A COMPARISON OF IMAGERY RELAXATION AND AN EDUCATIONAL TREATMENT MODALITY FOR DYSMENORREA

DISSERTATION

Presented to the Graduate Council of the North Texas State University in Partial Fulfillment of the Requirement

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By

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This study is a comparison of four treatments involving education and imagery relaxation for the amelioration of dysmenorrhea. Treatment was presented to 76 subjects by videotape during a one-hour session. A six month follow-up was performed using one of the original instruments, the Symptom Severity Scale (Cox & Meyer, 1978) and a questionnaire designed for the study.

Analysis of the test instruments indicated a significant treatment effect for the educational group. The second most effective treatment was a combined treatment utilizing imagery relaxation and education, although this group did not produce significant results. The no-treatment control group was more effective in diminishing symptoms than the fourth group, imagery relaxation alone.

The lack of effectiveness of the imagery relaxation treatment was hypothesized to be due to lack of reinforcement of the technique. The educational treatment modality offered the individual an opportunity to learn about many different etiological facets of dysmenorrhea, including
biological, learning, and cognitive factors. The presentation also introduced the individual to several different treatment modalities in order to provide an armamentarium of effective methods for diminishing or eliminating dysmenorrhea. These results suggest that there is a need for education about dysmenorrhea before menarche, in order to prepare, prevent, treat, and cope with this syndrome.
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A COMPARISON OF IMAGERY RELAXATION AND AN EDUCATIONAL TREATMENT MODALITY FOR DYSMENORREA

Dysmenorrhea is defined as menstrual pain and difficulty with menstrual flow. Included in this classification are two subtypes, primary and secondary. Primary dysmenorrhea includes pain not associated with pelvic pathology, whereas secondary dysmenorrhea is due to pelvic pathology of any origin such as anatomical malformation. Dysmenorrhea should also be distinguished from premenstrual syndrome which does not usually include pain as a manifesting symptom. Dysmenorrheic pain is related to the first day of the menstrual flow and is present during each menstrual cycle, continually, from menarche or within a year afterwards (Dawood, 1981). It is the greatest, single cause of lost work hours among women (Leannane, 1980). An estimated one-half or more of all young women experience dysmenorrhea severe enough to seek relief (Dawood, 1981).

Leannane (1980) describes the history of dysmenorrhea and states that symptoms and various treatments for this disorder were first found in the ancient writings of Greek and Chinese physicians. Many remedies have been prescribed through the centuries as well as suggestions regarding women's lifestyles, hygiene, diets, habits and restrictions
during the menstrual phase. Taboos, such as compulsory separation of menstruating women, were found in many ancient cultures and are still a part of some contemporary societies such as the Navajo Indian. The Zeitgeist regarding menstruating women has changed from "delicate semi-invalids" to almost equal members participating fully in our society. During the last two decades, there has been an increase in the number of women presenting to physicians' offices with symptoms of dysmenorrhea and this is perhaps due to the dramatic increase of women in the American workforce (Leannane, 1980).

Several theories have been postulated regarding the cause of dysmenorrhea. Each of the following theories will probably prove to be an etiological component for the multifaceted dysmenorrhea. Shick, (as cited in Pickles, 1979), proposed the existence of a chemical, "menotoxin", circulating in the blood during menstruation which he thought to be responsible for the negative symptomology of dysmenorrhea. No specific, unique chemical has been isolated during menstruation. Pickles (1979) observed that some menstruating women who arrange cut flowers cause the flowers to wilt. He postulated that an unidentified substance may be secreted via the sweat glands during menstruation which causes the flowers to wither and he further speculates that this substance may inhibit the synthesis of
the neurotransmitter dopamine. He concludes that this interruption of dopamine production may be partially responsible for emotional changes during menstruation.

Anovulatory women seldom have dysmenorrhea and therefore hormones are thought to be a contributing factor. Oral contraceptives which include a combination of hormones reduce dysmenorrhea (Dawood, 1980). The action of estrogen and progesterone on the myometrium and endometrium has not been completely explained because the receptor sites for these hormones in the uterus have not been isolated. The gonadotrophins, prolactin, and vasopressin, which are all hypothalamically controlled, are also postulated to play a role in dysmenorrhea. Antidiuretic hormone (ADH) stimulates the uterus at the onset of menstruation causing a decrease in blood flow by acting directly on the vessels of the uterus. Ackerlund (1979) demonstrated, by intravenous administration of ADH, that uterine contractions precede the decrease in blood flow, lending further credibility to the implication of hormonally induced, hypothalamic secretions.

Until the 1930's, treatment for dysmenorrhea was extremely frustrating for both the patient and physician. A correlation was observed at the beginning of this decade between anovulation and a lack of menstrual pain. Wilson and Kurzrok, (as cited in Henzel, Massey, Hanson, Buttran,
Rosenwaks, & Pauls, 1980), began to treat dysmenorrhea by administering estrogen. The rationale for this therapy was elucidated in the 1940's by Sturgis and Albright, who demonstrated that the administration of estrogen suppressed ovulation and also diminished uterine contractions during the menstrual phase (Henzel, et al, 1980).

The discovery of prostaglandins (PG) in 1970 and the isolation of PGs, which are biologically active in the reproductive cycle, lead to a new method of treatment for dysmenorrhea. An explanation of the physiology involved in PG production and PG effects on the body is presented in Appendix J. PGs have been demonstrated to partially mediate the abnormal activity of the uterus during dysmenorrhea as well as an increase in the sensitivity of pain receptors (Ackerlund, 1979). PGs are produced in the endometrium and their synthesis is stimulated by estrogen and progesterone (Dawood, 1981).

The myometrium may be more sensitive to mechanical and chemical stimulation in dysmenorrheic women. Evidence for this has been demonstrated by measuring the parameters of myometrial contractions (Ackerlund, 1979). Frequency is the number of contractions per unit time and amplitude is the pressure of the contraction and is highest during peak measurement. Asynchronous contractions are different patterns of amplitude and frequency recorded concomitantly at
different uterine and cervical sites. Pressures vary within individuals and during different phases of the menstrual cycle (Dawood, 1981). During the proliferative phase, normal baseline tone is 10-25 mmHg. During periovulation the resting tone elevates to 40-60 mmHg. In the secretory phase the level drops to 10-30 mmHg. The onset of menstruation increases the tone to 50-150 mmHg in normal populations (Dawood, 1981). Ackerlund (1979) has demonstrated, using the microtransducer technique, that dysmenorrheic women often have tones ranging from 200-350 mmHg during their reports of painful cramps. These women also report increased frequency of contractions and increased asynchrony (Ackerlund, 1979). The above data reflect a spastic hypercontractility pattern. High tone (resting level) over the entire menstrual cycle as well as between contractions are also indicative of dysmenorrhea (Dawood, 1981).

Uterine blood flow is diminished during dysmenorrhea. Resulting uterine ischemia is thought to be responsible for a portion of the pain. Patients who report prolonged pain in the form of a continuous ache have demonstrated, clinically, a prolonged fluctuation in uterine blood flow. When blood flow is measured as being high, patients subjectively report moderate pain, but when the blood flow levels are low, they report severe pain (Ackerlund, 1979). Ischemia, caused by hyperactivity of the myometrium compressing the
uterine vessels is another component of dysmenorrhea (Dawood, 1981).

Menstrual clots and subsequent cervical obstruction may cause uterine distension and provoke hyperactivity of the myometrium due to stretching of the muscle fibers. Lack of blood flow may also cause an increase in absorption of prostaglandins by the uterus. Cervical factors are estimated to play a minimal role in most cases of primary dysmenorrhea (Dawood, 1981).

