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ORIGINAL ARTICLE

Genital and heart rate response to erotic stimulation in men with and without premature ejaculation

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This study compared genital and penile response patterns in men with and without premature ejaculation (PE) so as to identify the potential anomalous psychosomatic relationships among men with PE. Genital and heart rate response profiles of 25 men with PE were compared with those of 13 age-matched sexually functional counterparts during visual sexual stimulation presented in combination with vibrotactile penile stimulation. Although no differences were found between men with PE and controls on maximum penile circumference change, overall penile response was significantly lower in the PE group and PE men who ejaculated during the session exhibited shorter latencies to maximum circumference change. Furthermore, significant differences were found between groups in patterns of heart rate. These findings indicate differences in physiological responses between men with PE and sexually functional counterparts during erectile tumescence and progression toward ejaculation. Such differences might be explained by 'premature' sympathetic activation during the sexual response cycle in men with PE, thereby diminishing parasympathetically controlled penile response and triggering sympathetically mediated seminal emission prematurely.
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Introduction

Ejaculatory response is the efferent (motor) component of a spinal reflex that typically begins with sensory stimulation to the glans penis.[1] This reflex is modulated by centrally mediated cognitive, affective and behavioral processes; therefore, it is not surprising that most men report some degree of control over its timing.[2–4] Men who ejaculate 'prematurely'—before or shortly after vaginal intromission—may do so in part because they reach high levels of sexual arousal very quickly or because their threshold to ejaculation is extraordinarily low.[5–6]

If short ejaculatory latencies result from high levels of sexual arousal, then we might also expect short *erectile* latencies in these men—that is, the interval of time required to achieve an erection beginning from a flaccid penile state. Yet existing evidence does not necessarily support this expectation—the few studies investigating this possibility have found no differences between premature ejaculation (PE) and control groups on various erectile parameters, including latency to maximum erection.[7–9] On the other hand, and somewhat counterintuitive, even though PE men ejaculate rapidly, they might actually exhibit weaker erectile responses compared with functional counterparts. Specifically, compared with functional counterparts, men with PE report higher negative affect, greater difficulty getting an erection, and weaker overall erections—despite greater self-reported proximity to ejaculation—in response to erotic stimuli.[3,10,11]

Such affective states and self-perceptions of erectile response in these men suggest potential disruption of the typical autonomic processes involved in erection and ejaculation. Specifically, parasympathetic action early in the sexual response cycle is typically necessary to initiate and sustain erection, with concomitant or subsequent sympathetic activation responsible in part for mediating ejaculation. In men with PE, this typical progression may be disrupted, such that sympathetic activation prevails earlier in the response cycle (for example,

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due to anxiety or negative affect), which in turn might interfere with parasympathetically controlled erectile tumescence and trigger ejaculation prematurely, perhaps even before the man reaches maximum genital or subjective arousal.

The above scenario underscores the two overlapping genital phases that occur during normal sexual response in men. The first, the excitement or erectile tumescence phase, extends from the beginning of sexual arousal/stimulation to the point of achieving an erection sufficient for intromission. The second, the plateau or ejaculatory latency phase, begins with intromission and terminates with ejaculation. Although erectile and latency phases have traditionally been associated with parasympathetic and sympathetic activation, respectively, both systems are undoubtedly activated simultaneously.[4] Perhaps less understood is the manner in which these systems might interact with one another or with cholinergic or non-adrenergic non-cholinergic (NANC) systems.[12,13]

If, indeed, sympathetic and parasympathetic activation differentiates PE from non-PE men,[14] these differences should be apparent in autonomically sensitive genital and non-genital physiological response patterns manifested during sexual arousal. That is, both penile response and sympathetically mediated responses such as elevated electrodermal response and heart rate might differentiate PE men from controls, particularly during early stages of arousal. Although few studies have tested such hypotheses by carefully comparing various autonomic measures across PE and non-PE men at different stages of arousal,[7] indirect support is garnered by the finding that during papaverine-induced erections, PE men showed less suppression of sympathetically mediated skin potentials than controls,[15] suggesting greater sympathetic activation than normal during the earlier phases of sexual response.

