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THE PREMENSTRUAL TENSION SYNDROME

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Table of Contents

	<u>Page</u>
I. Introduction	2
II. Incidence	4
III. Signs and Symptoms	8
IV. Etiology and Physiologic Changes	10
V. Treatment	15
VI. Psychiatric Correlates	21
VII. Summary	26
VIII. Conclusions	28
IX. Bibliography	30

I. Introduction

Women have been plagued from time immemorial with aggravating physical and mental alterations in the few days prior to the onset of menstrual flow, but it was Frank⁹, in 1931, who first described the premenstrual tension syndrome as a definite entity. Its clinical features were defined as tension, irritability, depression, and anxiety manifested by a "fear of the unknown". The physical signs he described were swelling of the abdomen and limbs, itching, thirst, and various tendencies to migraine, asthma, and epilepsy.

Morton¹⁵ categorizes premenstrual tension as "a symptom complex occurring 10-14 days before menstruation with patients showing anxiety, headache, insomnia, emotional instability, fatigue, painful swelling of the breasts, abdominal bloating, low abdominal pain, and nausea with occasional vomiting" - the above occurring in varied combinations. He further emphasized that if and when these signs and symptoms rise above a position of relative insignificance to one of serious manifestations in a particular patient, premenstrual tension may definitely be implicated as a "disruptive force that may act against the harmony of the home and community; as a major economic problem resulting from decreased productiveness and increased absenteeism in business and industry; and - in its most severe forms - as an etiologic factor in some premeditated crimes of passion and violence".

It can be seen that this syndrome includes not only a symptom complex but infers far-reaching complications which may be super-imposed upon the basic problem. These complications are, for the most part, psychiatric in nature and will be treated in a separate section of this paper.

II. Incidence

It is difficult to state even an approximate incidence of the premenstrual tension syndrome, but comprehensive studies would appear to indicate that the syndrome occurs in over 60 percent of all menstruating women. In Morton's¹⁵ prison study of 249 inmates, he reported an incidence of 84 percent. Suarez-Marias²⁴ reported over 80 percent of 107 student nurses presented a periodic syndrome that could be defined as mild to moderate premenstrual tension. Pennington¹⁸ surveyed 1000 housewives, students, and business women and found that 95 percent suffered from symptoms defined as premenstrual tension. Rees²¹ in a study of 145 subjects further subdivided the syndrome and noted that 15.6 percent had severe premenstrual tension, 24.6 percent moderate and 56.5 percent no significant symptoms.

Green and Dalton,¹¹ Rees,²¹ and Morton¹⁵ also studied the symptom incidence of the syndrome. Tables A and B taken from their studies illustrate their findings.

TABLE A
Symptom Incidence in Premenstrual
Tension Syndrome

	<u>Greene & Dalton</u>	<u>Rees</u>
Headache	69.5%	63%
Nausea	29.7	37
Lethargy	13.1	63
Rheumatism	16.7	--
Vertigo	10.6	--
Depression	6.0	80
Irritability	6.0	100
Edema	6.0	73
Rhinorrhea	7.2	--
Mastalgia	2.4	63
Tension	--	100
Anxiety	--	73
Insomnia	--	40
Pruritus	--	40
Marked Thirst	--	20

TABLE B - (from Morton)

<u>Symptomatology</u>	<u>% of Cases</u>
Nervous and emotional instability	100
Painful swelling of breasts	76
Increased appetite	59
Lower abdominal pain	55
Abdominal bloating	52
Gain in weight and edema	45
Menstrual disturbances	41
Headaches	31
Nausea and vomiting	10
Increased sexual desire	10
Vaginal itching	7

Lamb¹³ and her associates, with a questionnaire study of 127 student nurses learned that 92 of them (73 percent) had symptoms of premenstrual tension. The most common symptom in her survey was abdominal fullness which 66 (52%) of them experienced; then followed in order, irritability (49%), backache (47%), tenderness of the breasts (43%) and mental depression (42%).

