

Dyspareunia: An Integrated Approach to Assessment and Diagnosis

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Dyspareunia, or painful intercourse, is frequently referred to as the most common female sexual dysfunction. It can occur singly or be manifested in combination with other psychosexual disorders. Diagnosis of dyspareunia is appropriate in cases in which the experience of pain is persistent and severe. There has been little agreement concerning the origin of dyspareunia. Organic conditions and psychological variables have alternately been presented as major factors in causality. There is a presumed high incidence of physical disease associated with dyspareunia when compared with other female sexual dysfunctions. In the majority of cases, however, organic factors are thought to be rare in contrast with sexual issues and interpersonal or intrapsychic difficulties as a cause of continuing problems.

The finding of an organic basis for dyspareunia does not rule out emotional or psychogenic causes. Thorough and extensive gynecologic and psychological evaluation is essential in cases of dyspareunia. The etiology of dyspareunia should be viewed on a continuum from primarily physical to primarily psychological with many women falling in the middle area.

A recurrent pattern of genital pain during or immediately after coitus is the basis for the diagnosis of dyspareunia.¹ *The Diagnostic and Statistical Manual of Mental Disorders (DSM-III)*² has included dyspareunia under the classification of psychosexual disorders. Dyspareunia is defined as functional when the occurrence of recurrent and persistent genital pain in either the man or woman is associated with coitus. Additionally, diagnostic criteria state that it is not caused exclusively by a physical disorder, is not because of a lack of lubrication (criteria for inhibited sexual excitement), and is not the result of functional vaginismus or another clinical syndrome. Dyspareunia is far more common in the woman than in the man,^{3,4} and female dyspareunia is more likely to involve psychological factors than is male dyspareunia.⁵ This article will restrict its discussion to dyspareunia in women.

Dyspareunia and vaginismus are undeniably linked, and repeated dyspareunia is likely to result in vaginismus, as vaginismus may be the causative factor in dyspareunia.^{6,7} The difference between vaginismus and dyspareunia is that intromission is generally painful in the latter condition but virtually impossible in vaginismus because of the involuntary muscle contraction of the vaginal sphincter.⁸ If vaginismus is present, concurrent dyspareunia is not considered to be a separate entity, as any persistence or success with forced penile entry is invariably painful.⁹ If vaginismus is the cause of dyspareunia, the primary diagnosis is vaginismus.¹

Many women with dyspareunia are anorgasmic as well, and for a significant number the anorgasmia precedes the dyspareunia.^{3,10-12} Although there are supportive data, no conclusive evidence is available that confirms anorgasmia necessarily precedes the development of dyspareunia.

Even though precise statistics regarding the incidence of dyspareunia are not available, Brant¹³ notes that the complaint of dyspareunia is a common clinical presentation. Kaplan¹² and Masters and Johnson³ report that next to anorgasmia, dyspareunia is the most frequently occurring sexual dysfunction. Lamont⁶ points out that probably every sexually active woman

Submitted, revised, August 5, 1986.

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has had occasional dyspareunia; consequently, the incidence of the isolated symptom may be said to be universal. Fordney and Settlege¹¹ report that in women of lower socioeconomic class with a higher proportion of non-European white racial background, dyspareunia is the most common sexual dysfunction complaint. Fordney⁹ comments, however, that this finding may simply reflect that dyspareunia is a more disabling dysfunction than anorgasmia. Fordney points out that women with fewer resources may live with the absence of orgasm without requesting treatment, but in the presence of pain that seriously alters their ability to engage in sexual behavior, they will seek assistance.