Heart rate, under both sympathetic and parasympathetic control, is also known to change during sexual arousal and therefore represents a further tool for the investigation of differences in autonomic activation in PE and functional men. Specifically, the parasympathetic system maintains heart rate at resting tonic levels, whereas the sympathetic system, through the secretion of epinephrine, increases the heart rate. During sexual activity, heart rate may increase by as much as 100% in males, with considerable variation existing across individuals.[16–20] In studies where physical exertion is minimized during arousal, such as when men are exposed to visual sexual stimulation while seated in a laboratory, heart rate shows variable and complex patterns. For example, several studies indicate decreasing heart rate during arousal,[21,22] perhaps due in part to increased attentiveness to sexual stimuli.[23,24] Others suggest a biphasic response—initial deceleration followed by acceleration—or a diverging response, with some men showing accelerated heart rate and variable penile tumescence and others showing a more stable heart rate with smooth tumescence.[17,25,26] Whatever the pattern, few studies have attempted to utilize autonomically sensitive measures such as heart rate to differentiate sexually functional from dysfunctional men, and, to our knowledge, none has done so in an investigation of men with PE.

The present study was designed to identify potential differences in psychophysiological response patterns between PE men and sexually functional counterparts.[5,14,27] Such relationships might predict weaker erectile response (lower maximum and/or longer latency to maximum) and higher heart rate in PE men during the early stages of the sexual response cycle due to early sympathetic activation. To this end, we compared genital and heart rate patterns across PE and control men during sexual arousal in response to visual sexual stimulation and penile vibratory stimulation in the laboratory. The incorporation of penile vibrotactile stimulation permitted a high degree of control over the stimulus parameters likely to elicit ejaculation while ensuring that the heart rate would not be confounded by significant physical activity, as participants sat quietly in the laboratory during testing.[2]

Participants and methods

Participants

A total of 38 Dutch men participated, out of which 25 men with PE and 13 healthy men having no sexual dysfunctions (and serving as a comparison group) were recruited through a newspaper article on *PE* in a major Dutch daily newspaper. The article indicated that men—whether they had the condition or not—interested in participating in a study related to this problem should contact the investigators.

Participants were 18 years or older, in a stable heterosexual relationship, had no current depression as determined by a standardized psychological assessment instrument[28] or substance abuse (alcohol or drugs), and were free of diseases, medication use and surgical procedures that might impact sexual function as determined through a semi-structured interview. Men with a significant history of or ongoing erectile dysfunction were excluded.

PE men reported a minimum 6-month history of PE. Their condition was assessed through a structured diagnostic interview using Diagnostic and Statistical Manual-IV (DSM-IV) criteria by an experienced sexologist. Consistent with this clinical diagnosis, these men reported an estimated intravaginal ejaculatory latency time under 90 s (mdn = 0.9 min, mean = 1.1 min) and a lack of ejaculatory control ('1' or '2' on a 7-point scale, where 1 = none at all). These criteria, known to reliably discriminate premature ejaculators from sexually functional

men,[29–32] eliminated eight self-identified 'PE' men from the study, resulting in the final 25 men with PE included in this study.

Men in the comparison group underwent a similar intake procedure and responded to relevant items on the retrospective questionnaire assessing sexual functioning. These men reported an estimated intravaginal ejaculatory latency time of 3–12 min (mdn = 7.5) and moderate to strong control over the timing of their ejaculation (4–7, where 1 = none at all)—values consistent with those reported in a recent study on a US population.[33]

Response measures

Heart rate was monitored with three 3M Red Dot electrodes; the signal was amplified, averaged over 60-s periods, and stored for later analysis using an ECG Siemans SC9000 recorder (Berlin, Germany). Penile response was measured using the Rigi-scan Plus (Osborn Medical Systems, Augusta, GA, USA). This device consists of two rings, one placed at the base of the penis and the other just posterior to the glans. These rings enable continuous recording, sampled and averaged at 30-s intervals, of circumferential (base and tip) changes during the session. Only base circumferences are reported in this study, although base and tip measures were well correlated ($r = 0.79$, $P < 0.001$).