Ferguson⁷ undertook an extensive study of the prevalence of the syndrome utilizing an amazing list of 51 symptom categories. Two groups of healthy, employed

women, 75 in each group, were studied. Symptoms of the syndrome were found in 92% and 91% respectively. The most common symptoms, in order of prominence were: sensation of abdominal fullness, breast tenderness, headache, nervousness, backache, breast enlargement, mental depression and facial enlargement. Eighty-one private gynecologic and obstetric patients were also studied by Ferguson and 95% of them more or less regularly experienced premenstrual symptoms.

The age incidence as studied by Bickers revealed that the syndrome occurred most often in women between the ages of 25 and 35, and only occasionally in younger women. In a group of 72 patients studied by Bickers² the average age of those seeking relief was 28.6 years. The youngest patient was 14 and the oldest 43. There appeared to be a tendency for the malady to be of shorter duration and of lesser intensity in the younger individuals. In the younger group (under 25 years) symptoms rarely persisted longer than three days, whereas in the older group (over 25 years) the symptoms continued an average of seven days.

III. Signs and Symptoms

The major signs and symptoms have been fairly well defined as abdominal bloating, swelling of the breasts, emotional instability, low abdominal pain, and fatigue. Other symptoms which frequently accompany the above can be found listed in Tables A and B. Perhaps the most extensive list of occasional to frequent symptomatology found in the premenstrual tension syndrome has been compiled by Ferguson⁷ as is shown in Table C.

Table C

Signs and Symptoms Associated with Premenstrual Tension

Head:

Headache
Blurring of vision
Eyes, soreness

Mental:

Lack of orientation
Decrease in alertness
Confusion
Nervous
Wanting to cry
Instability
Depression
Moody
Antagonistic
Easily upset

General:

Fatigue
Malaise
Insomnia
Drowsiness
Weakness
Dizziness
Weight gain
Swelling of feet & ankles
Energetic
Increased perspiration
Tightness of fingers

Breasts:

Soreness
Tenderness
Painful
Enlargement

Gastro Intestinal:

Nausea
Vomiting
Increased appetite
Craving for sweets
Thirst
Constipation
Diarrhea
Loose Bowels
Abdominal distention

Skin:

Pruritus
Eruption of face

Urinary:

Frequency
Increased urination
with onset menstruation
Decreased urination

Table C (continued)

Pelvic:

Lower abdominal pain
Vaginal discharge

Skeletal:

Backache
Leg ache
Weakness of legs

In contrast to the normal female who gains approximately one-half to three quarters of a pound during the premenstrual week, associated with a minimum of premenstrual disturbance, Bickers² states that patients with the true premenstrual tension syndrome gain at least three pounds and those with severe symptoms gain much in excess of five pounds. He also has observed a significant rise in diastolic and systolic blood pressures (6-10 mmHg) during the premenstrual week in patients with rather severe symptoms, the pressure reverting to usual levels with the onset of menstruation.

Morton¹⁵ emphasizes signs of abnormal physiological alteration demonstrable by cytological, biopsy, temperature, and blood chemistry studies. These last "signs" of premenstrual tension will be further discussed when the problem of etiology of the syndrome is presented.

IV. Etiology and Physiopathology

The etiology of the premenstrual tension syndrome has long been a subject of debate. Many etiologies have been proposed throughout the years, some of which have stood the test of time, others of which have been discarded completely. Some have been dropped by the wayside for a time only to be re-proposed with increased fervor. Hippocrates, puzzled by the strange changes observed in the women of his time during the premenstrual week, ascribed their occurrence to "agitated blood seeking its escape at the uterus". Soranus of Ephesus²³ wrote: "When menstruation approaches it is accompanied by malaise, as we have indicated. Rest in most cases is salutary. For just as people full from a drunken bout if they move about vigorously may lay themselves open to sickness, and as those who have a congested head develop pain when they shout very loudly, in the same manner the uterus filled with menstrual material suffers compression accompanied by a feeling of weariness".

