of the primary group.

In 1947 Tucker (14) reported one of the largest series reviewed in this paper. It is also one of the most documented and well discussed papers with excellent reporting on follow-ups. The series of patients is broken up into two groups, those done between 1931 and 1941 and those done from 1941 to 1945. This was done with the expectation of showing any progress in the results as they might relate to time or experience. The results can be seen in Table I. In the first group, those done from 1931 to 1940 inclusive, there was a total of 78 cases of the essential type. Of these, 57 (73%) got complete relief, 11 (14%)

got partial relief, and 10 (13%) were classified as complete failures. In the second group, those done from 1941 to 1945, there was a total of 59 cases. Of this group, 50 (84.6%) were completely relieved, 4 (6.8%) were partially relieved, and 5 (8.4%) were classified as complete failures. The one drawback in this article, and an important point when one is considering the results, is the complete absence of information regarding the exact mode of treatment in terms of whether or not the neurectomy was used alone or in combination with some other corrective procedures. Neither is there any mention made of the ages of the patients involved.

In Phaneuf's report (17) published in 1948 is the results of 76 patients of the primary type treated with presacral neurectomy. There is no mention made of its use with other procedures. The results can be seen in Table I. The ages of the patients ranged from 13 to 38. The age grouping was broken down as follows: fifteen from 13 to 19, forty-five from 20 to 29, and sixteen from 30 to 38. His results were based on voluntary responses of the patients. He used three criteria: satisfactory (pain absent or barely noticeable), improved (some pain still present but considered better), and unimproved (as much pain as before the surgery).

In 1948 Ingersoll and Maigs (26) reported a series of 89 cases with essential dysmenorrhea treated with presacral

neurectomy between the years 1930 to 1946. Their results can be seen in Table I. They discussed their use of diethylstilbestrol in the pre-operative picking of patients and found that in those in whom painless bleeding was produced with the drug, 27 of 32 had successful results with the presacral neurectomy whereas in those in whom bleeding was painful after the administration of diethylstilbestrol there were three failures and no successful results. From this one can see the value in producing anovulatory bleeding in these patients and how its correct interpretation is necessary before surgery is contemplated.

In 1950 Fertitta et al (46) reported a series of 105 patients with primary dysmenorrhea treated with a presacral neurectomy. Ninety-two per cent of the group was married at the time of surgery. Forty-nine per cent of the group had borne children before, during, or after the development of the dysmenorrhea. Ninety per cent of the group had been treated with endocrines before surgery was contemplated. Forty-eight per cent of the group had a uterine suspension in conjunction with the neurectomy. The results of the study can be found in Table I.

In 1954 Hoge (58) analyzed 36 cases of essential dysmenorrhea treated between 1943 and 1953 in which a presacral neurectomy was performed. The results are tabulated in Table I. These 36 had no demonstrable lesion to explain their pain. In

addition, he included nine cases in which only cervicitis was found, nine in which incomplete retroversion was found, and eight in which both cervicitis and retroversion were found. This increased his total number of patients with primary dysmenorrhea to 62. All were treated with presacral neurectomy. In addition, 15 of the 17 with retroversion had a suspension and all of those with cervicitis were treated with cauterization or conization. These patients were all grouped into one group and reported as such. In addition to this, Hoge had another group of 16 patients which he referred to as "complicated primary dysmenorrhea". In these there were pelvic lesions, but he felt that they were not the cause of the dysmenorrhea. The group included nine with adhesions (mostly post-operative), two with small simple ovarian cysts, two with small leiomyomata, one with relaxed vaginal outlet, one with varicosities of the broad ligament, and one with an adherent retroversion. All of these had an operative procedure directed at the pathology in addition to the neurectomy. In this group he had nine (56.3%) with successful results and seven with inadequate follow-ups. He reported no failures and no recurrences.

In 1955 Black (47) reported a series of 70 patients with dysmenorrhea, 50 of whom were classified as primary. The average age of this group was 25. Seventy-six per cent of the group were married and twenty-six per cent had children. Twenty

per cent of the group had had a previous dilatation and curettage. His results can be found in Table I.

In addition to the above mentioned articles there are many others available in which the presentation was not adequate to evaluate. However, two are being mentioned because they apparently represent preliminary reports on large series of patients with primary dysmenorrhea. In 1951 a discussion following a paper presented by Wetherell (45), Phaneuf reported on 125 cases, 76 of which have already been discussed in this paper, in which he had 80 (66%) complete relief of pain, 28 (22.7%) partial relief of pain, and 14 (11.3%) failure. No mention was made as to what combination of procedures was used or what type of dysmenorrhea the patients had, although it is known that at least 76 were of the primary type.