The uterine nerves have both adrenergic and cholinergic components and are thought to be involved in menstrual pain because of the following findings. Patients with Myasthenia Gravis, treated with choline esterase inhibitors, report an onset of dysmenorrhea subsequent to the administration of these drugs, suggesting that the cholinergic system may be involved (Ackerlund, 1979). Short, adrenergic neurons are abundant in the nullipara woman, but disappear after pregnancy. Some dysmenorrheic women do not experience symptoms after delivery of their first child. This suggests that adrenergic neurons may be involved (Dawood, 1981). Another set of nerves has been isolated containing peptides such as substance P and other polypeptides. Substance P may be found in the peripheral endings in primary sensory neurons and if diminished, subjects report a concomittant decrease in pain (Sjoberg, 1979).
All of the above physical findings contribute to dysmenorrhea within any given individual. Psychological factors also play an integral role in the manifestation of this syndrome. Prior to the development and increasing use of birth control pills, most physicians attributed dysmenorrhea to psychological factors only. In a 1983 monograph on dysmenorrhea (Harvey) a chronology of important factors reveals that in 1972, Pickles recommended the use of non-steroidal anti-inflammatory drugs (NSAID) for the treatment of dysmenorrhea. These are the current drug therapies for dysmenorrhea and approximately 70 percent of dysmenorrheic women have reduced their menstrual pain using this class of drugs (Rosenwaks, 1982). In 1973, psychology texts stated that "dysmenorrhea is caused by women feeling ambivalent about their sexual and feminine roles owing to early anxieties, doubts, or lack of healthy identification and training for feminine sexual, maternal, and homemaking roles" (Harvey, 1983, p. 26). In 1977, medical encyclopedias still described dysmenorrhea as a "functional disease, reflecting a resentful attitude toward menstruation, sexual identity, and sexual attitudes" (Harvey, 1983, p. 27). Psychiatry and gynecology texts as late as 1980 assert that the "appropriate treatment for dysmenorrhea is psychotherapy, but that there is little that can be done for the patient who prefers to use her menstrual symptoms as a monthly refuge
Classical psychotherapy, as stated above, did not alleviate the symptoms of dysmenorrhea. Behavioral therapy was then initiated. Behavioral therapy for dysmenorrhea may be categorized into self-control modalities, and techniques such as desensitization, biofeedback, hypnosis, and exercise. These orientations have evolved into the teaching of coping strategies for the self management of pain (Denny & Gerrard, 1981).

Hypnotherapy focuses on education about dysmenorrhea and menstruation indicating that menses is a normal process and that it will be painless. Cervical muscle relaxation is emphasized as well as deep breathing and focal anesthesia. Hypnoanalysis may be utilized if difficulty persists. Underlying psychological conflicts are investigated in order to alleviate the source of uterine congestion and then it is suggested that the client reinterpret the pain as merely congestion (Rees & Scott, 1982).

Exercise involves training in progressive muscle relaxation (PMR), shifting of attention to nonpainful stimuli, light abdominal massage, and exercise to increase circulation, thereby relieving pelvic congestion (Fleischauer, as cited in Denney & Gerrard, 1982). Shangold (1983) states that women who exercise regularly report reduction
of menstrual pain. She postulates that this may be "due to exercise-induced production of endorphins and/or vasodilating PGs" (p. 16). This may increase their threshold for pain. She further states that female athletes may substitute the exercise for attention to their menstrual cycle and thus decrease the awareness of pain.

Denney and Gerrard (1982) compared several studies involving biofeedback procedures and desensitization-based strategies for the amelioration of dysmenorrhea. Desensitization, as defined by Mullen (1968, as cited in Quillen & Denney, 1982), includes PMR and practice in the imagery of scenes concerning menstruation. Mullen postulated that anxiety of menstruation may exacerbate the painful symptomology of dysmenorrhea. Significant reduction in menstrual symptoms were found by the following authors utilizing desensitization procedures: Quillen and Denney (1980), Cox and Meyer (1978), Chesney and Tasto (1975), Tasto and Chesney (1978), Reich (1972), and Mullen (1968), (as cited in Denney and Gerrard, 1982). Carcelli (1986) found relaxation, desensitization, and a combination of these to be equally effective in reducing dysmenorrheic symptoms, negative attitudes, and pain behaviors. Duson (1976) and Rosenthal (1978) found nonsignificant reductions in menstrual symptoms and other negative effects (as cited in Denney and Gerrard, 1982). Wood (1982) also found no sig-
nificant differences using systematic desensitization and cognitive coping skills training.

Quillen and Denney (1982) promote the rationale that voluntary, active self-control rather than passive acceptance of dysmenorrheic symptoms underlies the successful, behavioral treatment of this disorder. They included training in relaxation and positive coping strategies, guided rehearsal of symptoms in the treatment setting, and home practice in their treatment regimen. Their results were significant at the end of the study and at eighteen month follow-up. They also reported that the subjects generalized the coping strategies to other areas of their lives and especially in the realm of interpersonal relationships. One finding of particular interest in this study was the delay of onset of menses after pain management intervention. The authors attributed this phenomenon to either a physical (focal relaxation) or psychological (anticipation of the next menses) side effect. They concluded that it is necessary for "women to acquire control over menstrual pain and receive repeated demonstrations of this control during several menstrual cycles" (p. 128).

The biofeedback studies that were reviewed by Denney and Gerrard (1982) yielded many conflicting results. Sedlecek and Heczey (1977), using frontalis EMG and hand and vaginal temperature biofeedback reported all three of their
cases improved on this regimen. Tubbs and Carnahan (1976) utilizing frontalis EMG and hand temperature biofeedback found this treatment to be effective with 50 percent (N=8) of their subjects. Heczey (1975) compiled a therapy of vaginal temperature biofeedback and autogenic training. He found no significant differences in the reduction of symptoms but did report an increase in all subjects' thermal biofeedback readings. Dietvorst and Osborne (1978) examined hand temperature biofeedback and autogenic training and found partial relief of symptoms in a single case study. Russ (1976), using hand temperature biofeedback alone, reported significant reduction in symptoms while Clayman and Simkins (1975) did not. Denmark (1975) combined alpha-EEG biofeedback with autogenic training and found no differences (As cited in Denney and Gerrard, 1982).

Balick, Elfner, May, and Moore (1982) used frontalis EMG and thermal biofeedback coupled with autogenic relaxation practice and reported significant decreases in the following: the amount of time in bed; the amount of interference with daily activities; the severity and duration of the dysmenorrheic symptoms; and the amount of medication administered. Bennink, Hulst, and Benthem (1982) compared relaxation training and EMG biofeedback and concluded that the concomitant use of these procedures is superior to
relaxation training alone. These authors also point out that one reason for previously reported mixed results was because of the placement of the EMG electrodes. The protocol of their study placed the electrodes on the surface of the lower abdomen which they felt more reasonably reflected the myometrial contractions.

Conflicting results continue to be presented. Anderson (1983) found clinical meditation and relaxation to be an effective treatment for total symptom severity. Mohler (1984) found NSAIDs to decrease painful symptoms of dysmenorrhea and cause less disruption of activities of daily living. Behavioral treatments utilizing hypnotherapy, biofeedback, Lamaze exercises, and desensitization procedures were not effective in reducing pain but did reduce negative affect towards menstruation and disruption of daily living. Shebroe (1985) found psychological (PMR) and drug therapies to be equally effective.

It appears that a multimodal approach may be the most efficacious treatment for dysmenorrhea. It is suggested that biofeedback procedures should be directed towards the pathophysiology of the disorder (Dawood, 1981). Increased vaginal temperature appears to offer relief to a small number of people who have been studied. Abdominal EMG electrode placement requires very expensive, frequency band filtering systems to block out diaphramatic, arterial (de-
ascending aorta and other large abdominal arteries) and abdominal wall muscle interference. All of these above cited behavioral studies involve intensive training utilizing many contact hours which are costly and time consum-

Social learning influences appear to be another determinant in the individual's perception of dysmenorrheic pain. Many anthropological studies have found isolated cultures where dysmenorrhea does not exist. In these cultures no parental or sibling example has been provided for pain behavior associated with the menstrual cycle. Modeling studies (Craig and Prkachin, as cited in Dawood, 1981) have demonstrated a lower physiological activity when shocked subjects are exposed to a tolerant model. Adolescents who have dysmenorrheic adults as their role models may learn that pain behavior can be rewarded by increased attention and lack of responsibility to normal duties during the menstrual phase. The anticipated rewards and punishments of pain behavior fit nicely into the individual's information processing system. Pain is a function of many modalities operating within the human body. It traverses the spectrum from the cellular level of sensory reception to one of the highest cognitive levels, cultural values. It may be beneficial to assess the motivation of an indivi-
dual towards a learning, coping, and self-control approach involving health therapy.

