Placing Erection in Context: The Reflexogenic–Psychogenic Dichotomy Reconsidered

BENJAMIN D. SACHS

Department of Psychology, U-20, University of Connecticut,
Storrs, CT 06269-1020 USA E-mail: bsachs@uconnvm.uconn.edu

SACHS, B. D. Placing erection in context: The reflexogenic-psychogenic dichotomy reconsidered. NEUROSCI BIOBEBHAV REV 19(2) 211-224, 1995.—Penile erections are usually classified as arising from “reflexogenic” or "psychogenic" causes. In practice this dichotomy has translated, somewhat circularly, to a distinction between spinal vs. supraspinal mediation, pelvic vs. hypogastric neural mediation, and perineal somesthetic stimulation vs. stimulation of receptors innervated by the cranial nerves. Evidence for differential regulation of erection in different contexts is reviewed. Research ascribing a physiological role to the hypogastric nerves in psychogenic erection, exemplified by classic studies of cats and spinaly injured men, is suggestive but not compelling. Somewhat stronger is evidence that erection in some contexts (e.g., nocturnal penile tumescence (NPT) in humans or touch-stimulated erection in rats) is more sensitive to androgen levels than in other contexts (e.g., visual erotic stimuli in men or copulation in rats). However, some of these differences may arise from the relative erectogenic strength of the stimuli, rather than from qualitative differences in androgen sensitivity of different contexts. More compelling is the possibility that conflicting interpretations of the role of dopamine in erection may stem in large part from differences among laboratories in the context in which erection is evoked. In light of the evidence reviewed, it seems unlikely that the conventional reflexogenic-psychogenic dichotomy should be retained, at least in its present form. As a first step, it may be worth considering that reflexive erections may not be limited to somesthetic perineal stimulation, but rather may also include stimuli received via the cranial nerves. Two alternatives to the standard reflexogenic-psychogenic dichotomy are proposed. The first is a minor revision in which two senses of psychogenic erection are distinguished: the weak, commonly used, sense would include erection resulting from any extrinsic nonsomesthetic stimulation, whether visual, auditory, or chemosensory. In this sense, reflexive erections and psychogenic erections may not be mutually exclusive. The strong sense of psychogenic erection would be limited to memory and fantasy. The origins of psychogenic erection in both senses need not be available to consciousness, which may account for apparently spontaneous erections. In the second alternative taxonomy, erectogenic stimuli are classified as contact (somatic) or noncontact, and their action in evoking erection is placed on a continuum of reflexivity. Erectile contexts could then be considered as orthogonal to the other two dimensions. Even without a change in taxonomy, the conduct and interpretation of research into erectile function may be expected to benefit from closer attention to differences and similarities between contexts and species, and to context-sensitive differences in the regulation of erection.

Erection Penis Sexual behavior Humans Animals

IT has been said that there are two kinds of people, those who dichotomize and those who do not. In fact, humans have a natural inclination toward categorical perception of even the most continuous features of their environments, such as mass and the visible spectrum (93), and this tendency is evident in many other species (85). Our proclivity to dichotomize, thereby forming the minimum number of categories, extends into the realm of concepts: mind-body, nature-nurture, motivation-performance, appetitive-consummatory are but a few of the psychobiological dichotomies that, if not immortal, at least have lives that have proved resistant to extinction despite decades of critical attacks upon them. Whatever our biological imperative to dichotomize may be, it is clear that unjustified or irrational dichotomies may impede the acquisition of knowledge. In his consideration of “the psychobiology of sexual experience,” Julian Davidson (18) critically addressed some of the polarities that have pervaded discussions of sexuality. The libido-potency distinction was central to his concerns, but he identified other problematic dichotomies: arousal-copulation, voluntary-involuntary, central-peripheral, reflex-nonreflex. This tribute to Davidson offers an occasion to examine how the last of these contrasts has been applied in the commonly made distinction between reflexogenic (or reflexive) and psychogenic penile erections.

One reason for undertaking this reexamination is increasing evidence that the physiological regulation of erection var-
ies depending on the context in which erection occurs. Much of the evidence comes from research on rats in which erection has been assessed in copula (9, 29, 39, 58, 68, 77), and in several ex copula settings including (a) unanesthetized males placed in supine position with penile sheath retracted but no further stimulation applied (9, 21, 29, 36, 39, 40, 51, 58, 77); (b) anesthe-
tized males given mechanical stimulation of the urethra or electrical stimulation of various peripheral nerves (35, 57); and (c) isolated, freely moving males receiving drugs systemically (6, 27, 76, 94) or directly into the brain (5, 60). To these contexts another has recently been added with the discovery that rats, unlike many animals, display intense erections during noncontact exposure to estrous females (75). As we shall see, this condition meets standard definitions of psychogenic erection. The discovery of these erections in rats permits experimental research into their physiological mediation, research that would be ethically impossible in humans and ethically or practically difficult with many other species that display comparable responses [e.g., chimpanzees (26), rhesus monkeys (67, 69), and squirrel monkeys (53, 54)]. However, if the typology of erections is defective, then the search for principles relating physiology to erectile responses is likely to suffer. It will become evident that the current taxonomy of erections carries explicit or implicit assumptions about physiological mediation that may not be warranted. At least these assumptions bear closer inspection than they have recently received, to assure that the taxonomy promotes accurate description and does not impede scientific or medical analysis.

It is already evident that much of the research cited in this review is from studies on rats and other animals. The arguments for the relevance of this and other animal research to the understanding of erectile processes in humans has been presented previously (e.g., 31, 59) and will not be recapitulated here. Suffice it to say that across the large evolutionary gap between rodents and humans there has been a striking conservation of relevant elements, including the basic anatomy of the penis, the mechanics of erection, and the neural structures regulating erection (18, 92). These similarities, however, should not blind us to the impressive species-typical variations that are readily evident, not the least of which may be in the cognitive domain that remains relatively unexplored. As Everitt and Bancroft commented (31, p. 106), "It may surprise the reader to even consider any parallels between psychological mechanisms affecting sexual response in the human and in the rodent. There is an understandable tendency to assume that such mechanisms in the humans will involve processes of a higher cognitive order that have no parallel in lower mammals." Equally, erection in humans may more often involve noncognitive factors than we usually acknowledge.

**PHYSIOLOGICAL BACKGROUND AND DEFINITIONS**

Before going further, it may prove useful to summarize briefly some of what is known, or at least largely agreed upon, concerning the physiological regulation of erection. Figure 1 provides a convenient point of reference for this brief review. [For further details, see, e.g., (2, 15, 23, 59, 61)]. In humans and other species (e.g., horses, dogs, rats) with a so-called vascular penis, erection results when the cavernous spaces in the penile body (the paired corpora cavernosa) and the penile glans (corpus spongiosum) fill with blood, often achieving suprasystolic pressure. The relaxation of the smooth muscles in the corpora and other vascular changes that contribute to erection are due to innervation from two sources. Parasympathetic erectile fibers in the pelvic nerves originate in the lower lumbar (in rodents) or sacral (carnivores, primates) portion of the spinal cord and reach the penis via the major pelvic ganglion. Sympathetic fibers thought to promote erection arise in the lower thoracic and upper lumbar segments of the spinal cord and sympathetic chain, and they reach the penis via the inferior mesenteric ganglion, the hypogastric nerves, and the major pelvic ganglion. For both sets of autonomic erectile fibers, the cavernous (penile) nerves are the final common path from the major pelvic ganglion to the penis. The major striated penile muscles (ischio cavernous and bulbospongious) are innervated by the motor pudendal nerves, and contraction of these muscles can augment—but can not by itself cause—penile erection, except perhaps in species with fibroelastic penises, such as ruminants (e.g., cattle, goats (59, 80)). Most penile somatosensory stimuli are conveyed to the spinal cord via the pudendal sensory nerves, where they synapse via interneurons with the pelvic and pudendal motor nerves. The erectile route from the brain to the penis is commonly ascribed to the hypo-