In 1954 Lipman (9) reported a series of 50 cases in which he obtained 92% excellent results. He mentions that these did not necessarily have complete relief of pain. He later had to reoperate on four of these patients. He makes no mention of the procedures used. From his paper one cannot determine what his criteria for excellent results are nor how broad a concept he used in classifying his patients.

Cotte (8) states that there are three main reasons for failure of this operation. One, and probably the most important, is the failure to pick out the patients with psychoneuroses. He feels that in a certain per cent of the patients

there will be regeneration of the nerves, but if the surgery is thorough this is a very small per cent. The other failures can be accounted for on the basis of the surgeon failing to completely remove the plexus. He feels that this accounts for about ten per cent of the failures.

In addition to the material available for the evaluation of the success of the presacral neurectomy on primary dysmenorrhea, a few of the authors evaluated the effect of the operation on pregnancy and labor.

Ingersoll and Meigs (26) in 1948 reported a series of 24 patients out of a group of 111 who had had a presacral neurectomy and later became pregnant. Eight of the group had no severe cramps with the uterine contractions and required no medications. All of the patients experienced pain with the passage of the baby through the perineum but this was so rapid that it was only a few brief moments in duration. There were no reported complications in the 24 patients. Of the eight patients who failed to experience pain six had primary dysmenorrhea and two had secondary dysmenorrhea. No mention is made of the effect of the neurectomy on the original complaint.

Rutherford (15) reported a follow-up of 23 cases treated with a neurectomy in which eight later became pregnant. In the group of eight there were no prenatal problems and no complications at the time of delivery or after. Seven of the eight

needed no medications until dilatation was 50 per cent or more and five of the seven needed no medication through the entire first stage of labor.

Several other authors have published remarks on a few patients who have undergone a presacral neurectomy and then later become pregnant (56). Most of them found that the patients would progress well into the first stage of labor without discomfort. A few report that the severity of the pain was unaffected while others report precipitous labors. These figures actually mean little since they have not been correlated with the results of the presacral neurectomy.

Summary

1. Some of the early history associated with the surgical attempts as relief of pelvic pain is discussed.
2. The definition of the etiology and pathogenesis of primary dysmenorrhea is evaluated and the current concepts are discussed.
3. The anatomy of the presacral nerve is presented.
4. The resultant effect of the presacral neurectomy on various pelvic organs is discussed.
5. The operative technic is presented.
6. The evaluation of the patient by various authors is discussed and some of the tests employed in picking of patients

are presented.

7. A review of the available literature is presented.

Discussion

In considering the surgical management of primary dysmenorrhea the picking of the patient is all important. One should never consider a patient as a candidate for a presacral neurectomy until conservative medical treatment has been exhausted. In this way the consideration is arrived at by a process of elimination.

The evaluation of the patient starts with a detailed history. One is interested in the patient's background with particular emphasis placed on the home atmosphere as it was directed toward the patient's early indoctrination into the matters of sex and reproduction. The mother's response to menstruation and type of explanation that the mother gave to the patient in her formative years has a definite relationship to the patient's later response to marriage, reproduction, and the function of menstruation. If the patient grew up in a healthy atmosphere she is much more likely to accept these turning points in her life without undue stress.

Of equal interest is a detailed history of the patient's menstrual life. At what age did the menarche occur? When did dysmenorrhea begin? Typically, it begins at or soon after the onset of ovulation which may follow the onset of the menses by

a year or two. If dysmenorrhea begins after the middle twenties one should begin to think more in terms of secondary dysmenorrhea although this is not true in every case. If it begins after marriage functional overlay should be strongly considered and further investigation along this line should ensue. Along these lines questioning into the wife-husband relationships is to be considered.

The character of the periods is important. Does the patient pass clots? Is the flow heavier than average? Are the periods irregular? Is there intramenstrual pain? Is there a discharge? These points are not of too great importance except that any abnormality will direct one to think along the lines of definite pathology although this is not necessarily true in all cases.

Perhaps the most important information about the menstrual cycle is the pain and its character. When does it begin in relation to the onset of menstruation? It may begin anytime within two or three days preceding the onset of menstruation or within a day or two after menstruation has started. Where is the pain and to where does it radiate? The patient with primary dysmenorrhea has midline colicky pain with radiation to the areas known to be innervated by the hypogastric plexus, i.e., bladder, vagina, vulva, and rectal areas. Lateral pain is usually ovarian in nature and is more often associated with secondary dysmenorrhea. How much does the patient suffer

and to what extent is this incapacitating to her? If the discomfort is mild one can anticipate that mild analgesics and conservative treatment will be all that is needed. If, however, the pain is severe and incapacitating and requires narcotics for relief one may well begin to think of surgery. This is true regardless of whether one is dealing with primary or secondary dysmenorrhea. The rest of the history inquiring into the general health of the patient may elicit some valuable information as to past infections or operations.