The discovery of the role of PGs as the main etiologic factor of dysmenorrhea has changed the assumptions that faulty personality, inadequate coping strategies, and increased pain awareness are responsible for this disorder. Bloom, Shelton, and Michaels (1978) administered the MMPI, Tennesse Self Concept Scale and the Personality Research Form to their subjects. They concluded that there is no significant difference in the personalities of normal and dysmenorrheic women. Aberger, Denney, and Hutchings (1983) administered the Multiple Affect Adjective Check List, the McGill Pain Questionnaire, and the Coping Strategies Questionnaire to 423 dysmenorrheic and nondysmenorrheic women. The experimental procedure precipitated a pain experience using a blood pressure cuff to induce ischemic pain. The purpose of the study was to measure pain thresholds and tolerance. While these parameters were greater when the subjects were in the premenstrual phase of their cycles, no significant differences were found between the dysmenorrheic subjects and the normal controls in their effectiveness to control pain behavior. No studies to date have found any significant personality factors which correlate with dysmenorrhea directly.
Menstruation begins for most women between the ages of 10 to 14 years and continues until menopause which occurs around the age of 45 to 50 years. In the United States today there is a trend to delay child birth and thus increase the years that dysmenorrhea may be more severe. Over 50 percent of the women in America are currently employed in the work force. Many of these women feel obligated to maintain their attendance at their place of employment or school and then acknowledge their dysmenorrheic symptoms during their rest hours. It therefore appears that time consuming treatment will not be utilized sufficiently as more time constraints are placed on this population.

Drug therapy is certainly beneficial and the recent release of ibuprofen to an over the counter drug status by the Federal Drug Administration has made NSAIDs more available to a large segment of the population. A detailed description of the drugs available for the treatment of dysmenorrhea is provided in Appendix K.

Relaxation techniques have been effective for the relief of dysmenorrhea and coupled with drug therapy, they help about 80 percent of the women who are aware of the availability of these treatments. However, only 10 percent of the population actually seeks help from a physician for this disorder (Sobczyk, 1980). A review of the elementary,
junior high, and high school health textbooks available at the North Texas State University Textbook Library reveals a serious deficit in the presentation of information about dysmenorrhea. No sex education textbooks mentioned the etiology of dysmenorrhea or effective treatments. Family and peers appear to be the main source of education about this syndrome and factual information about this problem has changed considerably within the past decade.

Therefore, the purpose of this study is to test the effectiveness of a videotaped presentation, about dysmenorrhea and its current treatment, to reduce menstrual pain and its accompanying side effects. These treatments will not be reinforced by any therapy other than the original presentations. It is hypothesized that an imagery relaxation treatment coupled with an educational videotape explaining dysmenorrhea and its treatment, will be more effective in relieving menstrual cramps and changing attitudes towards menstruation and dysmenorrhea than either of these treatment components separately. The dysmenorrhea videotape will include information about the physiology of the menstrual cycle, associated female anatomy, pain behavior with accompanying secondary gains, and existing pharmacological interventions. The imagery relaxation treatment will provide a simple technique which can be learned in 10 minutes and utilized within one minute. It will focus on
breathing and associated relaxation imagery. Transcripts of the videotapes are presented in Appendices F and G and include a flow chart diagram of symptom progression.

Method

Subjects

Eighty-eight volunteer women originally participated in this study. They were recruited from the undergraduate psychology classes at North Texas State University and they received four extra credit grade points for their participation as an incentive. The subjects were randomly assigned to four treatment groups: No Treatment Control (NTC), Education (ED), Imagery Relaxation (IR), and Combined Education plus Imagery Relaxation (CMB). The subjects ranged in age from 18 to 43 years, with the following mean age per treatment group: (NTC) 22 ± 7 years; (ED) 19 ± 1 years; (IR) 19.5 ± 1.5 years; and (CMB) 21 ± 3 years. Randomization of the subjects reflected the following cultural distribution: (NTC) 15 Caucasian, 4 Black, 1 Hispanic; (ED) 15 Caucasian, 4 Black, 1 Asian; (IR) 12 Caucasian, 6 Black; (CMB) 12 Caucasian, 6 Black, and 1 Hispanic. The mean age of menarche was: (NTC) 12.5 ± 1.7 years; (ED) 12.5 ± 1.5 years; (IR) 12.4 ± 1.5 years; (CMB) 12.6 ± 1.0 years.
Instruments

Health Attribution Test (HAT). Developed by Achterberg and Lawlis (1980), this instrument lists 22 statements regarding individual perceptions of health factors. Subjects respond to each statement with one of the following categories: Strongly agree, agree somewhat, slightly agree, slightly disagree, disagree somewhat, and strongly disagree. These responses are then factored into three variables of health attribution: Internal, powerful others, and chance. Each response is scored on a scale from minus three to plus three and the scores in each variable are added for three total raw scores. These scores are then transformed to standardized scores from an interval table. The standardized scores (STEN) are divided by the sum of the three STENs for percentile scores which when totaled equal 100 percent.

Symptom Severity Scale (SSS). This scale, developed by Cox and Meyer (1978), lists 18 symptoms which are associated with dysmenorrhea such as abdominal pain, cramps, headache, irritability, digestive system disturbances, and dizziness. Frequency of each symptom is rated on a five-point scale from nonoccurrence of the symptom to lasted for several days. Severity is also rated on a five-point scale which ranges from unnoticable to very severe. The indivi-
dual is asked to rate the symptoms for the last menstrual period. See Appendix C.

Procedure

All subjects read the description of the study (Appendix A) and signed the consent form (Appendix B). They were assigned to a treatment group and completed three forms including the above named instruments and a demographic data form which included medical history questions (Appendix D). The (NTC) group received no further treatment. The three other treatment groups then watched their respective videotapes which were original productions for this study. The (IR) and (CMB) groups were given home practice sheets for the (IR) treatment which requested a daily notation of practice for three months. They were also given a stamped, addressed envelope for the return of the practice sheet. All subjects were invited to ask questions before they left the contact room and the (IR) and (CMB) groups were encouraged to practice the treatment.

A six month, follow-up questionnaire was completed via telephone during the second week of the subject's menstrual cycle in order to control for consistent recall of the last phase. The follow-up included a retest of the SSS and a series of questions related to the treatments (Appendix E).
Results

Seventy-six of the initial 88 subjects were available for the follow-up study and therefore 12 of the original subjects were eliminated from the study. The total number of subjects remaining in each group was (NTC) 20 subjects, (ED) 20 subjects, (IR) 18 subjects, and (CMB) 18 subjects.

The effectiveness of the four treatments was analyzed by two different instruments. The first was the symptom severity scores and the second was a general statement about rating the last menstrual period. The frequencies of the 18 symptoms by group is described in Appendix H. A repeated measures, multivariate analysis of variance (MANOVA) revealed no significant differences between the treatment groups on the pretest ($p > .11, F = 1.29, df = 18, 72$), with the exception of the symptom dizziness. A one way analysis of variance (ANOVA) shows a significant difference between the groups ($p < .03, F = 3.36, df = 3, 72$), on this pretest variable. The MANOVA of the post-test symptoms revealed a questionable significance between the groups, over time, ($p = .058, F = 1.41, df = 18, 72$). The symptoms which showed the most significant positive change were depression, dizziness, and appetite loss. A oneway analysis of variance, with a Scheffe multiple comparison, revealed a significant difference between the (NTC) and (IR) groups ($p < .05, T = 2.078, df = 3, 72$) for appetite loss, between
the (NTC) group and both the (ED) and (CMB) treatment groups for depression, and no significance between the groups for dizziness. The contrast comparison also showed a significant difference between the (ED) and (IR) groups for effectiveness of treatment as measured by the total symptom severity improvement score. Table 2 is a summary of the above variances and contrasts.