Stimulus conditions

Each participant viewed a 9-min erotic videotape presented in combination with vibrotactile stimulation to the penis,[9,34] delivered in 15-s pulses at 15-s intervals with a mini-vibrator secured by an erectiometer[35] and attached to the underside of the penis. The vibrator generates vertical displacement up to 1.5 mm, with output frequency ranging from 10 to 60 Hz depending on the input voltage. The erotic videotape, an edited clip from a commercially available production, portrayed a variety of sexual activities (for example, foreplay, intercourse, fellatio, cunnilingus) and progressed from foreplay to sexual acts to climax.

Procedure

Potential participants were screened and interviewed by telephone by a trained sexologist. The purpose of the study was explained, and eligibility and willingness to provide written consent were determined. Participants were then sent through the mail the informed consent form, as approved by the Medical Ethics Committee of Erasmus University Medical Center, a description of the study, and a sexual history and functioning questionnaire.

During the psychophysiological testing session (see Rowland *et al.*[2] for details), a male experimenter positioned the ECG electrodes and then explained the placement of the penile devices to the participant who was then left alone to position them. Proper placement of the devices was verified by the experimenter. Each stimulus was preceded with 5 min of baseline and a set of instructions that ensured a permissive, but non-demanding atmosphere for sexual arousal and to 'normalize' ejaculation/orgasm, were it to occur.

Preliminary analyses and overall analytical strategy

These data represent a subset of measures drawn from a double-blind, placebo-controlled crossover 'at-home' investigation of the effects of clomipramine on ejaculatory response in PE and sexually functional men. Data on ejaculatory latencies from 25 mg clomipramine or placebo taken 4–6 h before intercourse have been reported elsewhere.[36] Heart rate and genital response (under the placebo condition only) included in this analysis have not been reported previously. Preliminary analysis indicated no *order* effects between placebo and drug conditions ($F[1,36] = 0.23$, $P = 0.63$).

A number of parameters arising from the experimental situation could affect the group profiles of genital and heart rate responses. First, several participants showed little or no genital response (< 5 mm circumference change) during stimulus sessions. Data from such subjects would not contribute meaningful information toward establishing a genital response profile (for example, latency to maximum). Therefore, such participants (one control (8%) and four PE (16%)) were removed from the genital analysis. All participants, however, were included in the heart rate analysis, as preliminary analysis using only those men showing penile response indicated no major difference in the data patterns relative to an analysis that included all participants.

Second, during the stimulus session, a number of men ejaculated and thereby terminated their session. For example, 14 of the 25 PE men and 1 of the 13 control men ejaculated during the session. For these analyses, we generated separate profiles for PE ejaculators and PE non-ejaculators and, when possible, further separated PE ejaculators into those with latencies under 3 min in the lab session from those over 3 min (see Table 3 for the numbers). (PE men were classified through clinical interview and verified by having estimated ejaculatory latencies during coitus of less than 150 s. However, during psychophysiological testing, ejaculation may have occurred any time within the 10 min session, a response pattern typical of men with PE and one that differentiates them from men without PE.) The 3-min criterion was chosen for expediency: PE ejaculators with latencies over 3 min enabled profiles of adequate length to allow several meaningful comparisons with controls and PE non-ejaculators.

The responses of PE ejaculators with latencies less than 3 min were profiled separately, but the low number of data points made the statistical comparisons impossible. In addition, PE men who ejaculated in more than 3 min did so at different times during the session (as early as 4 min, but also as late as 8–9 min into the session). However, their *group* profile had to end with the first ejaculation as participants began to drop out at that time. To capture a sense of how these subjects responded during the remainder of their pre-ejaculatory interval, the average of each subject's remaining pre-ejaculatory data points was calculated and included as a single additional (successive) point for analysis.

ANOVA was used to identify significant differences between PE and control men on penile response and on heart rate. For ease of understanding, results are presented in three sections: (1) comparison of the groups on various self-report measures of sexual functioning; (2) penile response; and (3) heart rate response.

