

# The false organic–psychogenic distinction and related problems in the classification of erectile dysfunction

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**The traditional distinction between organic and psychogenic erectile dysfunction (ED) was maintained in the recent report of the Nomenclature Committee of the International Society for Sexual and Impotence Research. Among the major problems with this distinction are that it is based on an obsolete view of mind–body distinctions, does not take into account knowledge of the neurobiology of ‘psychological’ disorders, disregards the fundamental meaning of ‘psychosomatic,’ is too often diagnosed by exclusion, and may imply to the patient that his ED is ‘all in the mind.’ As a result, the distinction has become counterproductive in the diagnosis, classification, and treatment of ED, and in research into the causes of ED. An alternative taxonomy, based on that proposed by the Nomenclature Committee, reclassifies as organic several of the causes of ED now considered to be psychogenic, and considers others as situational ED, a class reserved for episodic occurrences of ED clearly due to particular attributes of sexual encounters.**

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The goal of this paper is to bring an end to the traditional distinction made between ‘organic’ and ‘psychogenic’ causes of erectile dysfunction (ED). This distinction may once have been useful, but it is demonstrably flawed in several respects, and is probably counterproductive in terms of diagnosis, treatment, research, and even as a pedagogical device. Before presenting this argument, some background may prove useful.

Some medical bodies have formal diagnostic taxonomies. For example, the American Psychiatric Association has over several decades developed and published the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), currently in Version IV.<sup>1</sup> The DSM has evolved over its successive editions, including several fundamental changes in classification, while other changes just involved the names of conditions. Other medical groups have not found it necessary to adopt such taxonomies formally, although they may use them as a matter of convention, for example, the classification of headaches by neurologists.<sup>2</sup> Even when not formally adopted by a

medical organization, classification systems are often explicit in the questionnaires or billing forms used by health care providers and insurers, and they are implicit in the jargon of medical journals.

The International Society for Sexual and Impotence Research (ISSIR) has for some years considered adopting a formal taxonomy of ED. Toward that end, a Nomenclature Committee was formed, and it recently published its recommended classification,<sup>3</sup> which is summarized in Table 1. Although the committee debated whether or not to retain the distinction between organic and psychogenic ED, in the end they decided to maintain its centrality in the recommended taxonomy.<sup>4</sup> This nosology has not been formally discussed or adopted by the ISSIR membership; perhaps it never will be. However, publication of the recommendation offers risks and opportunities. One of the risks is that dissemination of the taxonomy *ipso facto* encourages its usage and serves as formal endorsement of its features and terminology, including the continued distinction between organic and psychogenic ED. One of the opportunities afforded (and probably expected by the committee) is to reflect further upon certain problems with the proposed taxonomy. (A step in that direction was taken during a symposium on taxonomic issues at a recent meeting of the ISSIR.<sup>5</sup>)

Before this or another classification system for ED becomes codified by formal vote or by custom, the

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**Table 1** Classification of erectile dysfunction recommended by the Nomenclature Committee of the International Society for Impotence Research\*

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Organic
I. Vasculogenic
A. Arteriogenic
B. Cavernosal
C. Mixed
II. Neurogenic
III. Anatomic
IV. Endocrinologic
Psychogenic
I. Generalized type
A. Generalized unresponsiveness
1. Primary lack of sexual arousability
2. Aging-related decline in sexual arousability
B. Generalized inhibition
1. Chronic disorder of sexual intimacy
II. Situational type
A. Partner-related
1. Lack of arousability in specific relationship
2. Lack of arousability due to sexual object preference
3. High central inhibition due to partner conflict or threat
B. Performance-related
1. Associated with other sexual dysfunction/s (eg, rapid ejaculation)
2. Situational performance anxiety (eg, fear of failure)
C. Psychological distress or adjustment related
1. Associated with negative mood state (eg, depression) or major life stress (eg, death of partner)

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\*Adapted from Appendix 1 of Lizza and Rosen.<sup>3</sup>