Table 2

<table>
<thead>
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<th>Symptom Improvement</th>
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<tr>
<td><strong>Contrast</strong></td>
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<tr>
<td><strong>Appetite Loss</strong></td>
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<tr>
<td>NTC and IR</td>
</tr>
<tr>
<td><strong>Depression</strong></td>
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<tr>
<td>NTC and ED</td>
</tr>
<tr>
<td>NTC and CMB</td>
</tr>
<tr>
<td><strong>Total Symptoms</strong></td>
</tr>
<tr>
<td>ED and IR</td>
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</table>

A MANOVA of the second measurement instrument revealed no significant differences between the groups over time on a general statement about severity of menstrual pain ($p > .80, F = .32466, df = 3, 73$).
The drugs taken by the subjects, either effective or non-effective were analyzed as a covariant of the improvement demonstrated on the SSS and had no effect on the outcome ($p > .76$, $F = 0.97$, $df = 2, 65$). The HAT and the SSS results were analyzed by a MANOVA with the HAT as a covariant and no significant difference was found on either the pretest ($p > .51$, $F = .88$, $df = 6, 63$) or post-test ($p > .10$, $F = 1.83$, $df = 6, 63$).

Forty-four percent ($N = 34$) of the subjects had a negative attitude towards menstruation while 42% ($N = 32$) felt ambivalence. Eighty percent ($N = 60$) of the subjects described negative feelings about menstrual pain while 20% ($N = 16$) felt that menstrual pain was not severe and could be tolerated. Twenty-three percent ($N = 18$) of the subjects said the treatments changed their opinion about menstruation in a positive direction and 33% ($N = 25$) stated that the treatments changed their opinion about menstrual pain to a more positive outlook. Sixty-eight percent ($N = 52$) of the subjects reported the treatment they received helped their menstrual pain.

Sixty-two (80%) of the subjects had never seen a physician for menstrual pain. Five (6%) of the subjects had delivered children while 94% of the subjects had no pregnancies. Fifty-one (66%) of the subjects did not use birth control pills. Table 3 shows the number of subjects
in each treatment group who used effective versus non-effective medications for treatment of their dysmenorrhea. Effective drugs include birth control pills, nonsteroidal anti-inflammatory drugs except aspirin, and Class II analgesics. Noneffective drugs include OTC menstrual pain preparations, aspirin, and diuretics.

Table 3

Use of Effective and Noneffective Drugs for Dysmenorrhea

<table>
<thead>
<tr>
<th>Group</th>
<th>NTC</th>
<th>ED</th>
<th>IR</th>
<th>CMB</th>
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<tbody>
<tr>
<td>Effective Drug</td>
<td>6</td>
<td>9</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Non-effective Drug</td>
<td>11</td>
<td>8</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>No drug taken</td>
<td>3</td>
<td>3</td>
<td>8</td>
<td>6</td>
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</table>

In response to the question of how much dysmenorrhea interrupts daily functioning, on follow-up, 17 (22%) of the subjects reported that it did not interfere at all, while 8 (10%) subjects reported that it totally interrupted their ability to carry out activities of daily living. Table 4 shows the number of subjects of each group who responded to this statement on a nine-point graded, interference scale.
Table 4
Dysmenorrheic Interference of Daily Functioning

<table>
<thead>
<tr>
<th>Group</th>
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<td>1</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>CMB</td>
<td>6</td>
<td>2</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Totals</td>
<td>17</td>
<td>12</td>
<td>12</td>
<td>3</td>
<td>4</td>
<td>15</td>
<td>4</td>
<td>2</td>
<td>8</td>
</tr>
</tbody>
</table>

* 1 is keyed as no interference with daily functioning to 9 which represents total disruption of daily functioning.

The (IR) treatment was practiced by 17 subjects in both the (IR) and (CMB) groups initially. At follow-up, 11 subjects in each group reported that they were still using the treatment. Fifteen subjects of the (IR) group and 16 subjects of the (CMB) group stated that their respective treatments were effective. Only eight subjects stated that they did not learn new information from the videotapes. Ten of the 20 (ED) subjects stated that the educational treatment helped them and they stated that increased exercise and commitment to functioning were the two most important factors they learned. Sixteen of the 18
(IR) subjects reported that their treatment helped. The most frequent statement reflected the ability to relax and gain control when facing pain. Fourteen of the 18 (CMB) subjects stated improvement with this treatment.

Sixty-six percent (N = 51) of the subjects stated that they had never received any education about dysmenorrhea. Seven subjects reported that their mothers had discussed painful menstruation with them, but none had given an explanation of why the pain occurs. Eight subjects stated that their physician had prescribed medication for menstrual pain without any explanation. Six subjects stated that a physician had explained dysmenorrhea partially when writing a prescription and four subjects had learned about dysmenorrhea in their college biology courses. In response to the question of how well prepared the subjects were for menstruation, five (7%) stated they had no preparation, 14 (18%) stated they knew very little, 35 (46%) said they were prepared somewhat, and 23 (30%) stated they felt well prepared.

All of the subjects stated that education about dysmenorrhea should be included in health classes.

Discussion

The hypothesis proposed in this study was not supported. The (CMB) group did not demonstrate the most significant amelioration of the dysmenorrheic symptoms.
All of the treatment groups showed improvement. The (ED) group showed the most improvement. This was significantly different from the (IR) group which was hypothesized to be the second most effective treatment. While the (CMB) treatment group did show the second highest degree of improvement, this was not significantly different from the (NTC) group.

The significant improvement demonstrated by the (ED) group and the second place performance by the (CMB) group lends credibility to the idea that education about this syndrome may be one of the most effective means of treatment. This study involved only one contact hour and it is suggested that three to five hours of education may be even more effective. Since this topic is not being addressed in health textbooks, perhaps it may be beneficial to include it for the age group of 10 to 15 years. Dysmenorrhea affects over 50 percent of the female population in the world, and there are more females in the population than males. Indirectly, this syndrome affects the male population as well, especially in the areas of understanding the nature of the pain, and the diminished female functioning for approximately 24 days annually.

The suggested reasoning for the significant improvement of the (ED) group is the variety of explanations presented to the subjects with which they may individually
identify, as well as the numerous treatments which may be selected on an individual basis. The varied treatments also provide each woman with an armamentarium to deal with this problem.

The least significant improvement, demonstrated by the (IR) group suggests that this type of treatment probably needs more reinforcement for effectiveness. Previous studies have shown that this treatment is effective, however they involved considerably more treatment contact hours over several weeks and this support and attention does contribute to the overall effectiveness of any treatment. The majority of the subjects practiced this treatment initially, however, over time, the practice of this treatment was not sustained.

Although the results of this study were not anticipated, it is apparent that there is a need for education about dysmenorrhea. All the biological factors are not yet known, however the role of prostaglandins is an important contributing factor and demonstrates that there is a biological basis for dysmenorrhea. Psychological factors contribute to pain behavior, however it is suggested that a greater understanding of the etiology of the biological and cognitive factors would lead to less negativity regarding this topic. Menstruation is a healthy, normal function of the female reproductive role and a more positive attitude
appears to be warranted. Medical research continues to provide us with explanations and improved treatments for dysmenorrhea. Currently, menstrual pain can be ameliorated significantly and in some cases eliminated. If this information is not properly disseminated, negative attitudes about menstruation, menstrual pain, and periodic female behavior will continue unnecessarily.

Onset of menstruation occurs usually between the 10th and 14th year. Preparation for this potential problem and factual suggestions for dealing with it are needed at the onset of the problem. While college level courses are addressing the issue in both biology and sex education courses, information is needed six to eight years earlier. Health text books address the problem of venereal disease in the sixth grade, and this problem affects less of the population than does dysmenorrhea. Education, as demonstrated by this study, is an effective treatment and therefore it is suggested that this topic be thoroughly presented in elementary and junior high school textbooks.
Appendix A

EXPLANATION OF THE STUDY

Dysmenorrhea, or painful menstruation (cramps), affects over one-half of all the women in the world. Only about 10 percent of these affected women seek medical help for their discomfort. This painful syndrome is the single, greatest cause of lost work and school hours among young women today.