DESCRIPTION AND CLASSIFICATION

Dyspareunic pain has been described and classified in a variety of ways. Claye¹⁴ categorized dyspareunia in two ways: superficial if difficulty or pain occurs during penetration, or deep when pain occurs after penetration. Fink¹⁰ separates pain experienced at the vaginal outlet from pain felt on deep penetration. Similarly, pain experienced during intercourse is described by Abarbanel⁵ as ranging from postcoital vaginal irritation to severe pain during penile thrusting. Lazarus¹⁵ reports that dyspareunia pain or discomfort may be described in terms of pressure, aching, tearing, or burning and may have a wide range of individual intensity and duration. Fordney⁹ states a coital episode may be slightly painful with intromission, momentarily during the first few thrusts, or minimally painful throughout coitus and not interfering with desire, receptivity, or orgasm; therefore she reserves the syndrome diagnosis for those cases in which pain is usually persistent and severe. Her definition excludes occasional dyspareunia caused by a lack of arousal and resultant lack of lubrication, prolonged coital contact, or transient conditions caused by infections. Several authors hypothesize that women who experience dyspareunic pain without supposed organic involvement are unlikely to be able to localize specifically or to describe the character of their pain, and will report it as diffuse and not typically persisting following termination of coitus.^{3,10}

A variety of classification systems have been used to delineate dyspareunia. Worchester¹⁶ utilized the terms primary and secondary to connote physical and psychological etiology, respectively. Hartnell¹⁷ approached the terms quite differently: primary as usually manifest immediately after marriage, and secondary appearing some years later, frequently after the birth of one or more children. Spano and Lamont⁷ regard dyspareunia as primary when penetration has always been painful, or secondary when the onset of painful intercourse occurs following previously comfortable intromission. Brant¹³ distinguishes superficial

type dyspareunia as originating from failure of lubrication, and deep dyspareunia as stemming from failure of vaginal relaxation and ballooning, occurring in an episodic or random fashion.

Given this lack of consistency in term usage, the subclassification of sexual dysfunction types utilized in DSM-III² provides a somewhat more comprehensive framework from which to assess and diagnose dyspareunia. The symptom is described as primary or secondary and as complete or situational. A primary condition is defined as existing throughout the sexual lifetime of a woman, whereas a secondary symptom is one that developed after a significant symptom-free period. A complete symptom is one that is present under all circumstances rather than occurring selectively with specific situations, such as one particular partner, a type of stimulation, or other external variables. By definition dyspareunia is restricted to coital activity as a type of stimulation. General current categorization of the term *superficial* is applied to pain perceived at or near the introitus or vaginal barrel, and *deep* as located at the cervix or lower abdominal area.^{10,11} As there is frequent occurrence of more than one sexual dysfunction in the same individual,^{3,12} the arrangement of these disorders by subclassification and relative time of onset can be extremely useful in determining etiology and implementing appropriate treatment.⁹

ETIOLOGY

The etiology of dyspareunia has traditionally been divided into two classes, organic and psychogenic.⁷ Lamont¹⁸ reports that some of the gynecologic literature on dyspareunia has tended to discuss conditions that could be treated medically or surgically, while conversely, behaviorists have expounded on psychogenic causes of the syndrome. Although gynecologists are frequently called upon by referring mental health professionals to rule out organic causes in dyspareunia, Grillo and Grillo¹⁹ state residency programs are woefully inadequate in this area of gynecology, and standard texts rarely devote even minimal attention to the disorder. Fink¹⁰ also comments on the existing gap between gynecologists and the psychological realm, and stresses that an understanding and acceptance of psychological causality in painful intercourse are essential. The literature obviously presents dichotomous viewpoints. Organic pathology and psychological variables are alternately proposed as the major factors in causality of dyspareunia. In illustration, Huffman²⁰ contends psychogenic dyspareunia is relatively uncommon; alternately, Spano and Lamont⁷ state organic factors underlying dyspareunia are usually temporary, easily correctable, and rare as a cause of a continuing problem.