It is well known that the menstrual cycle is under almost complete hormonal regulations. Hence, an endocrinologic etiology has been explored most thoroughly and today is perhaps supported by most individuals. Frank⁹, in 1931, first suggested that a cyclic excess of estrogens might produce the somatic and psychic symptoms of premenstrual tension. Israel,¹² working along this same

line, suggested seven years later that rather than an increased estrogen level being the causative factor, it was instead an estrogen-progesterone imbalance due to decreased progesterone secretion at a time when progesterone production in normal individuals was at its peak. That second hypothesis, that of relative estrogen excess has been studied further by many researchers and has been most conclusively shown to have merit by Morton¹⁵ who has demonstrated this estrogen-progesterone imbalance in patients with premenstrual tension in the following ways:

- "1. Basal body temperature records either failed to show an abrupt rise at mid-cycle or presented irregularities such as a step-like rise associated with diminished luteal activity.
2. Endometrial biopsy revealed a hyperplastic or mixed proliferative and luteal picture in the late premenstrual phase. Stromal edema was marked in most cases of severe premenstrual tension. The marked progesterone effect seen in fully developed secretory endometrium was often absent.
3. Vaginal and cervical smears by Papanicolaou technique often showed an abnormal persistence of cornified cells as well as poor luteal activity in late premenstruum.
4. In most patients with normal or slightly lowered estrogen values, the premenstrual pregnanediol

excretion values were low".

These four groups of findings would tend to indicate a relative increase in estrogen and decrease (absolute) in progesterone. Morton emphasizes however that the resulting signs and symptoms of the syndrome are most likely due to the estrogen increase. He describes the occurrence of occasional edema and other symptoms at midcycle when estrogen normally rises transiently. Similar symptoms have also been noted in postmenopausal women and castrates following administration of large doses of estrogen. Three major functions of estrogen ascribed by Morton to result in the appearance of the prime symptoms of premenstrual tension are:

1. Retention of extra cellular tissue fluid resulting in edema.
2. Stimulation of increased epithelial proliferation responsible for the hyperplastic changes that occur in the breast, uterus, and vaginal epithelium.
3. Alteration of carbohydrate metabolism by increasing sugar tolerance resulting in a "subclinical hypoglycemia" which could account for emotional and behavioral symptoms.

Ray²⁰ agrees that the syndrome may represent a hormonal imbalance and attributes this imbalance as resulting through "cyclic ovarian activity mediated through

the hypothalamic-autonomic system" with its consequential effects on intermediary metabolism of electrolytes and water. Ray, however, postulates a second etiology. He suggests that the syndrome could easily be due to allergic endocrine hypersensitivity to any number of steroid hormones or to the products of their metabolism. Implicated hormones included estrogens, estradiol, progesterone, pregnanediol, testosterone, androsterone, corticosterone, and chorionic gonadotropin. He further states that if this hypersensitivity reaction theory is true, the clinical signs and symptoms would quite likely be the same even though the allergen might vary in different cases.

Zondek²⁶ limits the above hypersensitivity theory, stating that the syndrome is most likely an allergic response to normal levels of estrogen. Bickers² disagrees with Zondek, stating that the allergic response, if present, is most likely due to antidiuretic hormone. He has reported abnormally high urine and blood titers of ADH in patients with the premenstrual tension syndrome and also has been able to produce typical symptoms by the administration of several doses of aqueous pitressin daily to sensitive individuals. He has shown also that this reaction to pitressin occurs only in sensitive individuals during the postovulatory phase of the menstrual cycle.

The possibility of allergic reactions to ovarian hormones or to pitressin does find some support in the occasional occurrence of premenstrual dermatoses, urticarial reactions, angioneurotic edema, and herpetic lesions; most of which will respond to antihistamines or cortisone therapy.

Biskind⁴ states that since estrogens are, for the most part, conjugated in the liver perhaps liver impairment as a result of vitamin B deficiency might play the prime role and with the secondary estrogen rise, also the sodium and water would be retained. Bickers² questions this since abnormal interstitial water storage does not occur in cirrhotics nor does vitamin B therapy alone induce diuresis indicative of estrogen level depletion in those with premenstrual water storage.

