Psychosomatic Aspects of Menstrual Dysfunction

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Each time I have a period . . . I have the feeling that in spite of all the pain, unpleasantness, and nastiness, I have a sweet secret . . . that is why, although it is nothing but a nuisance to me in a way, I always long for the time that I shall feel that secret within me again.

Anne Frank
The Diary of A Young Woman

Menstruation, in contrast with many important bodily functions, is invested with a variety of cultural, ethnic, and other highly personal beliefs. Historically, such myths surrounding the menstrual cycle have been substituted for an accurate and unbiased understanding of the phenomenon. Depending upon such beliefs, what constitutes normal versus abnormal menstruation may be perceived quite differently by individual patients and physicians.

As Anne Frank has recorded so poignantly in her diary, a woman’s menses represent a highly personal experience colored by both negative and positive emotions. Important insights into a young woman’s subjective experience with menstruation have been provided by Abplanalp,2 Chernovetz,3 and Whisnant,4 who have drawn attention to the importance of mother-daughter communications, appropriate educational materials, and other sociocultural experiences.2-4 As these and other authors have noted, a young adolescent’s first and subsequent menstrual experience appears to be positively influenced by the availability of relevant information, supportive adult relationships (particularly with one’s mother), as well as more general social support for developing a sense of self-esteem and femininity.

Although scientific data are lacking, anecdotal reports suggest that some women can deliberately extend menstrual cycle length, that young women participating regularly in team sports may synchronize their menstrual cycles to within a few days of each other and that female family members vacationing together may establish a common time of menstruation.

With the growing interest of women in both competitive and noncompetitive sports, of more practical concern to the practicing gynecologist is exercise-associ-
ated menstrual alterations. Frisch and co-workers (1977) proposed that body weight, particularly lean body mass, is of critical importance both in the initiation and maintenance of menstrual function. The Frisch "critical weight hypothesis" has been suggested as an explanation for the amenorrhea commonly seen with female gymnasts, ballet dancers, marathon runners and young women with anorexia nervosa. However, the studies by Shangold and Schwartz on female distance and marathon runners indicate oligomenorrhea and amenorrhea often exists even before competitive running begins. Katz and Weiner, in their review on anorexia nervosa, report that 20–65% of women with this condition develop their amenorrhea before weight loss occurs and, further, that return to normal weight was not necessarily associated with resumption of menstruation. In her studies on adolescent ballet dancers, Warren found prepubertal changes, particularly menarche and secondary sex changes, markedly delayed during active training. Initiation of menstruation and progression of sexual development began in two thirds (10/15) of her sample, only with a decrease in exercise or forced rest from an injury of at least 2 months duration. Interestingly, during the interval that menstruation began, changes in body weight and lean body mass were minimal or absent. Although an attractive explanation, the Frisch hypothesis appears insufficient to account for all the observations made from the limited studies available on exercise-associated menstrual disturbances.

Weiner, an advocate of psychosomatic mechanisms of disease, suggests that "health is associated with certain patterns of sleep, hormone, and neurotransmitter release." He goes on to state that, "It has only recently been recognized that some diseases may be associated with shifts in phase, or . . . age-inappropriate patterns of these rhythmic physiologic functions. This way of viewing disease goes beyond the usual manner of looking for an anatomical lesion or some quantitative alteration, such as the absence or structural alteration of an enzyme as a full explanation of pathological anatomy or pathophysiology of disease. It is within this broader psychobiologic framework that this chapter will focus.

Psychosomatic Concepts Relevant to the Management of Menstrual Dysfunction

An appreciation of psychomatic concepts may be particularly useful to the gynecologist who sees a significant number of patients who have complaints whose underlying organic factors are not readily apparent or are insufficient to explain the patient's level of distress. Commonly, such complaints are diagnosed as functional or "psychosomatic" without further exploration. Rarely are such patients satisfied with this approach, and therapeutic efforts frequently fail.