![FIG. 1. Schematic diagram of the excitatory (+) and inhibitory (-) innervation of the penis for the regulation of tumescence (T) and detumescence (D). The division of the penis represents the distinction between the cavernous structures of the penile body and glans, in which erection can proceed quite separately. Not depicted in the figure is the motor branch of the pudendal nerve, which innervates the striated penile muscles, but which probably also conveys fibers that promote detumescence in the cavernous bodies. The +/- signs within the spinal cord represent the potential influences of the brain on erection and detumescence, and should be understood to include the emerging hypothesis that erection may require not just the excitation of nerves promoting tumescence, but also the selective inhibition of the nerves that promote detumescence. Figure adapted from de Groat and Steers (25).](image-url)
mesent activity of these nerves is considered to be tonic, thereby keeping the penis flaccid until superseded by erectile impulses. An emerging hypothesis is that in parallel with excitation of erectile fibers, an essential part of the neural basis of erection may be an active inhibition of detumescence fibers (14,35,58). Such selective inhibition would help explain why, on the one hand, erection can occur together with such signs of sympathetic activation as increased cardiovascular activity and perspiration typical of sexual excitation, and, on the other hand, may fail to occur in stressful sexual contexts despite cognitive feelings of sexual arousal.

Perhaps because it is widely assumed that everyone knows what is meant by "erection," this and related terms are often undefined except operationally, (i.e., in terms of the instruments and measures used in particular studies). The widely different measures used among studies, especially in describing different species, limit the utility of such definitions. Therefore, it may prove useful to offer some tentative definitions relating to erection. All the definitions are based on behavior (physiology changing morphology over space, (e.g., movement), or over time (e.g., color change).

I take "erection" to mean the penile state when increased in size, form, or angle outside the normal range of its baseline state. Usually erection results at least partially from engorgement of the cavernous spaces with blood, but, depending on the species, erection can result from relaxation of the retractor penis muscle, from striated muscle contraction, or combinations of these (74,80). Erection and tumescence are syndrome, relative terms, that can apply equally to penile body and glans. This distinction between the two penile bodies is especially important in species that have only one of the structures or can externalize only one. For example, despite their similarity to humans in many respects, chimpanzees males have essentially no glans penis. Conversely, the penes of dogs have both a body and a glans, but only the glans can be extended from the sheath to be intromitted during copulation (36). The penile body of rats is made visible during reflexive erection tests by retraction of the sheath (74), and it is external but rarely glimpsed during intromission and other kins of testing (68). In species having both glans and body, erections tends to proceed synchronously in both structures, but they can erect quite independently in rats (74) and humans (13,87), at least in some contexts.

"Full erection" implies a change to or near maximum size or angle relative to resting state. "Rigidity" refers to the stiffness of the penis, which can be quite variable even after it has reached its maximum length and circumference. Among the external (noninvasive) measures of erection are changes in length, width, resistance to buckling pressure, skin temperature, and blood flow. Internal measures include blood pressure, electromyographic (EMG) activity, and activity of the nerves to the penis (17,35,39,57,71,72,80). Some of these measures (EMG and nerve activity, skin temperature, blood flow) are relevant to erection but do not, by themselves, constitute measures of erection, unless validated at some point by concurrent observation of changes in length, circumference, or pressure.

THE REFLEXOGENIC PSYCHOGENIC DISTINCTION

Representative Characterizations of the Dichotomy, and Problems Arising

With this background we can now examine more closely the common distinction made between "reflexogenic" and "psychogenic" erection. Table 1 presents 13 explicit or implicit definitions of these terms. The sample is neither random nor exhaustive, but I believe it to be representative of the range of meanings conferred on these terms. Upon inspection, several features of these examples emerge. Foremost, for present purposes, is that erections are dichotomized. The one exception (C) includes as a type the "mixed" erection that is both reflexogenic and psychogenic, a class that is implicit in the other examples. In defining the categories, some authors (e.g., I,J,M) refer exclusively or primarily to the stimuli that evoke erection, whereas others (A,K) emphasize or refer only to the neural systems that mediate erection. The emphasis is ambiguous in several cases. Furthermore, for some definitions (A,B,D,G,K) the distinction between reflexive and psychogenic erection most closely parallels that between spinal cord and brain, whereas for others (J,M) it parallels the distinction between parasympathetic (lumbosacral) and sympathetic (thoraco-columbar) nervous systems. Finally, it should be noted that where there is mention of the stimuli giving rise to psychogenic erections, none of the definitions restrict them to those arising from memory or imagination. One definition (F) gives only "erotic thoughts and fantasies" as exemplar stimuli, but the others include at least the stimulation of receptors by remote or distal stimuli (visual, auditory, olfactory); some definitions include proximal stimuli (i.e., taste and touch) the latter presumably of "erogenous" areas of the head or anterior torso.

When the excerpts are read in their original context, some of the apparent dissimilarities among the authors fade, and there seems to be a common indebtedness to Weiss (L; 91), whose review was particularly influential, and to Bors and Comarr (B; 10). One common thread running through the definitions is their apparent circularity. That is, stimuli received via the cranial nerves are by definition psychogenic for erection, whether or not higher cognitive processes may be involved in their mediation. Thus, "psychogenic" has no meaning apart from the supraspinal origin of stimulus processing. Another commonality among the authors is their ambiguous use of the term "reflex," making it difficult to extract the meaning of the term "reflexogenic erection." (I prefer the term "reflexive erection." "Reflexogenic" should mean that the response has its origin in a reflex, whereas in fact the erectile response is the expression of a reflex.) Sherrington (83, p. 116) called the "simple reflex a convenient but artificial abstraction," and the continued frequent use of the term reflex is eloquent testimony to its convenience (though few would argue that erection is a simple reflex). Perhaps it is time to reconsider whether the uncritical use of the term "reflexive" in the case of erection may be inhibiting a more critical examination of the causes of erection. At least three interacting questions are at issue here: the minimal neural system that can sustain the erectile response, the sensory system that evokes the response, and the speed of the response latency.