A general physical examination is important and any abnormalities are to be noted. Most important is the bimanual examination. A negative pelvis is more suggestive of primary dysmenorrhea whereas the finding of endometrial implants, adhesions, and ovarian cysts is highly suggestive of secondary dysmenorrhea. The question of the relationship of uterine displacement to primary dysmenorrhea is still not certain but except in instances of extreme retroflexion and retroversion, in itself, is not likely to be the sole cause of primary dysmenorrhea. The relationship of cervical erosion and cervicitis in relation to dysmenorrhea is not fully understood but is not thought to be implicated.

There are actually only two practical tests that need to be performed on these patients. One is the production of anovulatory bleeding by the use of stilbesterol or similar

substance. It has been conclusively shown that when ovulation is suppressed in these patients their dysmenorrhea disappears during that cycle. In the presence of continued pain one should be most suspicious of a neurotic personality or at least some functional overlay. These patients have been shown time and again to be poor candidates for any type of surgery for dysmenorrhea. The other test which seems worthy of mentioning involves the use of a placebo. In using this test the doctor must be extremely interested in the patient, enough so that he is willing to go to the patient during the height of her discomfort regardless of the number of patients in the office or of how tired he may be. Going to her home he gives her a placebo in the place of a strong narcotic, although he tells her that the medicine is a strong narcotic and should give her relief within a few minutes. The most time-consuming part of this test is the waiting. One should wait a minimum of 30 minutes and watch the effects of the 'medicine'. If she gets relief one should be strongly suspicious of some functional disorder. However, if the patient still has pain, then a strong narcotic is given and again one should wait until relief is obtained. In the sequence of events in studying these patients it does not matter which of these tests is done first since one is usually confirmatory of the other. Another important point, and a must if one is to be complete, is that both of these

tests should be repeated at least once and even oftener if the examiner is at all unsure of the interpretation of the test results.

As can be seen, the testing in itself involves a minimum of four months to complete. This may be long enough to evaluate a patient completely. This would be true in a patient who has already been treated conservatively with medications. However, one should not rely on the patient's history as to what type of treatment has ensued in the past. If a letter from the referring doctor is available with sufficient information, then one can progress to the testing right away. If, however, the information is not available or the patient has come to the examiner initially then the first course of evaluation is conservative medical therapy. If this controls the patient there is no reason to progress further, at least for the time being.

Of the data published to date few are well enough analyzed to draw any valid conclusions from, although they all indicate that the presacral neurectomy is of value in a limited number of patients. All too often the presacral neurectomy has been combined with some other operative procedure. By doing this it is impossible to evaluate the effect of the neurectomy alone. Although one can feel relatively certain that in most patients a uterine suspension or a dilatation and curettage does not afford any relief of dysmenorrhea, the very fact that the

procedures are done together makes any results obtained valid only for the combination used. Many times the authors publish what appears to be excellent results but make no mention of the criteria for the picking of the patient, the preoperative treatment, the procedures used, nor the method and time of follow-up of these patients. Many of the authors used the presacral neurectomy in both primary and secondary dysmenorrhea and then reported the results together, thus making evaluation of the effects on the primary group impossible.

Consequently, the results as reported are most likely overexaggerated due to their methods of statistical reporting. Regardless of the work to date, of all the written and verbal discussions concerning the pros and cons of the presacral neurectomy, there is still no absolute proof of the true value of the presacral neurectomy in the surgical management of primary dysmenorrhea alone. But to prove this, further evaluation by competent surgeons and honest statistical reports limited to primary dysmenorrhea treated by presacral neurectomy alone is needed. However, in spite of this, there apparently is a minority group in which the presacral neurectomy would be the surgical procedure of choice after a careful evaluation of the patient has ensued.