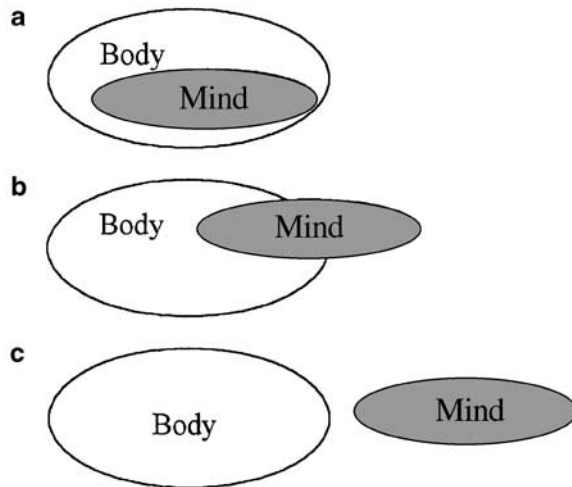
problems with ‘psychogenic’ ED as a category, and therefore with the organic–psychogenic distinction, should be examined more closely. Among these problems are that the category of ‘psychogenic’ ED (a) is based on an obsolete view of mind–body distinctions, (b) disregards knowledge of the neurobiology of ‘psychological’ disorders, (c) disregards the fundamental meaning of ‘psychosomatic,’ (d) is too often diagnosed by exclusion, and (e) may imply to the patient that his ED is ‘all in the mind.’

## Problems with the concept of psychogenic ED

(a) ‘Psychogenic’ ED is based on an obsolete view of mind–body distinctions

The distinction between organic and psychogenic ED reflects the historical division between body and mind, a division that takes inadequate account of modern physiological research and that belies the axiom that all psychological processes have a somatic basis. It is both unnecessary and beyond the scope of this paper to review the philosophical history of the so-called mind–body problem. Suffice it to say that one can idealize two camps of adherents. One camp comprises the strict reduc-

tionists, who believe that the mind is *nothing but* the brain at work, that is, that all mental processes can be explained in terms of brain processes. Their view is embodied in the Venn diagram in Figure 1a. An example of this view is that of the philosopher JR Searle, who has noted that: ‘Once we see that consciousness is a biological phenomenon like any other, then it can be investigated neurobiologically. Consciousness is entirely caused by neurobiological processes and is realized in brain structures.’<sup>6,7</sup> Others believe that mind is *something more* than the brain at work, that is, that there are ‘emergent processes’ of mind that will never be fully explainable by analysis of brain function. This view is depicted in Figure 1b. But to my knowledge, all agree that mind is not something altogether *other* than brain function; that is, they reject Figure 1c as a viable option. Countless experimental psychologists, linguists, anthropologists, and other behavioral scientists rigorously analyze mental processes without considering brain function, but they would nonetheless be likely to agree that *all psychological processes are regulated by brain functions*. It follows that there can be no *psychogenic* dysfunction that does not involve *organic* processes. There can be no ED that is ‘all in the mind.’ (see Section (e), below.) Brain function is of course also essential to the normal stimulation and inhibition of erection.<sup>8–11</sup> Neither ‘psychogenic’ erection nor ‘psychogenic’ ED can occur except through the mediation of the brain’s processes.



**Figure 1** Venn diagrams of three idealized views of the relation between mind and body. (a) View of strict reductionists: all mental processes can be explained in terms of brain processes. (b) Emergent process view: some mental processes will never be fully explainable by analysis of brain function. (c) Independence view (a null set?): mind is something altogether other than brain function.

(b) ‘Psychogenic’ ED disregards knowledge of the neurobiology of ‘psychological’ disorders

Some of the subcategories of ‘psychogenic’ ED in the taxonomy of the Nomenclature Committee serve well to exemplify this argument for rejecting ‘psychogenic’ as a category of ED. Thus, ‘negative mood states’ such as depression and ‘major life stress’ are included as types of situational ED that are ‘psychological distress or adjustment related.’ However, neurobiology has made great progress in discovering the neural bases of emotions and disorders of emotion.<sup>12,13</sup> Indeed, depression, stress, and anxiety are among the psychological conditions for which recent research has most clearly demonstrated major neurochemical and neuroendocrine changes in the brain.<sup>14–18</sup> Some of these changes might be expected to contribute to impaired erectile function. (Revealingly, Lue<sup>19</sup> also classifies stress and depression as psychogenic causes of ED, but demonstrates their organic nature by offering impaired release of nitric oxide as part of the underlying pathophysiology.) Furthermore, these neurochemical discoveries have led to drug treatments that can ameliorate these ‘negative mood states.’ Not surprisingly, these treatments sometimes reduce the ED associated with these conditions, but in other cases the drugs alter the neurochemical balance and impair sexual function through such ‘side effects’ as ED, delayed ejaculation, or impaired sexual desire.<sup>20</sup> In other words, when ‘psychogenic’ ED is viewed as a kind of brain ‘neurogenic’ ED, then it is to be expected that ‘psychotropic’ drugs,

through their effects on the brain, can either reduce or increase this neurogenic ED. Put another way, the neuroendocrine and neurochemical bases of anxiety and depression are no less organic causes of ED than is ED due to hypogonadism or hyperprolactinemia.