For example, in a patient for which an initial investigation has failed to reveal an underlying cause of hypermenorrhea, the clinician may wish to alter his line of inquiry and ask the following questions: 1) What value, either negative or positive, do the symptoms have for this particular patient? 2) Are there socioenvironmental stress factors, either general or specific, operative in the patient's daily life that may be contributing to the presenting complaints? 3) Is there reason to consider a more extensive diagnostic evaluation in a patient whose emotional makeup and current life-style do not suggest significant underlying emotional turmoil? 4) What are the patient's concerns about the particular presenting complaint? What is her explanation about what may be wrong? For example, is she afraid that abnormal bleeding may suggest underlying cancer, the menopause, or recent injury from intercourse? Is there guilt about clitoral manipulation or some other sexual behavior? A variety of psychosocial dynamics may be present, the more common of which
relate to concerns about sexual and reproductive disturbances, interpersonal relationships, and/or career or occupational conflicts. With the above information in hand, the physician can better determine the need for further diagnostic or surgical intervention or a more concerted exploration of the patient’s daily life experiences and socioenvironmental setting.

For many physicians, working in the psychosomatic mode can be distressing, particularly for those who wish to establish a firm diagnosis after the initial evaluation and to decide immediately on specific therapy. How does the patient make the physician feel? Certainly personal reactions of frustration, anger, or a sense of not being able to satisfy the patient should alert the physician to potential problems in the patient-physician relationship. Either such patients trigger off an idiosyncratic reaction in the physician or may reflect the presence of underlying emotional or social/situational factors. As we have noted, working with such patients involves a cognitive switch from being an objective diagnostician to standing back, getting a feeling for the patient, and what meaning such symptoms may have in her particular circumstances. In brief, psychosomatic medicine does not conceptualize clinical problems in strictly “cause and effect” or linear relationships but rather stresses the importance of placing patients and their health problems within a larger social and behavioral context. Further, it is recognized that psychosomatic facets exit in most, if not all, diseases.11

A Theoretic Framework for Psychosomatic Medicine

Near the turn of the century, Rudolph Virchow observed that knowledge of chemistry would become indispensable to the practice of medicine, however, at the time, chemistry had nothing of practical value to offer.12 The evolution of psychosomatic concepts in medicine has followed a similar history. Selye, in the late 1930s, experimentally demonstrated that the response of the body to stress depends on the brain.13 More recently, Mason (1968) has established a relationship between environmental stress and subtle, rapid, and specific fluctuations in various endocrine functions. Other more contemporary investigators including Stein,11 Wolf,14 and Weiner15 have further documented the importance of brain mechanisms in the control of the autonomic nervous system, neuroendocrine functions, and both humoral and cell-mediated immunologic responses. Current research in psychosomatic medicine has moved away from a preoccupation with specificity hypotheses, (i.e., that a particular personality type may be associated with a specific physical disease), psychoanalytic explanations of illness, and the use of psychotherapy on patients with organic disease. Increasingly, there has been a shift in focus to investigate general environmental stresses, all physical diseases to include social and emotional dimensions, and increasing interest in basic biologic mechanisms. It is within this more contemporary framework of psychosomatic concepts that the following discussion should be interpreted.

Brain-Body Interrelationships

Theoretically, brain mechanisms can alter menstrual functions through a variety of pathways including neuroendocrine, autonomic, immunologic, or other biochemical mediators. Preliminary data exists to suggest that several of the mechanisms above may be of etiologic importance in psychosomatic aspects of menstrual dysfunction.

Since Schildkraut1 (1965) first hypothesized that brain neurotransmitters (biogenic amines) are of etiologic importance in certain psychiatric disorders, particularly depression, psychiatrists and neurobiologists have become increasingly interested in certain brain functions commonly referred to as the limbic system. While not identified as a specific division of the brain, the limbic system appears to represent a locus of neurochemical and neurophysiologic activity that
is both functionally and anatomically related. Basic human survival behaviors, including the response to a perceived threat, eating and sleeping, and sexual and reproductive functions appear to be intimately related to limbic system activity. In brief, the limbic system is perceived by many psychiatrists as the "emotional brain," an area of increasing interest and presumed importance in linking higher cortical functions with automatic, neuroendocrine, and other involuntary responses. While psychosomatic mechanisms have been most thoroughly investigated in such disease states as hypertension, asthma, peptic disease, and anorexia nervosa, disorders of the female reproductive system have increasingly come under scrutiny.