Taylor²⁵ proposes that since the syndrome is often associated with vascular congestion of the pelvic organs and tissues and that this congestion was related to autonomic nervous system imbalance, the varied symptoms could be due entirely to this upset nervous system. The primary cause of the autonomic nervous system imbalance is not explained however, hence the primary etiology remains unsolved.

Barnes¹ reports that aldosterone levels may be increased in some women during the premenstrual phase. Since the power of aldosterone to limit sodium and water excretion is well-known, this excess of aldosterone could explain the water retention and resultant distress and weight gain in premenstrual tension.

V. Treatment

As can be well imagined, the treatment of premenstrual tension has undergone many changes as the theories of etiology also have been reviewed and revised. Remedies proposed run full circle: from herb extracts to psychotherapy and back again.

Ray²⁰ describes considerable success utilizing Femmes, a specific arrangement of certain active herbs freed from inactive material. The active principles are: hydrastine, galangin, columbin, angelic acid, ascoridol, santalol, cimicifugin, zingerone, and sennocide, with excipients and flavors. This weird sounding concoction reportedly acts on the hypothalamic-autonomic axis and causes inhibition of pregnanediol and of antidiuretic hormone. It also supposedly enhances biliary excretion of increased conjugated estrogens resulting in diminished cerebral and corporal edema. Finally as an overall therapeutic support, it acts as sedative, analgesic, relaxant, antispasmodic, antiallergic and tonic. This medicament was used on a controlled study group of 110 patients. The subjective reports of the patients when questioned following a specified check-list of signs and symptoms reveals astounding results as follows: success-107, failure-3. In Ray's opinion the drug gave consistently satisfactory results in almost all patients and caused no deleterious effects over a considerable period of time.

There was no evidence of habituation or dependence on the medication in any of the patients studied. The dosage was given as: two tablets at onset of discomfort and one to two tablets every two to four hours as required; as a prophylactic: one tablet four times daily for one to three days prior to the expected period.

Simmons²² discusses the management of 288 private patients utilizing four plans of treatment. These four plans are illustrated in Table D taken from his report.

Table D
Summary of Treatment for
Premenstrual Tension

<u>No. Cases Treated</u>	# Cases	Satisfactory Result % of cases treated
Plan #1a 23	0	0
Plan #2b 36	0	0
Plan #3c 34	19	55.9
Plan #4d 288	238	82.6

- a. Premenstrual salt restriction and diuresis.
- b. Premenstrual progesterone (50-100 mg by injection or 150 mg by buccal route).
- c. Premenstrual testosterone (50-100 mg). In about 1/3 of cases so treated, the menses became short, scanty, and delayed after several months of therapy and with cessation of therapy symptoms returned promptly.
- d. Hyposensitizing doses of pregnanediol (0.05 mg orally daily for three months).

It can be seen that therapy instituted on the basis of possible allergy as the important factor instigating premenstrual tension yielded by far the best clinical results in this series. Simmons proposed that on the basis of the above and similar findings that the "phenomenon of endogenous hormone allergy" was indeed a major factor in premenstrual tension.

As noted in Ray's success, the combining of medications in attempts to alleviate more than one of the proposed etiologies of the syndrome has been a common practice. Goldfarb¹⁰ combined, into an enteric coated tablet, ammonium chloride for diuretic effects, homatropine methyl bromide to "level the upset balance of the autonomic nervous system". D-amphetamine sulfate as a mild stimulant, and vitamins B₁, B₂, B₆, niacinamide, and calcium pantothenate, "helpful in increasing the breakdown of estrogens in the liver". Fifty patients were treated beginning ten days preceding the onset of menstrual flow and continued for twenty-four hours following its cessation. Low-salt, high protein diet supported the regimen. No major side effects were noted and "the major symptoms of premenstrual tension -- depression, instability, edema, abdominal distention and headache -- were relieved in 80 percent of the 50 women treated".