This problem with ‘psychogenic’ ED is also exemplified by the Committee’s inclusion in this category of ‘aging-related decline in sexual arousability.’ Many aspects of sexual function decline as men age, and they do so for many reasons.<sup>21–23</sup> Thus, the age-related decline in erectile function can result from degenerative changes in the vascular system of the penis, or in penile collagen, or in the peripheral nerves, all of which would presumably be categorized as ‘organic’ rather than ‘psychogenic’ age-related causes of ED. But some of these changes may also contribute to the age-related decline in sexual arousability, just as a reduction in taste and odor sensitivity or in digestive function may curb the appetite for food.<sup>24,25</sup> Some of the loss of sexual arousability with age may also result from age-related neurochemical changes in the brain. These changes and their possible effects on ED are not yet well documented or understood, but are deserving of research.

The difficulty of maintaining a clear distinction between psychogenic and organic ED is also evident in the ground-breaking analyses of sexual response by John Bancroft and Erick Janssen. Concurrent with their reviews of central inhibitory processes in sexual function, and their theoretical contributions to our understanding of those processes,<sup>26,27</sup> Bancroft and Janssen<sup>28</sup> used psychometric testing and statistical factor analysis to parse sexual arousal into three relatively independent underlying processes, one for sexual arousal and two for sexual inhibition, whose relative balance is predictive of men’s erectile problems. One of their contributions is entitled ‘Psychogenic erectile dysfunction in the era of pharmacotherapy: a theoretical approach’ (emphasis added), but Bancroft and Janssen allude throughout their analysis to the neurobiological regulation of sexual function, and they raise this question (p. 86): ‘If these predictions become supported by the clinical evidence, what will that tell us about the concept of ‘psychogenic ED’? The distinction between ‘psychogenic’ and organic’ is already of diminishing clinical value. More often than not a ‘mixed’ diagnosis is made.’ Rather than discard the distinction, Bancroft and Janssen suggest that the momentary balance between, on the one hand, central and peripheral organic inhibition and, on the other hand, ‘external problems’ determines whether a man is prone to have ED in a particular sexual encounter. Yet, Bancroft and Janssen note the neurobiological mediation of the processing of external problems: ‘Men with relatively high propensity for central inhibition of sexual response are more likely to lose sexual interest and erectile responsiveness when depressed or anxious. This

may not depend on cognitive processing primarily but on the related biochemical changes in the brain which are relevant to both mood and sexual arousability.’ (p. 87) However if these psychogenic factors are regulated by organic factors, why maintain them as separate classes? And even when cognitive processing is involved, that processing is no less biochemically mediated by neurochemical changes in the brain than are noncognitive processes.

(c) ‘Psychogenic’ ED disregards the fundamental meaning of ‘psychosomatic’

The well-established field of psychosomatics would seem, in the very roots of its name, to embody the same obsolete distinction between mind and body that has already been criticized here. However, when psychosomatics is viewed in terms of the Venn diagrams in Figure 1a or b, then it can be seen as the science of the interactions among the brain’s cognitive functions, its autonomic functions, and other bodily processes involved in health and disease. These interactions are epitomized by two of the more recently established subdisciplines of psychosomatics, ie. psychoneuroendocrinology and psychoneuroimmunology, and their respective journals, *Psychoneuroendocrinology* and *Brain, Behavior, and Immunity*. These disciplines emphasize the systemic interaction of their parts, rather than their separation. Analogously, we should consider psychological processes as inextricably bound with organic processes of erectile function and dysfunction, rather than as separate pigeonholes to which relative causation can be assigned. This view applies to ED the more general statement made by HG Wolff in his presidential address at the 1961 meeting of the American Neurological Association: ‘It is unprofitable to establish a separate category of illness to be defined as psychosomatic. Rather, man’s nervous system is implicated in all categories of disease.’<sup>29</sup>

One clear interface between the psychological and the somatic is in the raised fears about adequacy of sexual performance that organic conditions tend to provoke. As Bancroft and Janssen, among others, have noted, relatively slight or occasional impairments in erectile function can result from a large number of conditions, whether chronic (eg, vascular problems, peripheral neuropathy) or acute (excessive alcohol consumption). Additional deficits may result if a man worries about this mild impairment. That is, the cognitive feedback from slight erectile dysfunction can lead to ‘performance anxiety,’ which may summate with the other condition(s) to further impair erectile function. Performance anxiety, like any other anxiety, is clearly a psychological

term for a psychological state. However as we have already noted, anxiety is also and equally an organic/physiological condition that can be treated with anxiolytic drugs. Some of these drugs, in some doses, may be expected to interrupt the positive feedback that aggravates ED. Indeed, consumption of small amounts of alcohol is a traditional folk medicine for increasing sexual appetite and reducing performance anxiety, thereby promoting erectile function. (The most frequently cited authority on larger doses is probably William Shakespeare, who noted (*Macbeth* Act II, Scene 2), that alcohol ‘provokes the desire, but it takes away the performance.’)