In parallel with its use to define a type of erection, "reflex" most commonly seems to be used synonymously with "spinally mediated," (i.e., indicating that the response can be mediated by the lower spinal cord after interruption of its connection to the brain). However, the fact that a response can occur after removal of a structure (in this case the brain) does not mean that that structure is not normally involved in its mediation. Furthermore, there are important responses at the body's extremities that are conventionally called reflexes and that involve the brain, or at least the brain stem. The startle reflex, in which loud noises may cause the whole body to jump, is a clear example of such a response (22). A more restricted example of startle, readily observed in restrained supine male rats, is the immediate retraction of the scrotum in response to small noises, presumably as a result of contraction of the cremaster muscle. Clearly, stimuli impinging on the cranial nerves and
TABLE 1
SOME CLASSIFICATIONS AND DEFINITIONS OF TYPES OF ERECTION

(A) Bancroft: "The changes in the genitalia of both the male and the female mainly result from localised vasocongestion. These local vascular changes can occur within 10 to 30 seconds of the onset of sexual stimulation, whether psychic (i.e. mediated via the brain) or reflexive (i.e. via reflex pathways in the spinal cord), and before any other discernible physiological changes have occurred. In the male the principal effect is erection of the penis." (2, p. 53)

(B) Bors and Comarr: "Erection results from stimuli which are either psychogenic or reflexogenic. In last analysis psychogenic stimulation evokes also in reflex function, the difference being that the reflex function occurs in the brain rather than in the cord; thus stimuli of various types, visual, auditory, and olfactory perception, recognition, and recall (memory) reach supraspinal or central areas. Reflexogenic erection results from efferent and efferent stimulation. Exteroceptive stimuli must not be nociceptive, but are usually tactile and applied to the external genitalia. . . . Exteroceptive stimulation, i.e., stimulation of unknown origin causes "spontaneous" erection. Such unknown stimuli may originate in hollow viscera, bladder or rectum, of individuals with an intact or damaged neuraxis."

(C) Chapelle et al.: "It should be stressed that we had to distinguish between 3 types of erection: (1) The reflex erection, i.e. the erectile response to strictly subclinical cutaneous and/or mucous stimulation which . . . is only present if the corpus medullaris is included in the subclinical syndrome. (2) The psychogenic erection, which is independent of subclinical stimulation. It occurs when the sexual appetite is exacerbated and results only in swelling and lengthening of the penis, without rigidity and therefore without the possibility of intromission. (3) The mixed erection, which combines both of the preceding mechanisms." (91, p. 793)

(D) de Groat and Booth: "Penile erection is primarily an involuntary phenomenon that can be elicited by at least two distinct central neural mechanisms: psychogenic or reflexogenic. Psychogenic erections are initiated by supraspinal centres in response to auditory, visual, olfactory and imaginative stimuli. Reflexogenic erections are elicited by efferent stimulation of the genital areas." (23, p. 467)

(E) Karacan and Moore: "Erection may occur from either of two sources: (1) psychic stimulation (erotic fantasies, erotic stimulation of the five senses) mediated via the thoracolumbar erection center (located in L2, L3, and L4); and (2) stimulation of either the genitals themselves (exteroceptive) or of the bowel and bladder (interoceptive) mediated via the pudendal nerve and sacroccocygeal erection center (S3 and S4). Both of these systems reach the pelvic and penile region via the "nerve erigentes" [pelvic nerves]." (43, p. 134)

(F) Kennedy and Over: "Reflexogenic penile erection resulting from tactile stimulation of the genitals, prostate, or urethra occurs through activation of a reflex arc at sacral segments 2 to 4 (S2-S4), whereas psychogenic erections (induced, for example, by erotic thoughts and fantasies) are initiated by input from the higher central nervous system to thoracolumbar segments T12 to L1. . . . Because these two systems do not function independently, the effects that injury to the spinal cord has on the subsequent capacity of men to achieve erection can be difficult to predict." (46, pp. 15-16)

(G) Krane et al.: "Penile erections are elicited by local sensory stimulation of the genital organs (reflexogenic erections) and by central psychogenic stimuli received by or generated within the brain. Reflexogenic erections are mediated by a spinal reflex pathway. . . . The pathways for psychogenic erection are less well understood. A variety of stimuli, including visual, gustatory, auditory, imaginative, and tactile, elicit supraspinal erectile responses." (48, p. 1648)

(H) Lundberg: "The effect of . . . spinal cord lesions on sexual function is very much dependent upon the level of the lesion. If there is a lower motor neurone lesion, such as occurs in the destruction of the genital reflex center in the sacral part of the medulla (the conus), reflex erection [is] impossible. . . . Erection may also be provoked by psychically mediated influences, such as visual stimulation. This cerebral erection depends upon a different neural pattern, leaving the cord through the sympathetic outflow of the lower thoracic segments." (52, pp. 131-132)

(I) Meisel and Sachs: "Because the lumbosacral system also carries most of the penile afferece, erection in response to penile stimulation (reflexive erection) will be most affected by damage to the lower spinal cord or the nerves that project there. . . . [Other research] has implicated the sympathetic polyneurone pathway in mediating [psychogenic] erections resulting from sexual stimuli received via the cranial nerves or generated within the brain as memories, fantasies, or dreams." (59, pp. 25-26)

(J) Steers et al.: "Parasympathetic preganglionic input . . . is thought to be primarily responsible for tactile-induced penile erections (reflexogenic) as well as visual-, olfactory-, and imaginative-induced erections (psychogenic). Sympathetic preganglionic input carried in the hypogastric nerves to the pelvic plexus has been implicated in psychogenic erections." (86, p. R989)

(K) Wagner and Green: "Erection may be provoked from the brain (psychogenic erection) or from spinal centers (reflexogenic erection)."

(88, p. 18)

(L) Weiss: "Penile erection is a reflex phenomenon over which man has little direct voluntary control that is, man cannot will or demand an erection. The forms of stimulation that can reflexly elicit erection have been classified as either 'psychogenic' or 'reflexogenic.' Auditory, visual, olfactory, gustatory, tactile, and imaginative stimuli may arouse the erotic centers of the brain and result in so-called 'psychogenic erections.' Exteroceptive stimulation of the genital regions (for example, light stroking of the glans penis) or vague interoceptive stimuli arising from the bladder or rectum may produce a so-called 'reflexogenic erection.' Interoceptive impulses cause seemingly spontaneous erections, since man is not consciously aware of these visceral signals. . . . [Many] of the erections occurring during sleep . . . are probably psychogenic in origin; the remainder are reflex responses to visceral or tactile impulses." (91, p. 793)

(M) Zillmann: "The combined [thoracolumbar sympathetic] outflow forms . . . what is called the psychogenic sexual center. . . . Psychogenic sexual stimulation — which translates to exposure to distal (i.e., visual and/or auditory) sexual stimuli — is . . . capable of producing both sympathetic excitement and genital tumescence. . . . [T]he alternative, yet primary control of genital tumescence is via the [parasympathetic sacral outflow, known as the reflexogenic sexual center]." (95, p. 176)

The excerpts are listed in alphabetical order by source, and are referred to in the text by associated letter.
mediated by the brain can evoke short-latency responses at the caudal end of the body. Therefore, we should not dismiss a priori the possibility, implied by Weiss (L, Table 1; 91), that some erections are reflexively evoked by stimuli received via the cranial nerves.

We are most likely to consider a stimulus-response cycle as a reflex if its latency is short, as exemplified by the startle reflex or pain-withdrawal reflex. Note that erections evoked by appropriate penile touch are considered to be reflexive irrespective of their response latency, which may be a matter of seconds or many minutes, depending on the species and the context. However, in some species the evocation of erection by nongenital touch or by other modalities may be similarly rapid and, possibly, neurally direct. This possibility will be raised below regarding oral and/or ventral tactile control of erection in cats. In many avian species, visual stimulation from a female may prompt: erection of the male's feathers; the peacock's display is perhaps the best known example. Might not penile erection in response to visual, auditory, or other stimulation of the cranial nerves be similarly reflexive in some mammalian species?