Morton¹⁷ used a similar combination substituting caffeine for D-amphetamine and known by the trade-name Pre-mens. He found that in 249 volunteers, relief was obtained by 61 percent of the patients. When high protein

and low salt diet was added, the percentage of success improved to 79 percent. Eichner and Waltner⁶ administered an identical preparation to 87 patients. They reported complete relief of symptoms in 50 percent of their patients with partial relief in the others. They reported that for effective treatment, it must be begun seven to ten days prior to the onset of menstrual flow.

McGavack¹⁴ and his associates alternately treated nineteen patients with placebo, then an ammonium-chloride-vitamin-antivagal compound identical to that used by Morton and finally an antihistamine - theophylline preparation. (50 mg of 2-amino-2-methyl-1-propanol-8 bromatheophyllinate and 30 mg of pyrilomine maleate: trade-name - neo Bromth). Definite overall degrees of relief were afforded 13 or 68.4 percent of the subjects taking neo Bromth and 6 or 31.6 percent of the group receiving Pre-mens. These authors stated that although pelvic distress, breast enlargement, and headaches were about equally controlled by the two preparations, Pre-mens seemed to aggravate and sometimes cause digestive disturbances and resulted in less favorable response in water and salt retention, nervous and mental symptoms, and acne.

In general, most studies utilizing hormone therapy in treatment of premenstrual tension have revealed only occasional success. The use of chorionic gonadotropin has been reported by Morton¹⁷ to provide occasional dramatic relief, its value being in the stimulation of

the corpus luteum to increase progesterone secretion in the face of estrogen-progesterone imbalance. The dosage used was 500-100 units intramuscularly once or twice weekly during the middle two weeks of the menstrual cycle.

Bickers² summarizes the use of hormones in the syndrome in this manner: "Unfortunately, no rational or consistently effective treatment employing hormones has been developed. In my opinion the use of estrogen, progesterone, or testosterone is definitely contraindicated because of their water-fixing capacity".

In recent years the advent of increasingly effective diuretic agents has led to an upsurge of therapeutic trials along these lines in the syndrome of premenstrual tension. One of the most effective diuretics to date, chlorothiazide, has been studied extensively in this syndrome by Bickers². He treated 49 patients with this drug in a single daily dose of 250 mg after breakfast beginning with the onset of symptoms and continuing through the first day of menstruation. Thirty-eight were reported as having responded favorably to treatment with chlorothiazide. The average premenstrual weight gain was 4.5 pounds during the untreated cycle and 1 pound in the treated cycle. The clinical response was deemed unsatisfactory in the remaining 11 patients; the average weight gain in the untreated cycle being 3.25 pounds and in the treated cycle 2.75 pounds with little or no subjective

relief of symptoms. Bickers deduced that these failures were probably due to the fact that, for some reason, these patients were refractory to chlorothiazide diuresis; or therapy in addition to diuresis was required. Consequently 5 patients from the failure group were carried through a new regimen employing chlorothiazide plus an antihistamine (pyrilamine maleate). Four of the five patients subsequently reported "complete relief of all symptoms related to the premenstrual tension complex", and a coincident drop in weight gain from an average of 2.75 with chlorothiazide alone to 1.5 pounds with addition of the antihistamine.

Many types of supportive therapy have been utilized for this syndrome. By far the most commonly used have been the tranquilizers and most recently mood elevators with occasional success in alleviating the specific symptoms treated. Elimination of the primary cause cannot be accomplished by their use however.

VI. Psychiatric Correlates

Heretofore the discussion of the premenstrual tension syndrome has been limited in this paper to pathophysiological etiologies and consequences. Perr¹⁹ states that the relationship of mental symptoms to the syndrome is exceedingly complex and refers to Rees²¹ statement that while bodily changes account for most of the symptoms of premenstrual tension the patient's reaction is governed by the following factors:

"1. Constitutional

a. Stability of the autonomic nervous system and homeostatic mechanisms.

b. Personality type.