Some drugs have been shown to be beneficial in the treatment of dysmenorrhea. Other methods, that do not use drugs have demonstrated some effective relief from menstrual pain and discomfort. One of these methods is a behavioral medicine treatment.

In this study you will be asked to participate in one of four groups. You may not know which group you are assigned to until the end of the study. You may be contacted at home by telephone during the next two to three months. Today, if you agree to participate in this study, you will be asked to fill out some questionnaires and to watch a video tape which may be about menstruation, dysmenorrhea, or its treatment.

After the completion of the study you may watch any of the other experimental groups' video tapes. If you do not fully understand this explanation, please ask the experimenter for clarification.
Appendix B

INFORMED CONSENT

Name of Subject: ____________________________

I volunteer to serve as a subject in research conducted by Sally S. Skewis, Leon A. Peek, Ph.D., psychologist, and their associates, into the effects of information about menstruation and dysmenorrhea (painful menstruation). I understand that there may be different groups of subjects, including comparison groups, which have different programs.

I understand that I will be asked to complete questionnaires and watch video tapes about menstruation. I may withdraw my participation at any time by telling the researchers. I further understand that at the end of the study I may have a summary of its findings. All information will be confidential and my name will not be associated with the data after I have completed the study.

I have had all my questions about the study answered to my satisfaction.

Date: ____________________________

Subject: ____________________________
APPENDIX C

SYMPTOM SEVERITY SCALE

Please rate each of these conditions for the frequency and severity of occurrence, on the basis of your experiences of your last menstrual period. Total frequency refers to the total amount of time you experienced one of these symptoms during your last period. Average severity refers to the average level of pain or distress of the symptom.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency Rating</th>
<th>Severity Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cramps</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Nausea</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Vomiting</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Appetite Loss</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Headaches</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Backaches</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Leg aches</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Dizziness</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Weakness</td>
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<td>0 1 2 3 4</td>
</tr>
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</table>
### Appendix C—continued

<table>
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<tr>
<th>Symptom</th>
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<th>Severity Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diarrhea</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Facial blemishes</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Flushing</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Sleeplessness</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>General aching</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Depression</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Irritability</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
<tr>
<td>Nervousness</td>
<td>0 1 2 3 4</td>
<td>0 1 2 3 4</td>
</tr>
</tbody>
</table>

How much additional time did you spend in bed because of menstrual problems over the duration of your last period? Give estimated, total number of hours:

Considering the number of pills (any kind) taken for menstrual relief, and the number of days you take such medication, how many pills did you take during your last menstrual period?

Appendix D
DEMOGRAPHIC DATA

Circle the answer where appropriate.

Name: ________________________________

Date of Birth: ___/___/____ Age: __________

Race: __________________ Place of Birth: __________

Religious Preference: __________________

School Address: ______________________________

Home phone number during the semester: __________

Best time to reach you: __________________________

Work phone number during the semester: __________

Best time to reach you: __________________________

Home phone number during the semester break: __________

Best time to reach you: __________________________

Marital Status: Single Married Divorced Widowed

Have you ever seen a physician for menstrual cramps? Yes No

What was the diagnosis? __________________________

On a scale of 1 to 10, with 10 being the worst pain you have ever had, and 1 being no pain at all, please rate the severity of your menstrual cramps.

1 2 3 4 5 6 7 8 9 10

Does anyone in your family have bad menstrual cramps or painful menstruation? Mother, Sister, Aunt, Other.
Appendix D—continued

Education: Please circle the last year completed:
H.S. = 12 years.  13  14  15  16  17

College Major: ________________  Minor: ________________

Occupation: __________________________

Do you take any daily medication?  yes  no
If yes, please state what drugs: __________________________

Do you take birth control pills?  yes  no
Do you have an I.U.D.?  yes  no

Do you take any medication for menstrual cramps?  yes  no
If yes, what do you take? __________________________

Do you take this with every period?  yes  no

Date of the beginning of your last menstrual period:
How many days does your menstrual period usually last?
3  4  5  6  7  8

Is your menstrual period regular?  Yes  No.
How many days apart are your menstrual periods usually?
25  26  27  28  29  30  31  32  33  34  35  36  37  >38

Do you ever use street drugs to control your menstrual pain?  yes  no
If yes, what drugs do you use? __________________________

Have you ever been pregnant?  yes  no
How many times?  0  1  2  3  4  5  6+
How many children do you have?  0  1  2  3  4+
At what age did you start to menstruate?
Age 9  10  11  12  13  14  15

How well prepared were you for menstruation?
not at all  knew very little  somewhat  well prepared

Please write a brief statement describing how you feel about menstruation in general.

Please write a brief statement describing how you feel about any menstrual pain you may experience.
Appendix E

POST-TREATMENT QUESTIONNAIRE

What tape did you see during the dysmenorrhea study?

Did you understand the tape?

Did the tape change your opinion about menstruation?

If yes, how?

Did the tape change your opinion about menstrual pain?

Did you learn new information from the tape?

Do you think that this would be a good tape to show in junior high school health class?

Did any of the treatments help you relieve your menstrual cramps? yes no

On a scale of 1 to 10, with 10 being the worst pain you have ever had, and 1 being no pain at all, how would you rate your menstrual pain during this last period.

1 2 3 4 5 6 7 8 9 10

Would you recommend the tape you saw to your friends?

How many days did you practice during the last month?

Did you usually practice more than once a day on the days that you practiced? Yes No

Do you think that this treatment helped you?

Did you take any medication during your last menstrual period? Yes No

If yes, what medication did you take?

Has anyone ever talked to you about menstrual pain before?
Do you think that this subject should be included in health education? yes no
On a scale of one to ten, with ten being the worst pain you have ever had and one being no pain at all, please rate the severity of your menstrual pain during your last period.

1 2 3 4 5 6 7 8 9 10
Appendix F

EDUCATIONAL TREATMENT GROUP VIDEOTAPE TRANSCRIPT

The primary focus of this study is dysmenorrhea, or painful menstruation. It is menstrual pain and cramps. It can be defined as pain that accompanies your menstrual period. There are two kinds of dysmenorrhea. The first one is called primary dysmenorrhea and starts with your first period or within the year of onset of your first period and the pain stays about the same over time. The second kind, secondary dysmenorrhea, has pain that increases over time, and as the years progress, the pain gets worse. You need to consult a physician if this is what is happening with you. Menstrual cramps are the biggest single cause of lost work and school hours among women in the world today. Also, of the people who do have menstrual cramps, about 10 percent of these women go to a physician because the pain is so severe.

Now let's briefly review the female reproductive tract. I'd like you to look at this picture of the female reproductive organs. Here we have the vagina, the uterus, the fallopian tubes with the little fimbria on the ends that collect the egg, the ovaries and the ligaments that suspend the ovaries. This purple portion here on the inside represents the lining of the uterus which is what we shed during menstruation. The uterus has a thick wall in
The discovery of substances called prostaglandins, (let's look at the spelling of this word, prostaglandin, we are going to call them PGs or chemicals from now on), helped about 70 percent of the women who have dysmenorrhea. This chemical is found in every cell of the body, and different kinds can be found in different tissues or different places in the body as well as in different concentrations or amounts. In the uterus, PGs appear to be responsible for the contractions of the myometrium or middle muscle layer of the wall of the uterus. Contractions, or the muscles hardening up is what causes the cramps. Let's look at some of the other effects of the prostaglandins (Chart shown). Here is a picture of the muscles in the middle layer of the uterus (relaxed smooth muscle picture) and this is what happens when the prostaglandins affect the muscles (contracted smooth muscle picture). Another thing that happens is that we have pain receptors (picture of pain receptors) in the uterus. These receptors get stimulated and cause us a lot of pain. (Picture of sensitized pain receptors). Another effect is that the blood vessels (picture of normally dilated arterioles) in the uterus are normally dilated so that the blood flows through there
PGs (prostaglandins) secreted

Smooth muscle in myometrium

Contracting muscles

Pain receptors

Ouch!