Wabrek and Wabrek²¹ stress that although it is dif-

difficult for gynecologists to determine causality of dyspareunia as organic or functional, no woman should be labeled as having dyspareunia of a psychosomatic etiology without a very thorough, extensive gynecologic evaluation. Fordney⁹ states that dyspareunia is the only sexual dysfunction in which there is a presumed high incidence of physical disease etiology or association. Among the physical factors commonly cited as cause of dyspareunia are a rigid hymen, painful hymenal tags, endometriosis, pelvic inflammatory diseases, senile atrophy of the vagina, relaxation of the supporting uterine ligaments, pelvic tumors, childbirth pathologies, stenosis of the vagina, urethral carbuncle and hemorrhoids.¹² Imperforate and abnormally fibrous hymen and hymenal rings and remnants have been found to be a physical cause as well.¹⁹ Fink¹⁰ adds to the list episiotomy scars, Bartholin's gland inflammations, clitoral inflammation and adhesions, lesions of the vulva, a variety of vaginal infections, radiation vaginitis, iatrogenic causes, and allergic reactions to contraceptive and douching materials. Spano and Lamont⁷ report that many complaints of dyspareunia are caused by sensitivity to self-administered irritants, such as feminine hygiene deodorants or restrictive clothing. They additionally contend feminine hygiene products are unnecessary and may be damaging. Other possible organic causes of dyspareunia include trauma, irradiation tumors, cystitis, constipation, proctitis, and ectopic pregnancy.²²

Fordney⁹ states that dyspareunia is not a constant symptom of any one pelvic disease, as there are well-recorded instances of extensive disease in which dyspareunia was conspicuously absent from the symptom complex. Fordney reports that most women seeking treatment for dyspareunia have experienced chronic pain from two to six years, and that the presence of subtle or missed physical factors is common in women who have longstanding dyspareunia yet have been considered to be gynecologically normal.

Many authors cite insufficient vaginal lubrication as a major causal factor of dyspareunia.^{3,13,23} Insufficient lubrication may be a constant phenomenon or more situational in nature, and may be related to lack of interest, fear of pregnancy, pain, fear of loss of control, or anger at the male partner.²¹ Anxiety has been implicated in the etiologic origin of dyspareunia as a result of its interfering effect on lubrication, which can lead to pain, burning, itching, and aching during and following intercourse.^{3,24} Spano and Lamont⁷ cite failure of arousal as a potential cause of dyspareunia. The female sexual response cycle includes vasocongestion and neuromuscular excitation, which produces lubrication of the vagina and erection of the clitoris that results in vaginal expansion and elevation of the uterus.^{3,12} They maintain that each of these steps is necessary if penetration is to be accomplished without the experience of pain. DSM-III² assigns lack of lubrication as a diagnostic criteria for inhibited sexual excitement, and excludes it in the diagnosis of functional

dyspareunia. Fordney⁹ states that in 60 to 70 percent of the cases of longstanding dyspareunia, organic factors cannot be identified. If no physical factors can be found after a thorough gynecologic evaluation and the complaint of discomfort persists, dyspareunia is then often regarded solely as a symptom of underlying psychosexual dysfunction, and a diagnosis of psychogenic origin must be made.¹⁵

Psychological theories in regard to the etiologic origins of dyspareunia include fear-anxiety conflicts, phobic reactions, conversion reaction, and hostility toward sexual partners.^{3,10,25} Psychoanalytic theory considers dyspareunia as constituting a hysterical or conversion symptom, conceptualized as the symbolic expression of a specific unconscious intrapsychic conflict.¹² Learning theory relates the development of the dyspareunic response to lack of learning or faulty learning, and hypothesizes that a dysfunctional pattern is reinforced with each succeeding sexual contact.²⁶ Operant conditioning models theorize that based on prior accidental occurrences with either positive or aversive consequences, the individual woman develops a preconditioned set that is reinforced.¹⁰

Lazarus¹⁵ contends that dyspareunia is not a unitary disturbance, but involves a broad spectrum of personalistic and idiosyncratic variables. More generally, DSM-III² states any negative attitude toward sexuality because of particular experiences, internal conflicts, or adherence to rigid subcultural values predisposes an individual to psychosexual dysfunction. Wabrek and Wabrek⁴ report that an anxiety response may be manifested in anticipation of pain during intercourse as a result of ignorance or misinformation. Fear of pain or pregnancy and the resulting aversion to coitus may have their origins in childhood teachings or memories of distressing sexual experiences in childhood or adolescence.²⁰ Kaplan²⁷ reports on causes of psychogenic dyspareunia, including guilt about intercourse and erotic pleasure, fear of penetration, and anger at the partner. Masters and Johnson³ cite the residual aftereffects of associated psychological trauma resulting from rape as a cause of pain during intercourse.