Results

Comparison of PE and control groups on self-report measures of sexual function

Information about demographics, sexual function, and sexual activity of PE and control groups appears in Table 1. On retrospective measures related to sexual interest and erectile functioning, the groups showed no differences.

Penile response

The following parameters of penile response were compared across PE and control groups: maximum change in circumference from baseline, latency to maximum circumference and average penile response per minute as determined by area under the curve (Table 2).

Maximum change and latency to maximum. Figure 1 shows the penile response of responders in PE and control groups. Overall maximum circumference (change from baseline) did not differ across groups, but latency to maximum circumference was significantly shorter for PE men than controls ($F[1,31] = 6.29$; $P = 0.017$). As some men with PE ejaculated early in the session (which then defined their maximum point of tumescence), we further explored this measure by comparing PE ejaculators with PE non-ejaculators and controls (separately). PE ejaculators had significantly shorter latencies to maximum penile circumference than either PE non-ejaculators or controls ($F[1,20$ or $24] \geqslant 7.68$; $P \leqslant 0.011$), but no difference occurred between PE non-ejaculators and controls ($F[1,18] = 1.62$; $P = 0.219$).

Table 1 Sexual functioning and activity ($M \pm$ s.e.) in men with PE and men without PE[a]

Measure	*PE (n = 25)*	*Control (n = 13)*	P
Age	46.6 (2.1)	45.5 (1.9)	0.73
% Married or cohabiting at onset of study	82	93	
Satisfaction with sex life[b]	4.1 (0.2)	4.3 (0.4)	0.71
Intercourse frequency (times/week)	1.5 (0.1)	1.6 (0.2)	0.69
Masturbation frequency (times/week)	1.6 (0.2)	1.5 (0.1)	0.69
Ejaculation during any sex (times/week)	1.8 (0.2)	2.3 (0.4)	0.28
Erectile difficulty[b]	1.8 (0.2)	1.9 (0.4)	0.76
Sexual desire composite[b,c]	3.0 (0.3)	3.2 (0.2)	0.67
Opportunity for sex[b]	3.8 (0.3)	3.3 (0.6)	0.39
Mean time since last ejaculation before laboratory test (days)	4.8 (0.7)	3.3 (0.8)	0.05

Abbreviation: PE, premature ejaculation.
[a]Group comparisons were carried out using *t*-tests.
[b]For these items, 1 = none, never, or not at all, 7 = always or very high.
[c]This measure represents a composite of three items measuring 'interest in sex,' 'frequencies of sexual fantasies,' and 'frequency of sexual thoughts about women.'

Table 2 Comparison of penile response parameters between PE and control groups (mean ± s.e.)

	PE men (n = 21)	*Controls (n = 12)*
Max change from baseline (mm)	28.5 (2.3)	28.4 (3.3)
Latency to maximum (min)	5.34 (0.44)	5.90 (0.57)
Average penile response per min (determined by area under the curve)	77.1 (2.3)	86.0 (4.0)

Abbreviation: PE, premature ejaculation.

Table 3 Comparison of heart rate change from baseline between PE and control groups indicating *n* and mean ± s.e.

	Beats per min	N
Controls	−2.01 (0.57)	13
Men with PE (overall)	+1.81 (0.50)	25
Non-ejaculators in lab	+0.98 (0.43)	11
Ejaculating >3 min	+1.10 (0.39)	9
Ejaculating <3 min	+4.93 (0.65)	5

Abbreviation: PE, premature ejaculation.

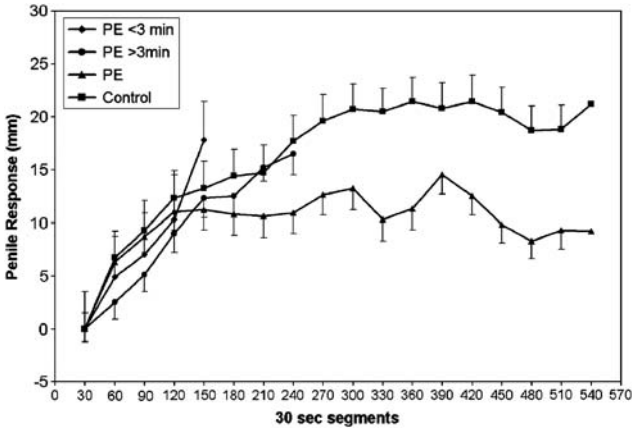

Figure 1 Penile response (change in mm from baseline) during visual sexual and vibrotactile stimulation in men with and without premature ejaculation. Abbreviations: VIB, vibrotactile stimulation to the penis; VSS, visual sexual stimulation.