Normal dilated blood vessel

Vasoconstriction

Vomiting/diarrhea

Sweating

Low back pain

Dizzy

Myometrial contractions

Headache

Edema
quite readily. These PGs cause the arteries to constrict (picture of constricted arteriole) and that also causes us a problem because the blood can no longer flow readily through the uterus. As a result, these PGs, operating on our bodies, cause severe things to happen to us during our menstrual periods. (A series of pictures is now presented to relate to the following text.) We can have vomiting and diarrhea; we can have increased sweating; many of us get low back pains; some of us get dizzy; many of us have to go to bed because the pain is so bad from all those muscles cramping; many of us, like me, get bad headaches; and some of us swell up like balloons and that's called edema. Now what can we do for all these symptom problems?

If we have these symptoms in an increased amount, then we can go to the doctor and we can get a drug. The class of these drugs is called nonsteroidal, anti-inflammatory drugs and what they do is stop the prostaglandins or PGs from being manufactured or produced. These drugs really work well for us. We can get these drugs from our physician in big quantities or we can get them over the counter from our pharmacist. So, one of the treatments we can use for menstrual cramps is drugs and these drugs are against the prostaglandins. Advil or Nuprin are one of these drugs. Another treatment that we can use is the birth control pill and what this does is stop ovulation. In
other words, every month we don't produce an egg. Taking the birth control pill makes our periods more regular and it also stops an increase in flow so we don't menstruate with as much fluid for as long as we did before.

Another treatment that has been found to be effective is exercise. If you go out and run or play tennis or do something active such as aerobic exercise, this will alleviate your menstrual cramps some of the time. However, all of these treatments are only effective to a certain extent. When used in combination, they usually relieve our menstrual pain.

There is another issue that I would like to talk about today and that is how we accept menstruation in general. This, too, has an effect on how we deal with our menstrual pain. We learn from our mothers, older sisters, and school friends how they deal with menstruation. We learn that they may accept menstrual periods as normal and not pay too much attention to them or they may complain about it, saying that it's a big burden to them and resent having a menstrual period each month. Or they may have much pain and they may exhibit what we call pain behavior. All of the ways that we approach how we feel about our menstrual period are learned and what we can learn, we can unlearn, and this enables us to change. Now let us look at some of
the specific factors that we must consider when we talk about pain behavior.

The first thing is complaining about it. There is a big difference between having pain and suffering. When people ask us how we are feeling, sometimes we tell them okay or if we don't feel so good we say, "Gee, I just feel awful today". When we say the latter, people will respond to us with comfort. If we have learned to gain attention by being sick, then we are reinforced for being sick and we tend to repeat this behavior over time because it is gratifying; it is good to receive attention from others.

We also learn very early that if we are in pain, we should take medication, but pain medications are not without side effects. Medications are also expensive and it is important to try to limit our intake of these substances.

When we are not feeling up to par, most people tend to limit their activities. They put things off that they normally do, indulge themselves, and sometimes go to bed. This low activity level contributes to the overall feeling of being sick. Exercise has, in some cases, eliminated menstrual cramps.

If we are prone to a manipulative style, in other words, we tend to manipulate others to get what we need and all of us do have a bit of that in us, then we may try to use this tool more when we are in pain. We may also become
hostile towards others and this can cause people to withdraw from us. Or we may become overbearing in our complaints in order to force people to do what we want them to do for a few days.

We also may worry that the pain may continue or may even get worse. This causes much anxiety. When we get pain, we often tense up all over. We may start to feel nauseous, get sweaty, and have a whole host of other responses that go on inside our bodies. Then we may wonder how long we will be able to stand the pain. We become increasingly tired or fatigued. We become preoccupied with the pain so that we don't think about much else and we are distracted from our normal attentiveness and energy levels and thus we become irritable and grouchy because we are no longer able to deal effectively with what is going on around us. As I mentioned earlier, we tend to reduce our activity, and this partially will reduce our anxiety, but in this way, low activity levels become reinforcing or rewarding for our tenseness and it perpetuates our low activity cycle.

Now that you are aware of some of the mechanisms underlying pain behavior, and why we may act strangely when we have painful menstrual cramps, let us look at what we can do about it. We may start by saying that we are responsible individuals; after all, we are women, and we have
a lot of different abilities that we can put to good use. One of our abilities is to unlearn what we learned about pain, especially those parts which are not beneficial to us and we can teach ourselves to have a healthier lifestyle. We can also change how we think about our bodies and how we think about menstruation. We can take an active role in our health care and our treatment. We are very fortunate that we have the right to choose how we want to deal with our health, to choose if we want to have a baby or not; and with all these choices involved along the lines of female functioning, there are some checks and balances. Menstruation is one of the balances. Our monthly cycle tells us that our hormones are in balance and that we are healthy. Menstruation is really a good sign for us.

In order to unlearn some of the not so beneficial behaviors surrounding menstrual cramps, we need to adopt what we call an internal sense of control. We need to believe that we can control the pain, that we can learn to relax, take charge of the pain, and get rid of it.

You have learned many different techniques during the past few minutes. I suggest that you choose one or a combination of them and experiment until you find an effective way to control your menstrual pain. You can do it. Good luck and thank you very much for your participation in this study.
Appendix G

TRANSCRIPT OF IMAGERY RELAXATION VIDEOTAPE

This is an imagery breathing treatment to help you control your menstrual pain. We start by using our heads. We are going to use a cue-control. That is an event that triggers us to begin the sequence. It can be anything in your environment, but it needs to be something that you see often such as waiting in a line. Every time you have to wait in a line at the bank or at the supermarket or you are put on hold on the phone, you can start the sequence. When you have pain, every time you feel a cramp, you can use this technique. The first part starts by imagining a relaxing place. I always like to go to the mountains and look at the snowfall, or the pretty wildflowers, or look at a cozy fire in my nice, warm cabin. The second part of this treatment is to count slowly and to breathe with your diaphragm. Let's begin and watch me. In order to learn to breathe properly, we put one hand on our chest, and the other hand on our tummy. When we breathe in, when we inhale, our tummy needs to go out. I know this seems a little strange but you will learn quickly. This top hand never moves and that's why we need to keep it there when we first learn to breathe this way. When we exhale, or blow air out, the tummy goes back down. Now, as we do this, I want you to feel what's happening in your mouth. We're
going to breathe in slowly as if you had a straw in your mouth and you are going to take in the air through the straw. We are going to breathe slowly and in a continuous increment of air. When we get it all in, we are going to hold it for a few seconds and then we are going to let it all out. Now, I'm going to have a friend of mine count for us. We must learn to use our diaphragms. That's the muscle underneath the lungs. When we breathe in, we push the tummy out and when we breathe out, the tummy goes in. Let's get into position and get ready to start. Inhale, two, three, four, five, hold it, exhale, four, three, two, one. Now breathe normally. Remember, as we take in air, I would like you to remember what the air feels like going in and what it feels like going out, and if there's a difference between those two. Let's try it again. Ready? One, two, three, four, five, hold it, exhale, four, three, two, one, and breathe normally. I feel better already. How did that air feel going in? I bet it was cool and when we have cool air, we need to think about energizing. Let's look up here at this chart. We see that on inhale, we think cool because the air going in through our mouths is very cool and that's energizing. And when we exhale, I bet the air felt warm coming out. We need to think warm. We also need to say to ourselves at that time, that it's relaxing to feel warm and all the pain is being carried out of our
body. Now, occasionally, you might feel a little dizzy or lightheaded when you finally blow out the air. That will go away in a few seconds and you will feel nice and relaxed.