* * * *

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BIBLIOGRAPHY

1. Novak, Josef and Rubin, I. O., Integrated Gynecology: Principles and Practice, New York. McGraw-Hill, 1956. Vol. 3.
2. O'Donel Browne, Litt D., A Survey of 113 Cases of Primary Dysmenorrhea Treated by Neurectomy, Am. J. Obst. & Gynec. 57:1053, 1949.
3. Shaul, Nathan, Presacral Sympathectomy as a Treatment of Obstinate Dysmenorrhea, Canadian Med. Assoc. J. NS35:53, 1936.
4. Evans, Albert L., Cofer, Olin S., and Gregory, Hugh H., Presacral Neurectomy, Medical Times 80:682, 1952.
5. Parsons, Langdon, Dysmenorrhea: Its Causes and Treatment, Medical Clinics of North America 38:1419, 1954.
6. Stalker, Leonard K., The Surgical Treatment of Dysmenorrhea, New York State J. of Med. 53:2240, 1953.
7. Keene, Floyd E., The Treatment of Dysmenorrhea by Presacral Sympathectomy, Transactions of the American Gynecological Society 60:93, 1935.
8. Cotte, Gaston, Resection of the Presacral Nerve in the Treatment of Obstinate Dysmenorrhea, Am. J. Obst. & Gynec. 33:1034, 1937.
9. Lipman, George S., Superior Hypogastric Sympathectomy for Primary Dysmenorrhea, Am. J. Obst. & Gynec. 68:1535, 1954.
10. Elaut, L., The Surgical Anatomy of the So-Called Presacral Nerve, Surg., Gynec., & Obst. 55:581, 1932.
11. Fontaine, Rene, and Herrmann, Lois G., Clinical and Experimental Basis for Surgery of the Pelvic Sympathetic Nerves in Gynecology, Surg., Gynec., & Obst. 54:133, 1932.
12. Counsellor, Virgil S., and Craig, Winchell McK., The Treatment of Dysmenorrhea by Resection of the Presacral Sympathetic Nerves: Evaluation of End Results, Am. J. Obst. & Gynec. 28:161, 1934.
13. Adson, Alfred W. and Masson, James C., Dysmenorrhea Relieved by Resection of the Presacral Sympathetic Nerves, J. A. M. A. 102:986, 1934.

14. Tucker, Arthur W., An Evaluation of Presacral Neurectomy in the Treatment of Dysmenorrhea, Am. J. Obst. & Gynec. 53:226, 1947.
15. Rutherford, Robert N., Presacral Neurectomy: A Gynecological and Obstetrical Follow-up, West. J. Surg. 50:597, 1942.
16. Doyle, Joseph Bernard, Paracervical Uterine Denervation by Transection of the Cervical Plexus for the Relief of Dysmenorrhea, Am. J. Obst. & Gynec. 70:1, 1955.
17. Phaneuf, Louis E., Presacral Neurectomy in Intractable Dysmenorrhea, New York Mount Sinai Hospital Journal 14:553, 1947-48.
18. Greenhill, J. P., Sympathectomy for the Relief of Pelvic Pain in Women, New York Mount Sinai Hospital Journal 14:363, 1947-48.
19. Meigs, Joe Vincent, Excision of the Superior Hypogastric Plexus (Presacral Nerve) for Primary Dysmenorrhea, Surg., Gynec., & Obst. 68:723, 1939.
20. Marshall, Samuel F. and Kennedy, R. J., Postoperative Results Following Presacral Neurectomy, Surg. Clin. North America 25.1:518, 1945.
21. Bunts, Alexander T., Technic of Resection of the Presacral Nerve for Dysmenorrhea, Surg. Clin. North America 16.2: 1031, 1936.
22. Sedgwick, Cornelius E., Presacral Neurectomy, Surg. Clin. North America, 29.1:861, 1949.
23. Colcock, Bentley P., Presacral Neurectomy for the Relief of Severe Primary Dysmenorrhea, Surg. Clin. North America June, 1941. P. 855.
24. Marshall, Samuel F., and Pappen, James L., Presacral Neurectomy in the Treatment of Dysmenorrhea, Surg. Clin. North America 17.1:927, 1937.
25. Phaneuf, Louis E., Surgical Measures in Dysmenorrhea, New England Journal of Medicine 231:872, 1944.
26. Ingersoll, Francis M. and Meigs, Joe V., Presacral Neurectomy for Dysmenorrhea, New England Journal of Medicine 238:357, 1948.