Overall penile response. This measure provided a more comprehensive view of penile response than maximum tumescence, as the former is based on responding throughout the entire session rather than on a single data point (Figure 1). Group differences were significant, with the PE group exhibiting less overall (average) penile response than the control group ($F[1,31] = 4.76$; $P = 0.037$).

Heart rate

Figure 2 illustrates the heart rate responses of all participants. As with penile response, because session lengths differed for PE men who ejaculated (as their session terminated shortly after ejaculation), PE ejaculators and PE non-ejaculators are depicted separately. Five men with PE ejaculated within 3 min of the onset of stimulation and are illustrated as a separate group. Nine men with PE ejaculated at or after 4 min of recording, so these nine were used to generate their initial 4-min profile plus one 'composite' data point (defined earlier). The remaining 11 did not ejaculate during the session.

The control group showed a pattern of decreasing heart rate during the initial part of the session,

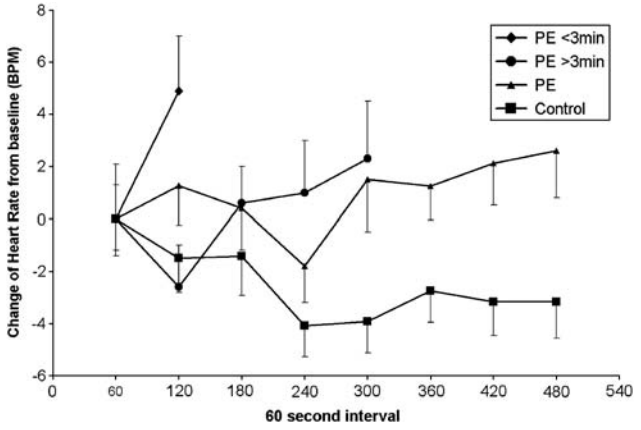

Figure 2 Heart rate (change in bpm from baseline) during visual sexual and vibrotactile stimulation in men with and without premature ejaculation. Abbreviations: VIB, vibrotactile stimulation to the penis; VSS, visual sexual stimulation.

followed by stabilization. In contrast, PE men showed several heart rate patterns. Specifically, 11 PE men who did not ejaculate during the session showed initial deceleration followed by acceleration to above baseline. Nine men who ejaculated after 3 min into the session also showed a deceleration in heart rate followed by a rapid rise as climax approached. But their biphasic response was advanced within the session, such that the entire response was compressed into a much shorter period of time. The remaining five PE ejaculators ejaculated in less than 3 min (Figure 2). These men showed no initial deceleration, but only a rapid increase in heart rate with the peak coinciding with ejaculation.

Analysis of heart rate. Comparisons of heart rate change from baseline, and were carried out using ANOVA (Table 3). PE and control groups were significantly different ($F[1,36] = 5.88$; $P = 0.020$), with PE men showing overall higher average heart rate than controls, and PE ejaculators (vs non-ejaculators) showing the greatest elevation relative to controls.

Discussion

This study compares PE men with functional counterparts on measures of penile response and heart rate under controlled laboratory conditions. Men with and without PE were similar on their maximal penile response, but showed differences in the latency to maximum. A measure capturing more global penile response patterns yielded group differences, with the PE group exhibiting an overall weaker response than controls. Consistent with this general trend, a greater proportion of men with PE

were eliminated from the analysis owing to a lack of response than controls (7.7 vs 16%); had non-responders been included, the group differences would have been yet more pronounced. Whether this difference resulted from, as hypothesized, greater inhibition of erectile response in PE men or from a selection bias in our groups (volunteer control subjects represent a self-selecting group) is not known. However, even those PE men responding with some erectile response showed lower 'average' (though not peak) penile responses than controls.