To help summarize this section, Table 2 provides a characterization (some might say caricature) of the conventional view of the stimulus origins and neural mediation of reflexive and psychogenic erections, as well as information about erection in some other contexts. The reflexive/psychogenic distinctions have already been reviewed. The table makes explicit what is implicit in some of the literature on erection: (a) that copulation probably comprises elements of both reflexive and psychogenic stimulation and mediation; (b) that we are ignorant of the mechanisms mediating erection in several other contexts; and (c) that these other contexts may not fit comfortably into the traditional dichotomy.

**Relation of Dichotomy to Classification of Causes of Impotence**

The reflexive-psychogenic distinction for erections is complicated by another distinction commonly applied to failures of erection (i.e., “organic” vs. “psychogenic” impotence). These dichotomies interacted in clinical practice for a time. As used, the term “organic impotence” refers primarily to disorders of erection resulting from pathology in the penis, the peripheral nerves, or the spinal cord. Therefore it was considered that organic factors in impotence could be ruled out by the occurrence of nocturnal penile tumescence (NPT) [i.e., erections occurring during sleep (especially REM sleep)]. Among the untested assumptions implied by this diagnostic procedure were (a) that spinal and peripheral control mechanisms were identical in all erections; (b) that psychogenic inhibitory factors, if present, could not affect NPT; and (c) that if NPT occurred there could not be an organic problem in generating erection. Subsequent research, ably reviewed by Bancroft (2) and Rosen (71,72), has revealed that these assumptions were not warranted, except in very limited form. Furthermore, psychogenic impotence and psychogenic erection are processes that should not be assumed to be opposites of each other. Nonetheless, implicit in the diagnostic question is the idea that mental phenomena are more important in impairing some “types” of erection than others, i.e., that the psychosomatics of erection varies with context.

**Does the Physiological Regulation of Erection Vary With Context?**

The concept that the physiological regulation of erection varies with context has emerged particularly from studies of the roles of the peripheral nerves, the gonadal hormones, and brain neurochemistry. These systems will be considered in order, using a highly selective, “case-study” approach to the literature, to illuminate the issues. The many relevant studies that are omitted here have been summarized elsewhere in more complete reviews (e.g., 2,15,23,59,61).

**Peripheral Nerves**

The contrast between brain and spinal mediation of psychogenic and reflexive erection constitutes the most prominent example of the hypothetical context-dependent control of erection. As already noted, if the two types of erection are defined by their physiological mediation, then the assertion of context-dependent control of erection is circular. Because there is little doubt that the pelvic nerve mediates erection resulting from penile touch, the focus in this section will be on the functions ascribed to the hypogastric nerve. The most widely cited studies supporting a physiological role for the hypogastric nerve in psychogenic erection are those by Root and Bard (70) on cats and by Bors and Comarr (10) on paraplegic men. The landmark status of these studies is well deserved, but can they support the weight of inference that they have accumulated since they were conducted?

Root and Bard (10) used a variety of operations on the spinal cord and autonomic nerves to investigate potential sympathetic pathways mediating erection. Males were tested with estrous females repeatedly (2–39 times) after surgery, perhaps in some cases beginning on the day after surgery and usually continuing for 2–3 wk after surgery. Most cats had at least two operations; some cats had three or four. The key findings were these:

![TABLE 2](image)

**CHARACTERIZATION OF THE CONVENTIONAL VIEW OF THE STIMULUS AND NEURAL MEDIATION OF PENILE ERECTION IN VARIOUS CONTEXTS**

<table>
<thead>
<tr>
<th>Contexts for erection</th>
<th>Stimulus basis</th>
<th>Neural mediation</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Reflexive</td>
<td>somatosensory</td>
<td>pelvic n.; lumbosacral spinal cord</td>
</tr>
<tr>
<td>b. Psychogenic</td>
<td>nonsomatosensory</td>
<td>hypogastric n.; thoracolumbar cord; brain</td>
</tr>
<tr>
<td>c. Copulation</td>
<td>(a) and (b)</td>
<td>(a) and (b)</td>
</tr>
<tr>
<td>d. Dominance; territory</td>
<td>unknown</td>
<td>unknown</td>
</tr>
<tr>
<td>e. Nocturnal tumescence</td>
<td>none?</td>
<td>unknown</td>
</tr>
<tr>
<td>f. “Spontaneous”</td>
<td>none?</td>
<td>unknown</td>
</tr>
</tbody>
</table>

The reflexive and psychogenic contexts are separated from the other contexts because they are commonly treated as exhaustive as well as dichotomous categories.
1. After the lumbosacral parasympathetic pathways were interrupted, generally by removing the sacral and lower lumbar spinal cord, males no longer achieved intromission, but they continued to attempt copulation and display erections. After such surgery a male cat typically "immediately seized the nape of [the female's] neck with his teeth and mounted as best he could, then executed limited but distinctive copulatory movements the extent of which depended on the amount of axial musculature still innervated. On many occasions there could be no doubt that the erection developed as rapidly as in normal animals. Reflex erection could not be produced by manipulating the penis. In every case there was complete anesthesia of the genital area (70, p. 83)."

2. Removal of the abdominal sympathetics alone was without effect on copulation and its associated erections, but rendered the males incapable of ejaculation.

3. Cats that had retained erectile function after initial surgery on the parasympathetic paths stopped having erections after subsequent surgery interrupted the lumbar sympathetic paths by (a) transection of the spinal cord above the L2 level; (b) removal of the abdominal portions of the sympathetic chains; (c) removal of the inferior mesenteric ganglia; and/or (d) cutting the hypogastric nerves.

Root and Bard concluded that their findings of an erectile role for the lumbar-hypogastric pathway supported those of several earlier investigators who had evoked erection in animals by electrical stimulation of the hypogastric nerves. And though Root and Bard ruled out penile tactile stimuli as the basis of erections after cutting the parasympathetic pathways, they drew no conclusion about the alternative effective stimuli. Rather, the inference that psychogenic stimuli were the effective ones was drawn by subsequent readers of their paper.

Two questions bear closer examination here. Did psychogenic stimuli mediate erections effected via the hypogastric nerves after transection of the lower cord? Do the hypogastric nerves normally mediate these responses in cats?

Concerning the first question, we must consider more closely the copulatory sequence of cats, as Root and Bard (70, p. 82) described: "The male cat, unlike the male of many species, rarely if ever shows an erection in the mere presence of an estrous female. None of the 272 complete and 3 incomplete erections we have observed in 25 cats developed until after the male had seized the female by the neck and mounted her. Under these circumstances a full erection usually appeared within a few seconds... and was maintained until the male dismounted" (emphasis added). As this description indicates, male cats may not display erection in response to distal cues from females, a feature generally considered to be a minimal criterion in the definition of psychogenic erection (Table 1). Rather, erection in cats appears to depend upon stimulation of the mouth from biting and/or at least a portion of the ventrum from mounting. The very short latency that intervenes between biting and erection is certainly suggestive of a reflexive reaction (an orogenital reflex?), if indeed the term "reflex" has retained any of its meaning.

Did Root and Bard demonstrate that the hypogastric nerves were mediating erection preoperatively, whatever their postoperative role may have been? The potential plasticity of the nervous system was hardly known at the time Root and Bard conducted their study, so it is not surprising that their research was not designed with such plasticity in mind. A recent experiment on erectile function in rats exemplifies the potential speed of plasticity. Dail et al. (17) stimulated the hypogastric nerve of intact anesthetized rats and observed no increase in cavernous pressure. However, within three days after unilateral section of the pelvic nerve, stimulation of the ipsilateral hypogastric nerve caused a significant increase in cavernous pressure. The mechanisms underlying this plasticity remain to be determined [see Dail et al. (17) for hypotheses], but the experiment raises the possibility that in intact rats with normal pelvic nerve function, the hypogastric nerve plays little or no role in promoting erection.