2. Degree of general stability

a. General life adjustment

b. Personality reaction

c. Incidence of neurotic and personality disorders."

Rees denies that neurosis or emotional instability alone can cause the syndrome since it is readily demonstrated that many neurotics do not have the symptoms. He does state however that the severity of the neurosis may result in a concomitant increase in intensity of the syndrome if it is present. This is exemplified in Table E taken from Perr's research.

Table E

Incidence of Premenstrual Tension in
Normals and Neurotics

Degree of Tension	61 Normals	84 Neurotics
None	78.7%	38%
Moderate	16.4	30
Severe	5	32

Incidence of Premenstrual Tension
Related to Degree of Neurotic
Constitution

Degree of Neurotic Constitution	Degree of Premenstrual Tension		
	Nil or Mild	Moderate	Severe
Nil	76%	17%	7%
Mild	56	36	
Severe	15	48	37

If these figures are valid one can infer from them that a definite correlation exists between neurotic tendency and severity of symptoms but any attempt to correlate severity of neurosis with severity of symptoms breaks down as can be seen in the second part of Table E. However, if the first correlation is accepted as valid and if one defines neurosis or psychoneurosis as a mental disorder whose chief characteristic is "anxiety" which may be directly felt and expressed or which may be unconsciously and automatically controlled by the utilization of various psychological defense mechanisms, it then follows that any form of anxiety per se might cause an exacerbation of premenstrual symptoms in any individual who might otherwise be totally free of the syndrome or experience only minor symptoms. Cooke⁵ points out that

many women who have a fear of being pregnant often have severe premenstrual tension. Along this same line, the classic statements made by many instructors in basic psychiatry might be inserted: "More women either have prolonged menstrual cycles or miss periods entirely because of fear of pregnancy than because of pregnancy itself.

Perr¹⁹ summarizes: "Emotional reaction to stress is dependent upon the personality and not the stress." Since the premenstrual syndrome is a mild physiologic stress, it is reasonable to anticipate that any emotional symptoms, especially those of the "neurotic" variety might be aggravated during this period. These emotional symptoms, manifested by anxiety can be related to many things as might be well imagined; fear of pregnancy, domestic difficulties, dissatisfaction with employment, family illness, etc.

Many attempts have been made to correlate psychosis and psychotic episodes with premenstrual tension syndrome. The concept of a premenstrual psychosis has largely been abandoned although it has been a common observation that many psychotics, especially schizophrenics, show exacerbations of their psychosis during the premenstrual syndrome and easing off with the onset of flow. Perr consulted many psychiatrists with extensive hospital experience none of whom could recall a case where an individual had a premenstrual psychosis with no basic psychotic personality between periods.

Fortin and his associates⁸ studied the problem quite thoroughly in a group of 45 patients, 25 of whom demonstrated both subjective and objective signs of premenstrual tension. A control group of 20 patients were completely asymptomatic. Physiological studies to ascertain the amount of fluid retention in both groups revealed statistically non-significant differences between the two groups. Psychiatric interviews revealed that in the symptomatic group there were two types of tension syndromes.

(a) Onset with menarche, usually combined with dysmenorrhoea. This type was said to have been characterized by "disturbing fantasies regarding menstruation fostered by the mother; by marital tension between the parents; by a hostile dependent relationship to the mother with intense feelings of guilt; by repudiation of the feminine role with marked feelings of envy of the male."

(b) Onset several years after menarche. This type characterized by "the onset of the disorder often precipitated by disturbances in the patient's life history, especially by guilt over sexual temptation."

The control group was stated to have "demonstrated a better acceptance of the feminine role and of the inevitable restrictions imposed on a girl; a reaction of pride to the menarche with emphasis on the positive aspects

of femininity; a dependent relationship to the mother with fewer hostile features; and a better sexual adjustment".