Abarbanel⁵ reports that other psychological factors may be feelings of guilt, shame, or tension occasioned by new sexual situations, or inept precoital techniques by the man, which contribute to a lack of arousal in the woman and ultimately result in insufficient lubrication and coital discomfort. Lazarus¹⁵ contends that a major variable in psychogenic dyspareunia involves the fundamental feelings and attitudes between sexual partners. Sexual issues, such as faulty information and intrapsychic problems, appear to be more prominent in primary dyspareunia, whereas in secondary dyspareunia, relationship issues may be more important.¹¹

As long ago as 1931, Dickinson²⁸ proposed an interesting conceptualization of dyspareunia that addressed the reasoning of both organic and psychogenic camps. He argued that physical dyspareunia is likely to be followed by psychic dyspareunia, as well as accom-

panied by it. There is also a basis for suspicion that psychic may precede physical dyspareunia in many cases. Psychic origins may take the form of physical pain, which can mask aversion, while, conversely, physical pain can be joined with psychological dissonance. Dickinson emphasized that while it is clearly apparent that logical development is difficult to establish in these cases, care should be taken to avoid one-way reasoning when reverse reasoning has an equal likelihood of proving to be true. The attitude conveyed in Dickinson's argument is in opposition to an either/or approach to assessment and diagnosis of dyspareunia. Fink¹⁰ further strengthens this point of view by stressing that finding a physical cause for dyspareunic pain does not mean that all psychological matters can be set aside or considered solved.

Dickinson's discussion clearly emphasizes careful evaluation of the emotional and psychological aspects of each case of dyspareunia in addition to the physical aspects. The preceding review of dyspareunia indicates that despite the above conceptualization having been present in the literature for over 50 years, a balanced approach to this sexual dysfunction has been largely lacking in clinical and research reports, with authors aligning themselves with one side of the continuum, paying only minor lip service to alternative etiologic factors. A more genuine consideration of both organic and psychological factors in understanding cases of dyspareunia should be established.

IMPLICATIONS

This review of organic and psychogenic causative factors in the development of dyspareunia suggests a general lack of agreement concerning etiology of the disorder. While authors in both camps present factual and convincing arguments, the singling out of organic or psychological causes is neither warranted nor essential and, in fact, could be counterproductive in many instances.

Implications of this review point to a need for the medical and psychological community to increase their level of awareness and expertise in regard to the complexity of factors involved in this syndrome. Heretofore, rigid assumptions about the strict causality of this disorder appear to be erroneous and misinformed. Approach to diagnosis in a stepwise fashion, emphasizing etiologic factors as they are discovered, would appear to be most beneficial. Viewing causality on a continuum as primarily of a physical or psychogenic nature with the potential for both to be equal contributors to dyspareunic pain is more practical and efficacious. The use of a consistent diagnostic battery is recommended, as is the development of new assessment procedures.

An integrated rather than dichotomous view of dyspareunia, combined with increased knowledge of

possible etiologic factors of both organic and psychogenic origin, would appear to enhance the chances for accurate assessment and diagnosis. Rather than medical and psychological professionals working at odds in a unitary fashion, collaborative research efforts incorporating the knowledge and expertise of both disciplines should be employed. Additionally, reciprocal consultation and referral by both physicians and mental health practitioners are essential and in keeping with the advocated approach.