Another thing you need to think about is how this imagery is going to help you. You need to start by thinking about being in a nice, relaxing place. You can be alone or with somebody else, but you need to feel secure when you first start this imagery exercise. Now, let's do it again and remember, when we inhale, we are going to have cool energy flowing in and when we exhale, we are going to feel warmth and relaxation. Ready? One, two, three, four, five, hold it, exhale, four, three, two, one and blow out the rest of the air. Okay, I think you've got the idea, so let's move on. You need to relax when you do this. You need to practice this, at the beginning, three to five times a day. You need to use it when you need it, especially when you have pain or when you start to feel anxious. It only take fifteen seconds to do this. You can do it anywhere, in the car, during class, standing up, lying down, and when you are sitting. I hope that this imagery and breathing helps you. I hope that it gets rid of your menstrual pain and that you practice the imagery a lot. Thank you for your attention and participation in this study.
## APPENDIX H

### TABLE 1

GROUP MEANS AND STANDARD DEVIATIONS FOR SYMPTOMS

\[ N = 76 \]

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>NTC</th>
<th>ED</th>
<th>IR</th>
<th>CMB</th>
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</thead>
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<td>( 4.8 \pm 1.9 )</td>
<td>( 3.1 \pm 2.1 )</td>
<td>( 4.6 \pm 2.7 )</td>
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<tr>
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<td>( 3.5 \pm 2.7 )</td>
<td>( 2.9 \pm 2.0 )</td>
<td>( 3.4 \pm 2.9 )</td>
</tr>
<tr>
<td>+++</td>
<td>( 0.4 \pm 3.1 )</td>
<td>( 1.3 \pm 2.9 )</td>
<td>( 0.2 \pm 1.6 )</td>
<td>( 1.2 \pm 3.0 )</td>
</tr>
<tr>
<td>Nausea</td>
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<td>( 2.1 \pm 2.2 )</td>
<td>( 1.2 \pm 1.9 )</td>
<td>( 0.8 \pm 1.8 )</td>
</tr>
<tr>
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<td>( 0.7 \pm 1.5 )</td>
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</tr>
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<td>( 0.3 \pm 0.8 )</td>
<td>( 0.0 \pm 0.0 )</td>
</tr>
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Appendix H—continued

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<th>Symptom</th>
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<td>0.8 ± 1.8</td>
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<td>1.3 + 2.0</td>
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<td>1.1 + 1.8</td>
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<td>2.6 ± 2.1</td>
<td>2.9 ± 2.0</td>
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<td>Depression</td>
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<td>12.0 ± 9.3</td>
<td>20.4 ± 15.2</td>
<td>25.6 ± 18.3</td>
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</table>

+ The first line of each symptom describes the pre-test score means. ++ The second line describes the post-test score means. +++ The third line identifies the reduction in symptom severity unless the score is preceded by a (-) sign and this indicates an increase in the symptom severity during the treatment phase.
APPENDIX I

REVIEW OF THE MENSTRUAL CYCLE

All of the following time sequences are approximate. The onset of menses begins the menstrual phase and lasts from day 1 to day 5. In this phase, luteinizing hormone (LH) is at peak production and follicle stimulating hormone (FSH) levels begin to rise. FSH production initiates the follicular phase which lasts about two weeks. The follicle develops in the ovary and migrates to the ovarian cortex where it releases the egg at ovulation. The developing follicle has secreted increasing amounts of estrogen. Under this influence the endometrium (innermost lining of the uterus) proliferates and thickens. After ovulation the ruptured follicle becomes the corpus luteum and the secretory phase is begun. The corpus luteum secretes progesterone which in turn causes the proliferated endometrium to secrete hormones. If implantation of the egg does not occur, the endometrium begins to degenerate and is shed and washed out during menstruation. LH and FSH production are controlled by the anterior hypothalamus, which monitors blood hormone levels, and then stimulates the release of these substances from the anterior pituitary gland. The rising levels of estrogen, from the ovary and uterus, peak two to three days prior to onset of menses, stimulate the production of LH and FSH and the cycle is repeated.
APPENDIX J

PHYSIOLOGY OF PROSTAGLANDINS

The prostaglandins (PGs) that are involved with the reproductive cycle and are biologically active include: PGE$_2$, PGF$_{2\alpha}$, PGI (prostacyclin), PGG$_2$, PGH$_2$, and the thromboxanes. All PGs found in humans are of the subscript two series. The letter following the PG is the sequence of when they were originally isolated and named. PGs are oxygenated metabolites of carbon-twenty, polyunsaturated, essential fatty acids. The series nomenclature of PGs depends upon the number of double bonds in each side chain and the type of PG is identified by the oxygen containing substituents on the ring structure. Arachidonic acid and eicosatrienoic acid are the main precursors of PGs. Arachidonic acid is stored in cell membranes within the phospholipid bilayer. PG synthesis is initiated by phospholipase A$_2$ which causes the release of arachidonic acid from the membrane. This release occurs in response to either physiological, pharmacological, pathological, or mechanical stimuli (Whittle, 1979).

Oxygenation of arachidonic acid is the first step in PG production. The endoperoxides, PGG$_2$ and PGH$_2$ are formed first and then catalyzed by cyclo-oxygenase, a fatty acid enzyme. This enzyme is inhibited by non-steroidal, anti-inflammatory agents (NSAIDs) such as aspirin, indomethacin,
piroxicam, ibuprofen, and naproxen. The endoperoxides cause platelet aggregation (clot formation), vasoconstriction (NE action on adrenergic receptors), and contraction of smooth muscle (Granstrom, 1979). PGG₂ produces PGE₂ via two different pathways. One involves reduction by a peroxidase enzyme into PGH₂ and then isomerization by endoperoxidase isomerase to form PGE₂. The other mechanism is by the reverse sequence with 15-hydroxy-PGF₂ being the intermediate product. PGF₂α is produced by the reduction of PGG₂ and PGH₂ which is catalyzed by endoperoxide reductase (Granstrom, 1979).

Synthesis of PG occurs within one to two minutes. PGs are metabolized in the blood stream within 10 to 15 seconds. The half-life of the metabolites is approximately 8 minutes (Green, 1979).

The changing balance of the ovarian hormones at the end of the menstrual cycle appears to trigger a sudden discharge of hydrolytic enzymes, such as phospholipase A₂, into the cytoplasm. This initiates the synthesis of PGs.

Lysosomes are subcellular vesicles formed by the Golgi complex in the cytoplasm. These vesicles contain lytic enzymes and their release is controlled by the lysosomal membrane. PGs are effective labilizers (making them chemically unstable) of lysosomal membranes. The release of acid phosphatase, a lysosomal enzyme, acts on the endo-
metrium causing damage to the vessels, epithelial and stromal cells, and initiates menstrual bleeding. PGs enter the general circulation during menstruation but the mechanism is not known (Andersson, 1979). PG formation is perpetuated during menstruation by the action of the lysosomal enzymes releasing the PG precursors (Pulkkinen, 1979).

Another mechanism mediated by PG is myometrial activity. PG facilitates an influx of Ca\(^{++}\) from the myometrial membrane. Progesterone binds calcium ions in or near the membrane. Cyclic-adenomonophosphate (cAMP) alters the rate of Ca\(^{++}\) transport across membranes. In the myometrium, cAMP dependent, protein kinase, when bound to the smooth muscle membranes causes phosphorylation of membrane proteins which alters the cAMP. The contractile force of the uterus is produced by the formation of actomyosin (mediated by Ca\(^{++}\)). This reaction causes the hydrolysis of ATP which then liberates phosphate to form energy for the muscle contraction. An increase in myometrial contraction is observed because of excess Ca\(^{++}\) due to the influence of PG (Dawood, 1981).

Other effects of reproductive PGs are found in various sites in the body. PGE\(_2\) and PGI are found at inflammatory sites. They produce changes in vascular permeability, blood flow, and potentiate the effects of bradykinin and other pro-inflammatory substances. They potentiate the
pain producing action of kinins and sensitize the peripheral pain receptors. PGE$_2$ inhibits gastric acid secretion and vasodilates the gastric muscosa (Whittle, 1979).