If the hypogastric nerve plays a major role in erection in intact cats, we would expect a deficit in erectile function after bilateral transection of this nerve or its sources. Only one such cat (no. 3) was reported by Root and Bard (70), all other males having received surgery on the parasympathetic efferents first. Male no. 3 had his abdominal sympathetic chains and inferior mesenteric ganglia removed, and was tested 10 times over the next 31 days. (The timing of the first postoperative test was not reported, although the general methods imply that most cats were first tested a day or two after surgery.) In each test this male displayed full erection. The retention of erectile function in a single male deprived of hypogastric mediation suggests a sufficiency for pelvic nerve mediation, and clearly does not compel attribution of a major physiological role in erection for the hypogastric nerve of cats. After this male's S1-S3 spinal cord was removed he displayed no further erection. The latter finding suggests that at this time the pelvic nerve was the major mediator of erection, but not necessarily of psychogenic erection, since it remains an open question whether the erections that follow in cats after neck bite and partial mounting can properly be classified as psychogenic.

In summary, the research of Root and Bard (70) clearly showed that the hypogastric nerves can mediate erection after destruction of the sacral parasympathetic erectile pathways. Their research, however, did not establish a normal physiological role for the hypogastric pathway in any type of erection, and, specifically, in psychogenic erection.

Now let us examine more closely the other major source of inference concerning the physiological role of the hypogastric nerves in psychogenic erection (i.e., the report by Bors and Comarr (10) of their interview survey of sexual function in 529 men with spinal cord damage). This discussion will be limited to those men (n = 391) who had "complete" spinal cord lesions. As Bors and Comarr noted, the complete/incomplete distinction is rarely confirmed by postmortem inspection or histology, but rather is based on the inability to move or to report sensation from parts of the body innervated by the spinal cord below the level of the lesion. Some somatic and autonomic or other fibers that communicate between the brain and portions of the body in dermatomes below the lesion level may nonetheless be intact in men diagnosed as having "complete" spinal lesions. Supporting this hypothesis is evidence (a) that vibratory stimulation of the penis can lead to orgasm in some "completes," who can even verbally direct the vibrator to the point on the glans that is most effective (87); and (b) that 38% of "complete" quadriplegics report a capacity for orgasm with ejaculation (I).

The salient relevant findings in the Bors and Comarr (10) study were these: 93% of 287 men with complete upper motor neuron lesions (i.e., above the cauda equina) reported that they could still have erections in response to penile touch, but only five of these men reported psychogenic erections. Conversely, men with complete lower motor neuron damage were more variable. Of 104 men with damage restricted to the cord below T9 (to humans the sympathetic trunks exit the spinal cord at T11-L2), 28 men (27%) reported some capacity for psychogenic but not reflexive erection. It is this finding
that erectile function was subject to neural plasticity. One erections had damage above this level. A number of other erectile pathway for intact men, why does erection survive in intact men, why does erection survive in the hypogastric nerve, whereas those who lost psychogenic erections had damage above this level. A number of other possibilities were considered by Bors and Comarr. Since we have no information about the posttraumatic course of sexual function in the men surveyed, it is not unreasonable to suspect that erectile function was subject to neural plasticity. One hypothetical change (10,16), supported by behavioral data from rats (77), is that damage to sympathetic anteriocerebellal fibers in the pudendal nerves causes a chronic hyperinvolution of the penile corpora that cannot be overcome by adequate excitatory stimulation. However, we cannot reject an alternative hypothesis: that the hypogastric nerve plays no role or only a minor role in most intact men, and that through plastic changes after damage to the pelvic effector pathway the hypo-

gastric nerve can come to exert an effective erectile role in a minority of men.

Interpretation of the Bors and Comarr (10) survey is further complicated by doubt about the utility of their interview data in estimating the erectile potential of their subjects. Kennedy and Over (46) formed two groups of eight men with spinal cord injuries of at least one year's standing, with one group reporting and the other denying that they had post-injury erections as a result of erotic thoughts, reading erotic texts, or seeing erotic material. These men were compared to eight intact men for their erectile responses (more than 6 mm increase in penile circumference) to erotic films, spoken texts, and guided fantasies. All the intact men had erections in at least one of the three erection tests. Of the spinally injured men who had reported a capacity for psychogenic erection, only one actually had an erection in the tests, and he only in response to the film. In contrast, four of the men who denied a capacity for psychogenic erection showed large increases in penile diameter during at least one of the tests, and three of the men had marked erections in at least two contexts. These data cast doubt on the value of relying on self-reports of spinally injured men to predict their actual erectile response to erotic stimulation.

In summary, the Root and Bard (70) experiments on cats and the Bors and Comarr (10) survey of men with spinal cord damage do not compel—though they are consistent with—the prevailing view that the hypogastric nerves are the only or the major neural pathway for psychogenic erections. Indeed, there appears to be no unambiguous evidence that warrants rejection of the hypothesis that erections are normally mediated exclusively or primarily by the pelvic nerves. (The neural control of ejaculation is beyond the scope of this paper, but for those species in which the male ejaculates immediately upon insertion, e.g., canids and most ungulates, the neural control of erection may be more directly controlled by the hypogastric nerve, which mediates ejaculation.)

**Gonadal Hormones**

Erection can readily be evoked from restrained supine rats by keeping the sheath retracted to the base of the glans penis, without phasic stimulation of the penis. These "reflexive" erections are under androgenic control, waning after castration and being maintained or restored by testosterone, in particular its dihydrotestosterone metabolite (21,37,51). Estradiol is ineffective in restoring or maintaining erections in this context (58,64).

In contrast to these androgen-dependent responses, erections displayed by rats in two other situations appear to be independent of androgen. The first of these contexts is the urethrogenital reflex, so called because pressure in or rhythmic probing of the urethra evokes erection and associated neurophysiological activity in anesthetized, spinalized males (57). Castration does not inhibit the response, and even females display the neurophysiological correlates of the urethrogenital reflex, though they are obviously incapable of showing the phallic elements.

Finally, the occurrence of erection during copulation may not be dependent on androgen. That is, when the mounting behavior of castrated rats is maintained with high levels of estradiol, then these rats achieve the penile body erections necessary for intromission at a normal rate (58,64). The EMG activity of the penile muscles of estradiol-maintained rats during copulation is at least as strong as that of intact males and testosterone-maintained castrated males (39). Thus, erection in copula, one of the most natural of erectile contexts for rats, appears to be androgen-independent. Whether these erections might be dependent on estrogen (a testosterone metabolite) can not be assessed because long-term castrated males do not mount without testosterone or estrogen treatment.