An excellent example of how expectations can affect erectile function comes from a study by Cranston-Cuebas *et al.*<sup>30</sup> They compared sexually functional and dysfunctional men viewing erotica after taking each of three placebo pills purported to enhance erection, to impair erection, or to be placebos. As might be expected, the dysfunctional men had lesser erections with the alleged impairing drug. Surprisingly, sexually functional men actually had stronger erections with the supposed detractor, a reverse placebo effect. These results may be viewed as examples of situational erectile (dys)function, where the function varies according to other aspects of the men, including their constitutional central excitatory and inhibitory states, their sexual histories, and the immediate conditions of the sexual encounter.

(d) ‘Psychogenic’ ED is too often diagnosed by exclusion

Ideally, the diagnosis of the causes of ED includes a thorough physical examination and extensive interviewing, possibly including standardized psychological tests, to establish the history of the dysfunction and the circumstances under which it occurs. Further testing may include a test for nocturnal penile tumescence, or sleep-related erection (SRE), which at least at one time was considered to be definitive.<sup>31,32</sup> That is, if SRE was normal and there was no evidence of organic pathology, then it was presumed that there was no physical problem to prevent erection during a sexual encounter, and a diagnosis of psychogenic ED was likely. This process exemplifies diagnosis by exclusion.

The unreliability of this conclusion is well documented, among other reasons because conditions like depression can themselves impair SRE.<sup>33,34</sup> Furthermore, research into the brain’s regulation of SRE in animals has revealed that some of the brain areas mediating SRE are different from those regulating erection during sexual stimulation. Specifically, lesions in the lateral preoptic area of

the hypothalamus of rats drastically curtail erection during REM sleep without affecting REM sleep *per se*, and without affecting erection in any other context.<sup>35</sup> This example is just one of many showing that the neural and endocrine mediation of erection differs from one sexual context to another, depending on whether the sexual stimulation is genital touch, copulation, a response to remote stimulation such as smell or sight, or sleep related.<sup>11</sup> Therefore, disorders in the organic basis of erection in one context may or may not be predictive of ED in another context.

*(e) ‘Psychogenic’ ED is not ‘all in the mind’*

In the United States and much of the world, the public regards ‘medical’ problems and ‘psychological’ problems differently, generally stigmatizing the latter but not the former (with certain exceptions like sexually transmitted diseases and addictions). Coincidentally, health care providers, the insurance industry, and government treat the two ‘sorts’ of problems differently in terms of coverage and compensation. ‘Mental’ problems are commonly not covered by insurance, and if they are, then fewer physician visits are permitted and less money is paid to them. Given this social background, it is not surprising that medical problems with psychosomatic features have often been dismissed as being ‘all in the mind’ and have been thereby stigmatized. Even when the diagnosis of psychogenic ED is not intended to imply that ‘it’s all in your mind,’ the patient may understandably make that inference, or at least infer that that is what the physician believes. In recent years, professionals have widely adopted the term ‘erectile dysfunction’ to avoid the stigma carried by ‘impotence.’ (Note, however, that ‘im-

potence’ persists in the name of this journal and its parent society.) Perhaps it is time to recognize that for those who receive this diagnosis, ‘psychogenic ED’ is probably not much less problematic than ‘psychogenic impotence.’ If physicians can come to understand that there is an organic basis for what is now considered to be chronic psychogenic ED, then the associated stigma may be reduced, and treatment may be more readily covered by insurance. One result might be that men would be more likely to seek treatment. Indeed, some currently available drugs are apparently effective with both situational and organic ED of many origins. However, the availability of effective medical treatments for situational ED does not argue against the utility of psychotherapeutic approaches to the problem. There is evidence that psychotherapy for certain ‘mental disorders’ changes the brain physiology that characterizes such disorders.<sup>36</sup> So too it is reasonable to infer that effective ‘talking therapies’ for ED may act by changing the underlying physiology, for example, by increasing excitation or reducing inhibition.