Consistent with previous studies, considerable inter-individual variation occurred in heart rate across the session, and therefore composite profiles represented herein should not be assumed typical of the individuals within their respective groups, particularly given the limited sample size in this study. Specifically, men in both groups showed acceleration, deceleration, no change, or a biphasic response over the 10 min stimulus session. Furthermore, changes in heart rate were small, about ± 12 bpm, relative to sexual arousal involving physical exertion. Nevertheless, patterns of heart rate appeared to differentiate PE men from controls at least as well as penile response. Men without PE exhibited a pattern of decreasing heart rate during the initial stages of sexual arousal and penile tumescence, a pattern similar to that seen in other psychophysiological studies measuring heart rate.[21,22] Toward the end of the session, when sexual action in the video was at its peak, some controls began to show a rise in heart rate, perhaps reflecting the shift as men moved from the erectile tumescence phase, dominated by attentional processes,[24] toward the plateau or ejaculatory latency phase. In contrast with controls, PE men exhibited a diminished deceleration phase and a consistent acceleration that was strongly portent of their rapid ejaculatory response. Indeed, for men who ejaculated in 3 min, the deceleration phase was absent altogether. Given that the intravaginal ejaculatory latency time of the men with PE in our study were not consistently under 1 min—a criterion level that represents the severest PE condition[37]—these results may actually underestimate the magnitude of such differences in heart rate patterns between men with and without PE.

Such patterns suggest that physiological responses of PE men and controls may serve as distinguishing characteristics and may, as hypothesized by others,[14] reflect a disruption of the typical autonomic processes involved in erection and ejaculation. Specifically, parasympathetic action early in the sexual response cycle is typically necessary to initiate and sustain erection, with subsequent sympathetic activation responsible in part for mediating ejaculation. In men with PE, this typical progression from parasympathetic to sympathetic dominance may be modified, such that sympathetic activation is initiated much earlier in the sexual response cycle, for example due to anxiety or negative affect, or weak or deficient parasympathetic activity. Early sympathetic control may account for the accelerating heart rate in these men and may be reflected in weaker parasympathetically mediated penile response. At the same time, this sympathetic dominance may trigger ejaculation prematurely, perhaps even before the man reaches maximum levels of cerebrally mediated sexual excitement—a common complaint among men with PE. The finding that PE men show less suppression of sympathetically mediated skin potentials (that is, an index of ongoing sympathetic activation) during papaverine-induced erections lends further support to the idea that parasympathetic-sympathetic interactions may be altered in PE men.[15]

Although 'early' or premature sympathetic activation might account for rapid ejaculation in PE men, the reason for this remains unexplained. For example, higher negative affect or deficient parasympathetic response [for example, see[38]] might sensitize the sympathetic system. Or strong negative affect might interfere with the typical cognitive-attentional processes during psychosexual stimulation that could delay sympathetic activation.[39] Or erectile response in PE men could be under greater sympathetic influence than in functional counterparts,[40] consistent with the idea that psychogenic genital vasocongestion in women (comparable to erectile engorgement in men) depends strongly on sympathetic innervation.[12,41]

Finally, this study suggests that, overall sexual hyperarousal may not provide an adequate understanding of PE, as such men actually show lower average erectile responses during the tumescence phase than controls. Furthermore, it underscores the point that an understanding of PE may benefit from analysis of both erectile/tumescence and ejaculatory phases of sexual response as well as from the inclusion of physiological measures such as electrodermal response and heart rate. Although a limitation of our study was the lack of stopwatch measured ejaculatory latency—which may have afforded greater precision in PE classification, overemphasis on the study of only the ejaculatory latency component in men with PE may well have obscured important differences between dysfunctional and functional men in their overall sexual response. That is, ejaculatory thresholds show interdependencies with both sexual libido and sexual arousal, so studies that isolate ejaculatory latency from the rest of the sexual response cycle may, in the end, be counterproductive to understanding the underlying causes and/or etiology of PE.

Conflict of interest

The author declares no conflict of interest.

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