PGE$_2$ stimulates the early proliferative and late secretory phases of the menstrual cycle. Both PGE and PGF$_{2a}$ increase uterine tone which elevates the frequency and amplitude of uterine contractions (Bygdeman, Bremme, Gillespie, & Lunstrom, 1979). PGF$_{2a}$ is found in increased amounts in the menstrual fluid and endometrium. PGI$_2$ has biphasic actions on the tone and motility of the uterus. Its action is relaxation of the myometrium while antagonizing the PGF$_{2a}$ contractile actions (Whittle, 1979). The following systemic effects are thought to be mediated by the above actions of the reproductive PGs: nausea, vomiting, diarrhea, dizziness, headaches, sweating, backaches, leg aches, general abdominal pain, and menstrual cramps.
APPENDIX K

PHARMACOLOGY OF DYSMENORRHEA

One of the two most effective drug groups for the treatment of dysmenorrhea is the non-steroidal, anti-inflammatory drugs (NSAIDs). The NSAIDs are prostaglandin synthesis inhibitors. The actions of this class of drugs include: (1) Stabilization of lysosomal membranes, thereby preventing release of PG precursors such as arachidonic acid; (2) suppression of uterine activity by decreasing the level of the regulation enzymes of free, intracellular calcium ions; (3) gastrointestinal side effects of varying degrees which may include indigestion, heartburn, nausea, abdominal pain, constipation, vomiting, anorexia, and diarrhea; (4) central nervous system side effects in man include headache, dizziness, vertigo, visual and auditory disturbances, depression, drowsiness, and sleeplessness; (5) a low efficacy except for the newer, synthesized drugs of this class; and (6) allergic reactions such as skin rash, edema, bronchospasm, hematological abnormalities (agranulocytosis and aplastic anemia), and idiosyncratic eye, kidney, and liver effects (Andersson, 1979).

The new NSAIDs have the following, overall actions: (1) Direct interference with the specific pathogenic processes caused by the various PGs; (2) drug use is now limited to the duration of the symptoms; (3) side effects
have been diminished; and (4) the analgesic effect is prompt (Henzel, Massey, Hanson, Buttram, Rosenwaks, & Pauls, 1980).

Three major classes of NSAIDs exist. They include the aryl carboxylic acids, aryl alkanolic acids, and enolic acids. Few are currently in use for the treatment of dysmenorrhea. Salicylic acid (aspirin) is a member of the aryl carboxylic acid class. It reduces PGE2 formation (inhibits gastric acid secretion), inhibits PG12 action (vasodilation), may stabilize the lysosomal membrane, and inhibits cyclo-oxygenase action in platelets (prevents clot formation). Aspirin does not have a significant effect on PGF2α, the major PG component of menstrual cramps, and therefore is of little benefit in the treatment of this symptom. Its use may also exacerbate menstrual flow (Dawood, 1981)

Anthranilic acids are the fenemates. Drugs in this class include flufenamic acid, mefenamic acid (Ponstel), meclofenamic acid, and tolfenamic acid. This group probably has PG antagonist action as well as an analgesic effect. There is a lack of long term safety data for the fenemates. Mefenamic acid (Ponstel) is currently used as a treatment for dysmenorrhea by some physicians (Chan, 1983).
The next class, the aryl alkanoic acids, contains four groups. The first is the aryl acetic acids which include alclofenac and diclofenac. This group is not used for the treatment of dysmenorrhea. The next group, the aryl propionic acids, includes ibuprofen (Motrin, Advil, Nuprin), ketoprofen, fenoprofen, and naproxen. Naproxen sodium (Anaprox) has previously been the drug of choice for dysmenorrhea, however ibuprofen is currently used. The mechanism of action of this drug group according to Segre (1980) is interference with microsomal PG synthetase and is unique because of its selective action on the uterine microsomes.

The aryl propionic acids are normally highly bound to plasma protein. They are more rapidly absorbed as salts because of the raising of the pKa towards neutral. The half-life varies from six to twelve hours and they are metabolized primarily by conjugation and somewhat by oxidation in the liver. Excretion is by the urine. Most drugs in this group have a high therapeutic index with shallow dose/response curves. Primary side effects are nausea and mild abdominal discomfort in a small percentage of users. Side effects associated with continual usage of these drugs are numerous and more severe. These drugs significantly reduce dysmenorrheic pain in 75% of the presenting cases, mildly ameliorate symptoms in nine percent of the population.
and has no effect in approximately 15% of the patients. Most drug studies report relief by placebo groups in 20-30% of the subjects (Granstrom, 1979).

The hetero aryl acetic acids include tolmetin (Tolectin) and fenclozic acid. No studies have been published using these drugs for dysmenorrhea.

The indole and indene acetic acids include indomethacin (Indocin) and sulindac (Clinoril). First used in treatment by Hill (as cited in Dawood, 1981), several studies have used indomethacin for the treatment of dysmenorrhea and demonstrate an efficacy rate of 75-90% amelioration of the symptoms. Indomethacin has also been demonstrated to have an additional effect of suppressing myometrial activity and relieving uterine ischemia. Indomethacin has considerable side effects and is not currently drug of choice for dysmenorrhea (Chan, 1983).

The last class of NSAIDs are the enolic acids and include the buterophenones. These drugs produce serious side effects and are not considered for the treatment of dysmenorrhea (Ylikorkala and Dawood, 1979).

Prophylactic treatments for dysmenorrhea may divided into three categories, the first of which is hormonal preparations. These include estrogen, gestagen, androgen, and danazol. These drugs are PG antagonists and their action is vasodilation and supression of ovulation. The
positive side effects of the birth control pill, including decrease in menstrual irregularities, less anemia, less premenstrual tension, fewer benign breast tumors, and pregnancy prevention, should be considered individually in comparison to the negative effects of these preparations which include headache, depression, vaginal discharge, urinary tract infections, skin problems, loss of libido, hypertension, and cardiovascular attacks (Dawood, 1981).

The antipyretic (anti-fever) analgesics act on the peripheral pain receptors and inhibit PG synthesis in most tissues. Examples of these drug include salicylates, pheacetin, and phenazone. However, they are not necessarily specific for $\text{PGF}_2\alpha$ which is the main PG contributor to menstrual pain.

Sedatives may be used in the treatment of dysmenorrhea. Drugs in this group include the neuroleptics such as chlorpromazine, and the anxiolytics or minor tranquilizers such as diazepam (Valium). These drugs act on the basal regions of the brain. The hypnotics and barbiturates may be useful in the amelioration of pain by raising the pain threshold. Possible psychological dependence to these drugs precludes their use as a primary therapeutic tool in dysmenorrhea (Heinrichs and Adamson, 1980).

Spasmolytic drugs are anticholinergic drugs that block muscarinic receptors and exert an inhibitory effect on the
parasympathetic pathways causing relaxation of smooth muscle (myometrium), pupil dilation, inhibition of saliva and sweat, and acceleration of the heart. Drugs in this class are atropine and scopolamine. Adrenergic receptor stimulants relax uterine muscle. Isoxuprine and ritodrine are examples of this drug group. The spasmolytic drugs produce systemic actions and may elicit serious side effects. These drugs are usually combined with analgesics or sedative hypnotics and are rarely prescribed for dysmenorrhea (Osler, 1979).

The use of NSAIDs in the treatment of dysmenorrhea was first discovered when patients being treated for rheumatoid arthritis noticed a decrease in their menstrual pain (Fox, 1953). The efficacy of the NSAIDs in the amelioration of the symptoms of this disorder is now well established. Chan, Dawood, and Fuchs (1979) document the correlation of PG synthetase inhibition with the PG levels in menstrual fluid. In 1978, Chan and Hill developed a methodology for extracting, qualifying and quantifying PGs in menstrual fluid. Other investigators, Akerlund (1979), Csapo, Pulkkinnen, and Henzl (1977), and Lundstrom (as cited in Dawood, 1981) have correlated the effects of NSAIDs with myometrial conductivity measurements.

Chan (1983) in his review of the efficacy of NSAIDs in the treatment of dysmenorrhea states that the different
classes of drugs inhibit different PGs. Ibuprofen supresses PGF$_{2\alpha}$ significantly. Naproxen sodium supresses PGE$_2$ and PGF$_{2\alpha}$ equally. He postulates that PGF$_{2\alpha}$ is responsible for dysmenorrhea and indicates that the search for a specific inhibitor of this PG is currently in progress.

Drug therapy is often indicated in acute ailments. Drugs also may have unpleasant and serious side effects. Drugs may place a financial burden on the patient. Behavioral medicine interventions, including education, when used as an adjunct to medication have often reduced the need for high doses of drugs, and in some cases have eliminated the use of drugs completely. Dysmenorrhea is a chronic syndrome, and alternative therapies in conjunction with effective drugs may reduce the possibility of harmful side-effects and relieve part of the financial burden that drugs incur.
References