27. Pemberton, Frank A., Resection of the Presacral Nerve in Gynecology, New England Journal of Medicine, 213:710, 1935.
28. White, James C., Conduction of Visceral Pain, New England Journal of Medicine 246:686, 1952.
29. Cannaday, John E., Presacral Nerve Resection for the Relief of Pelvic Pain and Dysmenorrhea, Annals of Surgery 103: 886, 1936.
30. Wetherell, Fredericks, Intractable Dysmenorrhea: Relief by Sympathetic Neurectomy, Am. J. Obst. & Gynec. 29: 334, 1935.
31. Masson, James C. and Shoemaker, Rosemary, Surgical Treatment of Dysmenorrhea, Am. J. Obst. & Gynec. 36:441, 1938.
32. Black, William T., Presacral Sympathectomy for Dysmenorrhea and Pelvic Pain, Annals of Surgery 103:903, 1936.
33. Cotte, Gaston, Technic of Presacral Neurectomy, Am. J. Surg. 78:50, 1949.
34. Kindel, Elmore A., Pelvic Pain in Women: Treatment by Resection of the Superior Hypogastric Plexus, Report on Thirty-nine Cases, Am. J. Surg. 30:435, 1935.
35. DeCourcy, Joseph L., Resection of the Presacral Nerve for Dysmenorrhea; Based on Favorable Results in a Series of Twenty-one Cases, Am. J. Surg. 23:408, 1934.
36. Abbott, Walter D., Resection of the Presacral Nerve for Dysmenorrhea and Pelvic Pain, Annals of Surgery 104:351, 1936.
37. Reeves, T. Kevin and Lipman, George S., A Review of Superior Hypogastric Sympathectomies Over a Period of Ten Years, Penn. M. J. 46:1274, 1943.
38. Graffagnino, Peter, The Value of Presacral Sympathectomy in Gynecology, Southern M. J. 28:353, 1935.
39. Duncan, Christopher J., Surgical Treatment of Dysmenorrhea, New York State M. J. 46.2:2757, 1946.
40. Patricelli, Liberino, Presacral Neurectomy in the Treatment of Primary Dysmenorrhea, Northwest Medicine, 46:677, 1947.

41. Wetherell, Frederick S., Relief of Pelvic Pain by Sympathetic Neurectomy: A Report of Seven Cases in Which the Superior Hypogastric Plexus (Presacral Nerve) was Resected, J. A. M. A. 101:1295, 1933.
42. Cannon, D. J., Resection of the Presacral Nerve for Intractable Dysmenorrhea Complicated by Severe Bleeding, The Irish Journal of Medical Science 76:150, 1932.
43. Greenhill, J. P., The Treatment of Severe Dysmenorrhea by Pelvic Sympathectomy, American Medicine 40:290, 1934.
44. Schuck, Franz, Pain and Pain Relief in Essential Dysmenorrhea, Am. J. Obst. & Gynec. 62:559, 1951.
45. Wetherell, Frederick S., Resection of the Superior Hypogastric Plexus: Modification of the Technique to Prevent Regeneration, Am. J. Obst. & Gynec. 61:738, 1951.
46. Fertitta, J. J., Fertitta, Sam, and Miller, K. T., Presacral Neurectomy in the Treatment of Dysmenorrhea: A Report of 125 Cases, Surgery, 28:729, 1950.
47. Black, William T., Presacral Neurectomy: Report on 70 Cases, Southern M. J. 48:120, 1955.
48. Scully, John C., Presacral Resection for the Relief of Pain, Mich. State Med. Soc. J. 40:979, 1941.
49. Peterson, D. L., Presacral Nerve Resection, Kentucky M. J. 43:337, 1945.
50. Novak, Emil, The Problem of Primary Dysmenorrhea, New Orleans M. & S. J. 102:594, 1950.
51. Simpson, Wyatt C., The Surgical Treatment of Dysmenorrhea, J. M. A. Alabama 13:291, 1944.
52. Evans, Albert L. and Cofer, Olin S., The Treatment of Intractable Dysmenorrhea by A Presacral Sympathectomy, J. M. A. Georgia 40:41, 1951.
53. Hoffman, Lloyd C., Surgical Management of Dysmenorrhea, Nebraska M. J. 32:353, 1947.
54. Filler, William, The Treatment of Dysmenorrhea: With Special Reference to the Primary Type, Med. Clin. N.A. 35:861, 1951.

55. Greentree, Leonard B., The Mechanism of Primary Dysmenorrhea, Western J. of Surg., Obst., & Gynec. 57:578, 1949.
56. Todaro, Samuel P., Hypogastric Sympathectomy for Dysmenorrhea: Evaluation and Technic of Operation, Texas State M. J. 46:28, 1950.
57. Hendrick, James W., Resection of the Presacral Nerve for Dysmenorrhea and Pelvic Pain, Texas State M. J. 37:26, 1941.
58. Hoge, Randolph H., Resection of the Superior Hypogastric Plexus for Dysmenorrhea, Annals of Surgery 139:661, 1954.
59. Gray, Henry, Anatomy of the Human Body. Philadelphia, Lea and Febiger, 1956. 26th Edition, p. 1112.
60. Boelter, William C., Personal communications with the author.