Evidence from hypogonadal men also suggests that erections in different contexts may have a differential dependence on androgenic hormones. These men have serum testosterone levels (20-50 ng/dl) well below the level that is normal for men (400-700 ng/dl), and they may seek treatment because of sexual dysfunction, usually expressed as disinterest in sex and/or impotence during encounters with sexual partners. In controlled laboratory studies, hypogonadal men tend to show a reduced incidence of erection during sleep and sexual fantasy, but when watching erotic films they experience erection with a latency and intensity that is indistinguishable from normal men (3,12,49). One interpretation of these data has been that visually mediated erection in men is qualitatively different from erection mediated by other modalities and is not androgen dependent (2,3,19,20). This interpretation must be regarded as preliminary. First, except in total darkness, encounters with sexual partners may obviously include moving visual images, some of which would be erotic for eugonadal men. However, these images are apparently ineffective for many hypogonadal men. Perhaps more to the point is a well-controlled experiment on the relative erectogenic quality of different modes of erotic stimulation for normal men (41). The stimulus materials were as closely matched across modalities as possible: the slides were scenes from the erotic film, the texts paralleled the action in the film, and the fantasies were guided to approximate the action depicted in different segments of the film. Film was the most effective mode for promoting erection and fantasy least effective, with spoken text, written text, and slides having intermediate values. The low effectiveness of slides relative to film, and their similarity to written and spoken text, suggests that visual erotica are not particularly erectogenic without movement. Also, the relatively weak effect of fantasy for these eugonadal men suggests that any condition that impairs erectile function would be reflected early on in fantasy-based erection. For hypogonadal men, or for other men near the threshold of erectile function, the erectogenic difference between films and other erotic stim-
uli may be one more of degree than of kind (49), and there may be a trade-off between androgen levels and the stimulus intensity needed to provoke erection.

A number of other caveats should be noted about most of the research on hypogonadal men. First, these men had very low levels of androgen but were not agonadal. Therefore, to the extent that the men responded to erotic stimuli, the erections could be said to be less sensitive to low androgen levels but could not be said to be independent of androgen. Then, as remarked elsewhere (59), there may be a population of hypogonadal men whose sexual function is relatively normal, and who therefore do not seek medical treatment. The hypothetical existence of such cases would imply that erection during sexual interactions with partners, rather than only in response to erotic films, may be relatively insensitive to androgen titer. Finally, there seems unfortunately to be no evidence on whether the penis of hypogonadal men is less responsive to the erectileogenic effect of appropriate touch. The only relevant data on this question are ambiguous. Burris et al. (11) determined that a sample of hypogonadal men had significantly greater vibrotactile sensitivity than eugonadal men. However, because of their specific pathology and treatment history, these men had never gone through puberty. A control group of previously hypogonadal men who had long since been virilized by testosterone therapy (and whose testosterone levels were in the normal range) were the least sensitive group tested. Whether the threshold differences were attributable to changes in penile morphology (e.g., skin thickness), receptor density or sensitivity, central processing, or other factors could not be evaluated. None of the men experienced erection during the tests, either because the touch was not intrinsically erotic or because it was not erotic in context. Therefore, the relation between touch threshold and erectile response to touch remains unclear.

A more general question about the relation between testosterone and erectile function in humans arises from a consideration of erections displayed during early stages of development. Except for the first few months of life when serum testosterone exceeds 200 ng/dl, testosterone in prepubertal boys is below the limits of detectability (65). Yet, from fetal life on, young boys have erections during sleep (44,45), and from early infancy they appear to derive pleasure and erections from masturbation (47). Furthermore, young boys are reportedly capable of multiple orgasms within a short span of time (47), something that few men are capable of (28,47). [The questionable origin of the Kinsey data on multiple orgasms in boys (47), collected at least in part by pedophiles (63), makes these data unusually suspect.] If confirmed, these data on prepubertal sexual responsiveness imply that the higher titers of testosterone and/or neural changes associated with puberty act to increase, rather than reduce, the postorgasmic refractory period. More generally, we might consider the possibility that some behavior patterns change from being hormone-independent before puberty to hormone-dependent after puberty. Clearly many questions remain unanswered concerning the relation of androgen to erectile function in humans and how that relation may change as a function of age and context.

**Brain Neurochemistry**

Pharmacological studies may provide some of the strongest evidence for context-dependent differences in the physiological regulation of erection. Research from two laboratories, those of E. M. Hull (40) and of M. T. Zarrindast (94), may serve well to illustrate this point. In the experiments to be reviewed, drugs of greater or lesser neurochemical specificity were injected systemically or via cannula into specific brain structures. (See also Refs. 8,23,59.)

Hull and Zarrindast are among the several investigators who have tested erection in rats after treatment with various drugs believed to act specifically or preferentially on D1 or D2 dopamine receptors. Table 3 was compiled from data presented or summarized in papers from each laboratory that synthesized their findings (40,94), as well as those from a study by Pomerantz (69) on rhesus monkeys. The contrasts between Hull and Zarrindast are striking. The difference is most evident for the one drug that both have used, SCH23390, a D1 antagonist, found by Hull to depress erections and by Zarrindast to increase erections. For D2 agonists, Hull reported increased erections after treatment with THP, whereas Zarrindast noted a decrease in erections after injection of SKF38393. The pattern of disagreement is maintained when D2 agonists and antagonists are considered.

In view of the differences in drug effects, it is not surprising that Hull and Zarrindast came to very different conclusions about the physiological roles of dopamine receptors in mediating erection. Their condensed hypotheses, and possible origins of the differences between them, are also summarized in Table 3. (a) Both laboratories are using rats, and it is unlikely that strain differences could lead to such dramatically opposite results. As indicated in Table 3, in the only clear tests with another species, Pomerantz (69) reported that quinelorane, a D2 agonist, increased erections in male rhesus monkeys exposed to receptive females behind a screen. These data are consistent with those of Zarrindast as is the recent finding that quinelorane enhances erections in solitary rats (27). However, since the rhesus males masturbated during exposure to the females, the extent to which their erections resulted from distal or proximal stimulation is unclear; (b) The route of drug treatment is very different, being into the medial preoptic area (MPOA) in much of Hull's research and systemic in Zarrindast's. There has been a hint that dopaminergic drugs injected into the hypothalamic paraventricular nucleus may have effects different from those after injection into the MPOA (29). Nonetheless, in Hull's laboratory as in others, consonant effects have generally been found with systemic and MPOA drug treatments, albeit at very different dosages (78). For example, systemic injection of quinelorane at 10 μg/kg significantly reduced reflexive erections (9), as did MPOA infusion of 3 μg quinelorane (5). Smaller doses (0.25 ng/kg SC, 0.1 μg into MPOA) had no reliable effect on number of erections (5,9). (c) More compelling as a cause for the different outcomes in the two research programs is the very different context in which erection was evoked in the two laboratories. The data from Hull that are summarized in Table 3 were based upon reflexive erection tests of the type described above (sphincter unanesthetized male, penile sheath retracted), whereas those of Zarrindast were derived from so-called spontaneous erection tests of isolated males. (It should be noted that the baseline rate of spontaneous erection for untreated rats approximates zero. Therefore, in Zarrindast's research, inhibitory actions of drugs were tested in males that had been pretreated with erecgetic drugs: apomorphine, bromocriptine, or quinelorane. Each of the drugs that promoted erections did so even in rats receiving no pretreatment.)