It has been well documented that emotional stress can disrupt ovarian function and induce infertility. Stress has been cited as the cause of disturbances in the timing of ovulation during the month that artificial insemination was anticipated. As reviewed be Drew, there appears to be a high incidence of secondary amenorrhea under stress environmental conditions, such as among factory workers, Air Force and Navy recruits, and hospitalized patients with psychiatric illness. Although the duration of posttraumatic amenorrhea following high-level spinal cord injury may extend for up to several years, a large percentage of quadriplegic women experience no interruption of menses, suggesting that social-environmental stress may significantly influence the timing of menstrual cycle return.

Psychogenic amenorrhea has been associated with a variety of abnormal gonadotropin patterns and estrogen-deficient symptoms suggestive of polycystic ovarian disease or premature menopause. Socio-psychologic distress may play a role in the elevation of prolactin secretion and acyclic gonadotropin patterns seen in women with postpil galactorrhea-amenorrhea; when psychologic dysfunction was corrected through psychiatric counseling, normal hormonal function resumed. In pseudo-cysis, a condition characterized by prolactin hypersecretion, a similar mechanism may be operative. A new hypothesis has been proposed and is described in detail by Reid and Yen elsewhere in this volume.

**Brain Catecholamines**

Psychogenic-induced menstrual cycle disorders may result from disruption of the neuroendocrine integrating system that controls reproduction, referred to as the hypothalamo-pituitary-ovarian axis. At the level of the hypothalamus, it is believed that gonadotropin-releasing hormone (GnRH) is regulated by a dual catecholaminergic system in which dopamine (DA) exerts inhibitory effects and norepinephrine (NE) exerts facilitatory effects on GnRH release. In addition, these two neurotransmitters appear to undergo cyclic changes in central activity coincident with the midcycle luteinizing hormone (LH) surge.

There is a growing amount of evidence that suggests that situational or emotional distress may promote or enhance an imbalance in brain biogenic amine formation and metabolism. Experiments in laboratory animals, using a variety of stress-inducing techniques, have shown that both acute and chronic stress can enhance brain catecholamine formation through stimulation of the rate-limiting first step in production involving tyrosine hydroxylase (TOH). One of the ways this effect may be mediated is through the action of stress-induced secretion of adrenal glucocorticoids, which have been shown to influence the steady-state levels of brain TOH. Stress in animals has also been shown to produce a decrease in monoamine oxidase activity in the hypothalamus, thus preventing the action of brain catecholamines.

Dopaminergic hyperfunction in the hypothalamus could result in inhibition of GnRH release and suppression of ovulation. In humans, administration of DA or DA agonists such as L-dopa and bromocryptine produce a dramatic suppression of the midcycle LH surge. Women diagnosed
with low-weight amenorrhea and who received the dopamine receptor antagonist metaclopramide showed a significantly elevated LH response to GnRH stimulation, suggesting increased central dopaminergic activity in this condition.\(^{(26)}\) (Larsen, 1981).

There is a growing amount of evidence suggesting that another biogenic amine found in high concentrations in the hypothalamus may play a role in stress-mediated events. Secretion of the endogenous amine \(\beta\)-phenethylamine (PEA), which resembles amphetamine both structurally and pharmacologically, has been shown to be markedly elevated in schizophrenic patients, as well as in normal men and women in response to parachute jumping.\(^{(27,28)}\) A relationship between stress-induced PEA secretion and menstrual dysfunction in women remains to be investigated.