## An alternative taxonomy of ED

Perhaps no taxonomy of ED can achieve the kind of decision-rule branching that is available for classifying some medical conditions or identifying species of birds or trees. However, Table 2 presents an alternative to the ISIR committee’s recommended taxonomy that addresses some of the concerns raised in this paper, while retaining several of its features, its terminology, and, perhaps, its problems too. In this classification, organic ED is assigned to peripheral or central problems, that is, outside of or within the brain and spinal cord. Central problems

**Table 2** Alternative taxonomy of erectile dysfunction free of the organic–psychogenic distinction

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I. Organic
A. Peripheral
1. Vascular
2. Neural (peripheral nerves)
3. Anatomical (structural problems with penis, eg, Peyronie’s disease)
4. Endocrine (problems with gonadal or adrenal hormones, or with somatic hormone receptors, eg, in penis)
B. Central
1. Neural (overt pathology in brain or spinal cord, eg, lesion, tumor, seizures)
2. Endocrine (problems with pituitary or releasing hormones, or with hormone receptors in brain or spinal cord)
3. Generalized type
a. Generalized unresponsiveness
i. Lack of sexual arousability
ii. Age-related decline in sexual arousability
b. Generalized inhibition (eg, chronic disorder of sexual intimacy)
4. Psychological distress or adjustment-related (includes negative mood states, eg, depression, and major life stress, eg, death of partner)
II. Situational
A. Partner-related (ED only with certain partners)
B. Performance-related (eg, anxiety reerectile failure or rapid ejaculation)
C. Environment-related (eg, problem in bedroom but not elsewhere)

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include not just those that might be detected by neurological and endocrine examination, but also such conditions as depression and stress, whose central mediation has been well documented, as reviewed earlier. Also included here are factors related to aging when age-related peripheral pathology has been ruled out. This taxonomy also allows for a distinction between peripheral endocrine problems, for example, primary hypogonadism or androgen-receptor insensitivity in the genital tissues, and those of brain origin, for example, inadequate gonadotrophin-releasing hormone or problems with hormone metabolism in the brain. Situational ED is reserved for clear cases of episodic, context-sensitive ED in which certain partners, environments, or perceived performance demands impair erectile function, whereas other contexts are problem-free.

Implicit in this taxonomy is that some organic disorders may allow sleep-related erection while impairing erection in other contexts. One of the problems with the proposed taxonomy is that it retains apparent dichotomies (organic vs situational, peripheral vs central). The separation of situational ED from organic ED should not be construed as indicating that there is no clear organic basis for the situational ED which might be treatable with drugs that act upon the central nervous system (eg, anxiolytics) or upon the penis (eg, NO synthase inhibitors). Limiting this category to the more episodic occurrences of ED implies that there is no chronic pathology of the CNS that would warrant its classification under organic ED. However, these should be viewed as idealized classes; in practice, most pathology includes central and peripheral factors, and the expression of these factors will usually be affected by a man's concerns about his partner, the environment, and his sexual performance. Diagnosis of the causes of ED may be a matter of assigning priority, very much like assigning priority to causes of death on death certificates.

The diagnosis and treatment of situational and organic ED doubtless presents a major challenge. However, it should be kept in mind that even when the organic cause for a sexual problem is not identifiable, there may nonetheless be an organic cure for it. For example, acetylsalicylic acid (aspirin) treated headaches effectively long before its organic action on prostaglandin was understood. It may be useful to consider developments in the treatment of another common sexual dysfunction, namely rapid (premature) ejaculation. Until recently, this problem was usually assumed to be of psychogenic origin, and men were commonly referred for psychotherapy to treat the condition—if treatment was offered at all. Now, however, treatment with serotonergic or antiadrenergic drugs offers help in many such cases,<sup>37,38</sup> giving evidence of the organic mediation of this 'psychogenic' problem. One may suppose that future research will

determine that men who have chronic difficulty in delaying ejaculation, as well as men with frequent situational ED, tend to lie outside of the normal range of men with respect to the neurochemistry (transmitter levels, receptor density/sensitivity, etc.) of those brain areas that regulate these functions. Even 'chronic disorder of sexual intimacy' may be firmly rooted in the brain's chemistry: in some species the hormones oxytocin and vasopressin, as well as the genes controlling the expression of these hormones, regulate the tendency of males and females to form pair bonds.<sup>39</sup> To be sure, pair bonding is not the same thing as sexual intimacy, but they are related enough to expect that neurochemical differences also mediate individual differences in the capacity for sexual intimacy.

Adopting situational ED as a category parallel to organic ED, as proposed here, is one alternative. However, even situational ED is mediated by organic factors. Perhaps organic ED should be discarded as a supercategory of ED; then situational ED could be classified as a third type of ED, parallel with peripheral and central ED, or as a fifth type of central ED. Any of these alternatives seems preferable to maintaining the current division between psychogenic and organic ED, which makes a false distinction between mental and nonmental organic processes and disorders. This distinction should be discarded, not just from the taxonomy of ED, but also from systematic thinking about the causes of erection and its disorders.

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