Associated with the difference between reflexive and spontaneous erections are very different maximum rates of erection: up to 25 erections in 15-min reflexive erection tests, compared with about 4 erections in 60-min spontaneous erection
TABLE 3
CONTRASTING EFFECTS OF DOPAMINERGIC DRUGS ON ERECTION

<table>
<thead>
<tr>
<th>Investigators</th>
<th>Hull et al. (40)</th>
<th>Zarrindast et al. (94)*</th>
<th>Pomerantz (69)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observed effects (↑ or ↓) on number of erections</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D1 agonists</td>
<td>THP ↑</td>
<td>SKF38393 ↓</td>
<td>quinelorane ↑</td>
</tr>
<tr>
<td>D2 agonists</td>
<td>quinelorane ↓</td>
<td>quinpirole ↑; bromocriptine ↑</td>
<td>quinelorane ↑</td>
</tr>
<tr>
<td>D1 antagonists</td>
<td>SCH23390 ↓</td>
<td>SCH23390 ↑</td>
<td>sulpiride ↓</td>
</tr>
<tr>
<td>D2 antagonists</td>
<td>raclopride ↑ (with 10 µg apomorphine.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interpretation of effects</td>
<td>Low dopamine levels stimulate erection via D1 receptors; high dopamine levels inhibit erection via D2 receptors.</td>
<td>D2 receptor activation stimulates erection; D1 activation inhibits erection.</td>
<td>D2 receptor activation stimulates erection.</td>
</tr>
<tr>
<td>Potential sources of difference</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Species</td>
<td>rat</td>
<td>rat</td>
<td>rhesus monkey</td>
</tr>
<tr>
<td>Drug route</td>
<td>MPOA infusion</td>
<td>s.c., i.p.</td>
<td>s.c.</td>
</tr>
<tr>
<td>Context</td>
<td>supine restraint, sheath retracted</td>
<td>isolated, freely moving</td>
<td>screened from receptive female</td>
</tr>
<tr>
<td>Approx. max. no. of erections/test duration; time of test</td>
<td>25/15-min; dark phase</td>
<td>4/60-min; light phase</td>
<td>30% &gt; baseline/15-min; light phase</td>
</tr>
</tbody>
</table>

*Inhibitory effects reported were based on pretreatment with apomorphine, bromocriptine, or quinpirole, because the baseline rate of erections in untreated rats approximated zero. Excitatory effects shown were reliable even in rats not pretreated with other drugs.

tests. The faster rate of erection in reflexive erection tests is much closer to that occurring during copulation, but it remains to be seen whether reflexive or spontaneous erection tests better predict a drug's effect on erection in copula, especially since the different erectile contexts probably differ also in their dependence on various neural systems. Although it is not possible yet to specify the reasons for the very different results obtained in different laboratories using similar drugs, the different contexts in which erection was tested are likely to be important among the reasons.

SUMMARY

The evidence reviewed in this section strongly suggests that the relative dependence of erection on particular hormones, peripheral nerves, brain systems, and neurochemicals varies according to the erectile context, but the data do not yet compel that conclusion. Further progress on this question will depend in part on conducting more experiments in which males are tested in more than one erectile context.

Reflexogenic-Psychogenic Distinction Reconsidered

Despite some gaps in the existing evidence and a clear need for more evidence, the studies just reviewed suggest that the physiological regulation of erection does vary in different contexts, including those comprising the distinction between reflexive and psychogenic erection. It also seems clear that this traditional dichotomy does not serve experimental and analytic purposes adequately, appearing to impede attention to and analysis of the erectile responses that occur, the stimulus complexes that contribute to them, and the physiological mechanisms that mediate them. Ultimately it may be desirable to discard these terms and develop a different classification system, one that is not unique to erectile functions among behaviors. [Rarely are other responses termed psychogenic (e.g., Refs. 32, 82). However, a parallel to the control of erection may be seen in the question of whether "conscious" and "reflex" micturition have different sensory inputs, and therefore different neural regulation (24, 55).] In the meantime it may be useful at least to be clearer about what we mean when we use certain terms; to assume that there already exists a consensus of mutual understanding is inconsistent with the evidence from the definitions in Table 1. The task is complicated by another goal, one that does seem to have an implicit consensus, which is that terms should be broadly translatable among species to facilitate comparative physiological research.

Reflexive Erection

I take the term "reflexive erection" to refer to unconditioned responses in the traditional senses: characteristic of the species, evoked by relatively specific extrinsic stimuli or stimulus patterns, independent of previous experience with those stimuli in a sexual context, and typically expressed by all males (at least breeding males) during particular seasons or stages of development. The traditional use of reflexive erection to refer only to penile, perineal, or even somesthetic stimulation strikes me as excessively restrictive, and it inhibits analysis of the commonalities that might exist between modalities (e.g., touch and vision) and within the same modality by different stimulus patterns or at different "erogenous" areas (e.g., touch of penis, lips, and ears). As we have seen, the observations of Root and Bard (70) suggest that erection in cats may be reflexive in response to oral stimulation from biting and/or ventral
stimulation from mounting. Similarly reflexive may be the erection and accompanying self-enurination that forms part of the sociosexual behavior of goats, possibly in response to vomeronasal stimulation (38). [Stimulation of the cat’s vomeronasal organ during biting might also contribute to erection (M. J. Baum, personal communication, 1994). The potential inclusion of other sensory modalities and of somatosensory stimulation of other areas would also avoid the circular definition of reflexive erections as those that can be expressed in males after spinal transection rostral to the pudendal-pelvic neural circuit.

**Psychogenic Erection**

If the term “psychogenic erection” is to be retained, then it may be useful to distinguish weak and strong senses of the term (Table 4). The weak—and commonly used—sense would include erection resulting from any extrinsic nonsomatic, whether visual, auditory, or chemosensory. (Some definitions have included nonperineal somatosensory stimulation. See Table 1.) The weak sense of psychogenic erection should not be assumed to be mutually exclusive with reflexive erection because, as I have just noted, some of these stimuli may prove upon analysis to be reflexively erectogenic. It is tempting to speculate that the erectogenic power of diverse visual erotica for men is based on a feature-extraction process, one that derives consistent stimulus patterns that may be as closely (reflexively?) linked to erection as is penile stimulation. Analysis of erectogenic stimuli in this weak sense is important for discovering effective stimuli in effective modalities, in part because these stimuli and modalities may prove to be the most important ones for psychogenic erections in the strong sense. In addition, it is important to consider the context-dependence of the response to potentially erotic stimuli, as Bancroft (2) has emphasized. Such stimulation at the wrong time, or by an individual who can not be viewed as a potential sexual partner, may be counter-erotic. Conversely, stimuli that would not normally be erotic can be erectogenic in context, especially after becoming associated with sex, but that type of association would be considered psychogenic in the strong sense.

The strong sense of psychogenic erection would be limited to memory and fantasy. By fantasy I mean “scripts” recognized by the agent never to have occurred, and obviously only humans can report these. Memories include (a) free recall of scripts thought by the agent to represent past experiences and (b) conditioned responses, in which the effective extrinsic stimuli acquire their erectogenic value only after being associated with sexual experiences.

In order to investigate experimentally whether animals exhibit psychogenic erection in the strong sense, one might have to rely on classical and operant conditioning techniques, and these have apparently not been systematically used. W. H. Gantt, one of the first American students of Pavlov, did not specifically attempt to condition penile erection in dogs. However, in a series of studies on “experimental neurosis” he placed dogs in severe conflict situations, in which some dogs reliably displayed prominent erection (34). To collect semen from horses for purposes of artificial insemination, stallions are commonly trained to mount dummies and ejaculate into artificial vaginas (89). The stallions achieve erection prior to mounting, just as they do when mating with mares. Since the dummies would not evoke erection prior to experience with them, these erections would be considered to be conditioned responses, but I am unaware of systematic studies of this response. When male rats are rewarded with copulation, they readily learn to press a bar or to exhibit a place preference for the copulation site (30,59), but it remains to be determined whether males have erections in these contexts.