**Endogenous Opiates**

A number of endogenous compounds in the brain have been purported to act as modulators of central neurotransmitter function to amplify, dampen, or set the tone of local synaptic activity by altering the effectiveness of a neurotransmitter.\(^{(24)}\) The endogenous opiate peptides may play a role in stress-induced menstrual dysfunction. Studies in normal women, as well as those with hypothalamic amenorrhea, suggest that \(\beta\)-endorphin suppresses LH while augmenting prolactin secretion.\(^{(29)}\) Blankstein, et al.\(^{(30)}\) postulate that excessive endorphin secretion may be involved in the pathophysiology of secondary amenorrhea and hyperprolactinemia, because naloxone administration to women with these conditions elicited a significant increase in serum LH. No response was elicited in two women with GnRH deficiency, suggesting that naloxone acts normally by uncovering the inhibitory action of an endogenous opioids pathway upon hypothalamic GnRH secretion. Opioids may increase pituitary prolactin through their inhibitory interaction with dopamine nerve terminals in the median eminence.\(^{(31,32)}\) A single large precursor molecule “pro-opiocortin” appears to be the prohormone for ACTH, \(\alpha\)-melanocyte-stimulating hormone, \(\beta\)-LPH and \(\beta\)-endorphin.\(^{(33)}\) A current hypothesis that is supported by animal and human studies suggests that \(\beta\)-endorphin is released simultaneously with ACTH from the pituitary in response to stress. This ACTH-linked release is believed to provide stress-induced analgesia to dampen the perception of pain during threatening circumstances.\(^{(34,35)}\) In the rat, the maternal pain threshold has been shown to rise progressively during pregnancy and can be suppressed by the administration of a narcotic antagonist. No changes were observed in nonpregnant animals, suggesting a pregnancy-specific response that is mediated by an endorphin system.\(^{(36)}\) In nonpregnant women, plasma concentrations of \(\beta\)-endorphin are not significantly different from those in men; but during pregnancy they progressively rise, reaching maximal values at delivery. The placenta has been implicated as the secretory source of this pregnancy-specific phenomenon.\(^{(37)}\)

Beta-endorphin may also act as a neuromodulator of the well known stress-associated increase in vasopressin (VP) secretion. Reid and Yen\(^{(38)}\) cite animal and human evidence for a stimulatory effect of endogenous opiates on vasopressin secretion, which they implicate as a major causative agent in the physical and emotional symptoms of the premenstrual distress syndrome. Women with dysmenorrhea demonstrate a significantly higher plasma concentration of vasopressin on day 1 of the cycle than do asymptomatic controls.\(^{(39)}\)

**Prolactin**

A significant relationship exists between emotional stress and hyperprolactinemia.\(^{(32)}\) Boyd and Reichlin (1978) argue that this stress-induced reflex is mediated through hypothalamic serotonergic neurons which increase prolactin releasing factor (PRF) secretion. Increased prolactin levels have been shown to suppress gonadotropin cy-
tility and induce anovulation and amenorrhea probably as a result of local as well as central inhibition of the reproductive cycle.\textsuperscript{40} In contrast, the hyperprolactinemia and elevated gonadotropin secretion associated with pseudocyesis is believed to result from a deficiency of hypothalamic dopamine activity, because the administration of dopa agonists can suppress hypersecretion.\textsuperscript{23} Hyperprolactinemia has also been associated with luteal phase shortening as a result of a direct effect on ovarian progesterone production.\textsuperscript{41} This mechanism could account for the commonly observed "honeymoon" phenomenon of stress-induced menses onset.

Other endogenous compounds that have been implicated as possible neuromodulators of stress-mediated menstrual dysfunction include melatonin from the pineal gland, melanocyte-stimulating hormone from the intermediate lobe of the pituitary, testosterone\textsuperscript{8} and hypothalamic catecholamines.\textsuperscript{38,40}

In summary, it appears that there exists several mechanisms whereby psychosomatic factors can lead to alterations in central neurotransmitters and/or neuroregulators of the reproductive axis, resulting in menstrual cycle dysfunction.

**Conclusions**

Patient and physician concerns with disturbances of menstruation continue to make up a significant proportion of gynecologic practice. In the not too distant past, menstrual disorders were managed by either empiric medical or surgical therapies, with little understanding of specific etiologic factors. Modern concepts of gynecologic neuroendocrinology and pathology have significantly extended the clinicians’ understanding and choices of therapy for menstrual complaints.

Unfortunately, a not insignificant number of patients continue to have problems of menstruation for which no apparent biologic abnormalities can be found, while others appear to demonstrate a clear-cut menstrual disorder but fail to respond to accepted therapy. Still other patients provide a confusing clinical picture with conflicting historic, physical, and laboratory findings. In just such clinical situations, a psychosomatic perspective is proposed. Early life experiences, socioenvironmental factors, and stress may be productively explored within a psychobiologic context. Recognition is given to the understanding that diseases are often heterogeneous and multifactorial in nature. Further, brain mechanisms most likely play a role in some stage of most diseases, including dysfunction of the female reproductive system. And finally, that psychosomatic principles of diagnosis and management are not merely intuitive or common sense approaches but increasingly rest on a growing body of behavioral science knowledge and experience.

**References**


7. Frisch RE, Ravell ER. Height and weight at menarche and a hypothesis of critical body