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BRIEF SUMMARY
DIABINESE® (chlorpropamide)
 TABLETS, USP

CONTRAINDICATIONS

DIABINESE is contraindicated in patients with:
 1. Known hypersensitivity to the drug.
 2. Diabetic ketoacidosis, with or without coma. This condition should be treated with insulin.

WARNINGS

SPECIAL WARNING ON INCREASED RISK OF CARDIOVASCULAR MORTALITY

The administration of oral hypoglycemic drugs has been reported to be associated with increased cardiovascular mortality as compared to treatment with diet alone or diet plus insulin. This warning is based on the study conducted by the University Group Diabetes Program (UGDP), a long-term prospective clinical trial designed to evaluate the effectiveness of glucose-lowering drugs in preventing or delaying vascular complications in patients with non-insulin-dependent diabetes. The study involved 823 patients who were randomly assigned to one of four treatment groups (*Diabetes*, 19 (supp. 2):747-830, 1970).

UGDP reported that patients treated for 5 to 8 years with diet plus a fixed dose of tolbutamide (1.5 grams per day) had a rate of cardiovascular mortality approximately 2 1/2 times that of patients treated with diet alone. A significant increase in total mortality was not observed, but the use of tolbutamide was discontinued based on the increase in cardiovascular mortality, thus limiting the opportunity for the study to show an increase in overall mortality. Despite controversy regarding the interpretation of these results, the findings of the UGDP study provide an adequate basis for this warning. The patient should be informed of the potential risks and advantages of DIABINESE and of alternative modes of therapy.

Although only one drug in the sulfonylurea class (tolbutamide) was included in this study, it is prudent from a safety standpoint to consider that this warning may also apply to other oral hypoglycemic drugs in this class, in view of their close similarities in mode of action and chemical structure.

PRECAUTIONS

General

Hypoglycemia: All sulfonylurea drugs are capable of producing severe hypoglycemia. Proper patient selection, dosage, and instructions are important to avoid hypoglycemic episodes. Renal or hepatic insufficiency may cause elevated blood levels of DIABINESE and the latter may also diminish gluconeogenic capacity, both of which increase the risk of serious hypoglycemic reactions. Elderly, debilitated or malnourished patients, and those with adrenal or pituitary insufficiency are particularly susceptible to the hypoglycemic action of glucose-lowering drugs. Hypoglycemia may be difficult to recognize in the elderly, and in people who are taking beta-adrenergic blocking drugs. Hypoglycemia is more likely to occur when caloric intake is deficient, after severe or prolonged exercise, when alcohol is ingested, or when more than one glucose-lowering drug is used.

Because of the long half-life of chlorpropamide, patients who become hypoglycemic during therapy require careful supervision of the dose and frequent feedings for at least 3 to 5 days. Hospitalization and intravenous glucose may be necessary.

Loss of control of blood glucose: When a patient stabilized on any diabetic regimen is exposed to stress such as fever, trauma, infection, or surgery, a loss of control may occur. At such times, it may be necessary to discontinue DIABINESE and administer insulin.

The effectiveness of any oral hypoglycemic drug, including DIABINESE, in lowering blood glucose to a desired level decreases in many patients over a period of time, which may be due to progression of the severity of the diabetes or to diminished responsiveness to the drug. This phenomenon is known as secondary failure, to distinguish it from primary failure in which the drug is ineffective in an individual patient when first given.

ADVERSE REACTIONS

Hypoglycemia: See PRECAUTIONS section.

Gastrointestinal Reactions: Cholestatic jaundice may occur rarely. DIABINESE should be discontinued if this occurs. Gastrointestinal disturbances are the most common reactions; nausea has been reported in less than 5% of patients, and diarrhea, vomiting, anorexia, and hunger in less than 2%. Other gastrointestinal disturbances have occurred in less than 1% of patients including proctocolitis. They tend to be dose related and may disappear when dosage is reduced.