**TABLE 4**

<table>
<thead>
<tr>
<th>Type of Stimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reflexive erection</td>
</tr>
<tr>
<td>Psychogenic erection</td>
</tr>
<tr>
<td>weak sense</td>
</tr>
<tr>
<td>strong sense</td>
</tr>
<tr>
<td>somesthetic, especially genital</td>
</tr>
<tr>
<td>nonsomesthetic</td>
</tr>
<tr>
<td>memory, fantasy</td>
</tr>
</tbody>
</table>

Classical and operant techniques have been used to induce or enhance men’s tumescence in response to neutral or weak sexual stimuli. For example, using no sexual stimuli, Rosen et al. (73) instructed men to try to develop erections. Some men were given visual feedback via lights that reflected increases in penile diameter, and they had greater success in gaining tumescence than did men who received no extrinsic feedback. This procedure is rather a hybrid of operant and classical conditioning techniques, and in any event does not address the mediating cognitive process.

No investigations into psychogenic erection in the strong sense have addressed the question of the intrinsic stimuli that mediate these erections. The relative erectogenic potential of memories or fantasies may depend on the degree to which an individual can simulate the effective extrinsic stimuli. This point is most easily seen with respect to human memories or fantasies, in which the erotic effect depends strongly on the extent to which the extrinsic stimulation can be approximated by intrinsic processes (e.g., the ability to visualize (or otherwise “sense”) the extrinsic erotic stimuli (84)).

The perceptual and cognitive processes of psychogenic (“cognigenic?”) erection in the strong sense need not be available to consciousness, as there already is evidence of a role for nonconscious processes in other behavioral domains. For example, there is increasing evidence that humans can act upon visual stimuli that they cannot report seeing. The process is called “blindsight” because it was first described in patients with damage to the visual cortex who reported that they could see nothing in circumscribed portions of their visual fields, but who could nonetheless make reliable sensorimotor judgments about visual stimuli to which they were “blind” (90). An analogous process occurs in normal individuals (56), and possibly in other sensory modalities (“nombouch”? “deafhearing”?).

Recent data support classical descriptions of a functional vomeronasal system in humans (62), and at least in rodents the vomeronasal organ, which is sensitive to sex-relevant pheromones, may have direct projections to the hypothalamus (50). Such a system, like the retinal-suprachiasmatic nucleus system, may be inaccessible to consciousness. Less speculative is the evidence for a process in humans called “implicit memory,” in which previous experiences that cannot be recalled nonetheless affect the performance of current tasks (79). It should be noted also that if such processes contribute to psychogenic erection in the strong sense, then that presupposes that they also can contribute to psychogenic erection in the weak sense.
The foregoing discussion has considered problems with the reflexive-psychogenic erection dichotomy, as well as with the more general reflex–nonreflex dichotomy. The latter distinction may be more properly viewed as a continuum reflecting number of synapses, complexity of neural circuitry, probability of stimulus–response association, and so on. In the long run it would appear to be counterproductive to maintain a typology of erection founded on two interacting untenable dichotomies, and alternative taxonomies of erection should be sought. One such effort in that direction is assayed in Table 5.

According to this scheme, stimuli are classified as contact (somesthetic) or noncontact (nonsomesthetic), and their action in evoking erection is placed on a continuum of reflexivity, depending on the criteria cited above for classifying stimulus–response relations. The hypothetical placements of targets and modalities on the continuum is estimated from studies (e.g., 41) reviewed in this manuscript. These placements are probably more subject to individual differences than are contact stimuli, but are obviously amenable to investigation. Erectile contexts, including not just sex but also agonistic encounters, sleep, and others, could be considered as orthogonal to the two dimensions depicted in Table 5.

**NPT and Other "Spontaneous" Erections**

Penile erection is displayed in many other contexts for which the effective extrinsic and intrinsic stimuli have escaped discovery or analysis. Not uncommonly, these responses are called "spontaneous erections," a term whose usefulness may be intermediate between "spontaneous generation" and "spontaneous combustion." The very use of the term tends to inhibit the search for its stimulus origins.

Men commonly experience erections with no conscious awareness of any erotic stimuli or thoughts. That does not mean that there are no active extrinsic or intrinsic stimuli, and it is tempting to consider whether processes like blindsight and implicit memory may be at work in this context.

Erections occurring during REM sleep are far more clearly attributable to intrinsic factors. REM sleep occurs in virtually every vertebrate species and, in humans at least, it occurs throughout the life span, beginning with fetal life. Such continuity should encourage developmental and comparative studies. However, systematic study of erections associated with sleep are lacking except in human adults. Long-tailed shrews (66), opossums [cited in (33)] and hyenas (S. E. Glickman, personal communication, 1993) are reported to display these hypothetical roles of full bladders in promoting erection. Furthermore, it is not readily reconciled with the rarity of erections associated with full bladders during waking hours. (Nor is there any evidence that men with erectile dysfunction avoid emptying their bladders prior to having sex.) More likely, these so-called "bladder erections" are attributable to REM sleep occurring just before waking. Nonetheless, it remains possible that bladder pressure plays a contributory role. Unfortunately there appears to be no systematic recording of NPT and waking erections in men who have bladder-draining catheters, or men whose bladders have been surgically removed. Such data could begin to test the hypothetical role of full bladders in promoting erection.

Erections induced by drugs administered systemically or into the central nervous system are also occasionally referred to as "spontaneous" (e.g., 4), but they are more appropriately described as "drug-induced" (6) to avoid presupposition about the causes. Pending analysis, one can not know whether the drugs are acting only on the efferent paths of erection, or whether they might also be simulating extrinsic and intrinsic erotic stimulation. [Analysis of these responses may also have been hindered by inadequate description. For example, the behavioral response of rats to apomorphine, does not—as sometimes alleged—closely resemble intravestibular or ejaculatory patterns, nor has there been evidence that ejaculation accompanies these erections (6,27,76).]

Some of the problems arising in interpreting drug-induced erection may apply also with electronic brain stimulation (14,53,54,67). Even if only efferent neural paths are stimulated, the potential erogenic effect of reafferent cannot be discounted, as it can be in studies of peripheral nerve stimulation by transecting the afferent nerves or the proximal portion of the stimulated efferent nerves (57,86). And as with any research, species differences can complicate interpretation. For example, as part of their territorial or agonistic behavior, several primate species develop erections (26,54). In dominance displays, male squirrel monkeys thrust their hips and erect penises toward the face of subordinates (54). Therefore, when brain stimulation causes erection, it is appropriate to inquire whether erection resulted from stimulation of sexual, agonistic, or common efferent pathways (53,54).

**CONCLUDING REMARK**

It appears that the question of whether the physiological regulation of erection varies with context can hardly be considered without confronting the dichotomous typology of erection, especially because the typology is based in large part upon the assumption of differential regulation. Great strides have been made in understanding the psychobiology of erec-
tion, despite apparent problems with the typology. However, as erection is studied in more contexts and more species, the search for principles of erectile function can only be helped by a better map of the relations among the contexts. These maps are unlikely to be identical for all species, so comparisons between species or contexts should not be based on the assumption of isomorphic processes.

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