Fortin therefore suggests that personality type plays a most important role and that the premorbid development of this pattern is perhaps more a factor in the severity of premenstrual tension than are the anxiety states which might come and go. In any event, it cannot be doubted that emotional instability plays no minor role in the etiology and in determining the severity of premenstrual tension.

Many treatments for this phase of the syndrome have been suggested. They run the gamut from mood elevators to tranquilizers, and from psychoanalysis to hypnotherapy. None of the above have been reported as producing continued or dependable relief although in any given patient success has been occasionally obtained. Undoubtedly the best therapy for this phase would be avoidance of stress or anxiety situations during the premenstrual period. Since this is impossible, the words of Greens and Dalton¹¹ will continue to debunk many attempts at rational physiologic therapy: "At times of stress, symptoms become unbearable and of increased severity, whilst when life flows along like a song, the symptoms decrease or may pass by unnoticed."

VII. Summary

A discussion of the premenstrual tension syndrome has been presented covering the incidence, signs and symptoms, possible etiologies, physiology, suggested therapy, and some psychiatric correlates.

It has been shown that this syndrome occurs in over 60 percent of all menstruating women. Some studies indicate an incidence as high as 95 percent. Heading the list of symptoms are nervous and emotional instability, mastalgia, abdominal bloating, and weight gain.

The etiology remains a subject of debate and has puzzled physicians since the time of Hippocrates. Estrogen-progesterone imbalance during the premenstruum, hypersensitivity to hormones, vitamin B deficiency, vascular congestion and high aldosterone levels have all been implicated as possible causes.

Treatment has been largely patterned to eliminate as many of the varied etiologies as possible and to eliminate specific symptoms. Hormone desensitization, herb extracts (Femmes), ammonium chloride-atropine-stimulant-vitamin preparations (Pre-mens), antihistamine-theophylline medications (neo-Bromth), specific hormones, and potent diuretics (chlorothiazide), have all been utilized and recommended by various workers.

Psychiatric correlation in this syndrome has been suggested; the incidence of premenstrual tension

tending to be higher in persons with neurotic tendencies than in so-called normal individuals. No correlation with psychosis was established however. It has also been suggested that pre-morbid experiences and personality may play a determining role in the incidence of the syndrome.

VIII. Conclusions:

Premenstrual tension is a symptom complex. It is characterized by mental symptoms of nervous tension, irritability, depression and anxiety, and physical signs of abdominal bloating, swelling and painful breasts, low abdominal pain and fatigue. Other physical and mental signs and symptoms may be manifest and vary a good deal from patient to patient and from period to period.

The syndrome occurs in over 60 percent of menstruating females, plaguing women from ages 25 to 35 most frequently but involving women from menarche to menopause. Older women appear to present more severe and prolonged symptoms.

The etiology of the syndrome remains unsolved but the bulk of evidence tends to point toward a hormonal imbalance, namely: an absolute progesterone depletion with resultant secondary estrogen increase. This increase in estrogen concentration is manifest by edema and hyperplastic epithelial changes. Hypersensitivity to certain ovarian and pituitary hormones and/or their break down products cannot be absolutely discounted however in view of the many triumphs utilizing therapy aimed at desensitization and supportive treatment of allergic-like symptoms.

The treatment of this syndrome is best accomplished at present by supportive therapy aimed at specific

signs and symptoms, e.g.: edema-diuretics, anxiety-tranquilizers, depression-stimulants. Up to recent years these medicines were combined in such a way as to combat as many of the proposed etiologies as possible with amazing success reported in some cases. If these combinations of ingredients were so effective, why the recent upsurge to the use of potent diuretics alone? Apparently the high percentages of success reported with use of these combination drugs leaves still more to be desired.

The incidence of premenstrual tension is somewhat higher in women who demonstrate some degree of neurotic tendency. The severity of the neurosis however does not determine the severity of the premenstrual tension. Symptoms of the syndrome may be magnified many times when the premenstruum is fraught with stress situations resulting in anxiety not present during other premenstrual periods. Premorbid fears, anxieties, and advice may also play no minor role in determining the severity of this popular yet distressing malady.

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