Dermatologic Reactions: Pruritus has been reported in less than 3% of patients. Other allergic skin reactions, e.g., urticaria and maculopapular eruptions have been reported in approximately 1% or less of patients. These may be transient and may disappear despite continued use of DIABINESE; if skin reactions persist the drug should be discontinued.

Porphyria cutanea tarda and photosensitivity reactions have been reported with sulfonylureas. Skin eruptions rarely progressing to erythema multiforme and exfoliative dermatitis have also been reported.

Hematologic Reactions: Leukopenia, agranulocytosis, thrombocytopenia, hemolytic anemia, aplastic anemia, pancytopenia and eosinophilia have been reported with sulfonylureas.

Metabolic Reactions: Hepatic porphyria and disulfiram-like reactions have been reported with DIABINESE.

Endocrine Reactions: On rare occasions, chlorpropamide has caused a reaction identical to the syndrome of inappropriate antidiuretic hormone (ADH) secretion. The features of this syndrome result from excessive water retention and include hyponatremia, low serum osmolality, and high urine osmolality.

DOSAGE AND ADMINISTRATION

There is no fixed dosage regimen for the management of diabetes mellitus with DIABINESE or any other hypoglycemic agent. In addition to the usual monitoring of urinary glucose, the patient's blood glucose must also be monitored periodically to determine the minimum effective dose for the patient, to detect primary failure, and to detect secondary failure. Glycosylated hemoglobin levels may also be of value in monitoring the patient's response to therapy.

The total daily dosage is generally taken at a single time each morning with breakfast. Occasionally cases of gastrointestinal intolerance may be relieved by dividing the daily dosage. A LOADING OR PRIMING DOSE IS NOT NECESSARY AND SHOULD NOT BE USED.

Initial Therapy: 1. The mild to moderately severe, middle-aged, stable, non-insulin-dependent diabetic patient should be started on 250 mg daily. Older patients should be started on smaller amounts of DIABINESE, in the range of 100 to 125 mg daily.

2. No transition period is necessary when transferring patients from other oral hypoglycemic agents to DIABINESE. The other agent may be discontinued abruptly and chlorpropamide started at once. In prescribing chlorpropamide, due consideration must be given to its greater potency.

Many mild to moderately severe, middle-aged, stable non-insulin-dependent diabetic patients receiving insulin can be placed directly on the oral drug and their insulin abruptly discontinued. For patients requiring more than 40 units of insulin daily, therapy with DIABINESE may be initiated with a 50 per cent reduction in insulin for the first few days, with subsequent further reductions dependent upon the response.

Five to seven days after the initial therapy, the blood level of chlorpropamide reaches a plateau. Dosage may subsequently be adjusted upward or downward by increments of not more than 50 to 125 mg at intervals of three to five days to obtain optimal control. More frequent adjustments are usually undesirable.

Maintenance Therapy: Most moderately severe, middle-aged, stable non-insulin-dependent diabetic patients are controlled by approximately 250 mg daily. Many investigators have found that some milder diabetics do well on daily doses of 100 mg or less. Many of the more severe diabetics may require 500 mg daily for adequate control. PATIENTS WHO DO NOT RESPOND COMPLETELY TO 500 MG DAILY WILL USUALLY NOT RESPOND TO HIGHER DOSES. MAINTENANCE DOSES ABOVE 750 MG DAILY SHOULD BE AVOIDED.

HOW SUPPLIED

Blue, D-shaped, scored tablets in strengths of 100 mg, tablet code 393, (100's, NDC# 0663-3930-66, 500's, NDC# 0663-3930-73, and 10 x 10, NDC# 0663-3940-30); 250 mg, tablet code 394, (100's, NDC# 0663-3940-66, 250's, NDC# 0663-3940-71, 1000's, NDC# 0663-3940-82, 100 unit dose of 10 x 10, NDC# 0663-3940-41, and 30's D-Pak, NDC# 0663-3940-30).

RECOMMENDED STORAGE: Store below 86°F (30°C).

CAUTION: Federal law prohibits dispensing without prescription.

