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Relationships Among Cardiovascular, Muscular, and Oxytocin Responses During Human Sexual Activity

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To determine the psychophysiological correlates of hormonal response during sexual activity, systolic blood pressure (SBP), anal electromyography (EMG), and anal photoplethysmography (APG) were monitored continuously throughout testing in 13 women and 10 men. Each subject completed two or more tests of self-stimulation to 5 min beyond orgasm. Blood samples were obtained continuously for measurement of oxytocin (OT) levels. In both men and women, very high positive correlations were observed between the percentage change in levels from baseline through orgasm of: OT and SBP; OT and EMG intensity prior to and during orgasm; APG and EMG. The number of anal contractions and duration of orgasm were also highly correlated. Two patterns of orgasm were defined by the presence or absence of a quiescent period between orgasmic contractions. EMG and APG amplitudes correlated with the pattern of orgasm. Subjective orgasm intensity correlated significantly with increased levels of OT in multiorgasmic women only. The positive correlations between measures are consistent with a possible functional role for OT in human sexual response.

KEY WORDS: oxytocin; orgasm; sexual response; psychophysiology; pelvic contractions.

INTRODUCTION

The circulating levels of the neuropeptide hormone oxytocin increase during sexual arousal and orgasm in men and women (Carmichael *et al.*, 1987). The classical functions of oxytocin (OT) are to cause smooth muscle

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contractions of the uterus during parturition and contractions of the myoepithelial cells for milk ejection during lactation. Recently, involvement of OT in maternal, sexual, and social behaviors has been proposed (see Carter, 1992, for review) but its function(s) in men and nonpregnant, nonlactating women is still unclear.

Both striated and smooth muscles contract during sexual arousal and it is believed that the uterus contracts during orgasm (Masters and Johnson, 1966; Fox and Fox, 1971; Fox, 1976). Although definitive evidence of this is lacking, a possible function of OT is to facilitate smooth muscle contractions of the reproductive tract of men and women during orgasm (Carmichael *et al.*, 1987; cf. Newton, 1978). If so, semen ejection from the male and transport within the reproductive tract of the female could be facilitated by OT. There is animal evidence to support this proposed function. For example, in the male mouse the anococcygeus smooth muscles (associated with the male urogenital tract in several species including man) produce sustained contractions in response to OT (Gibson *et al.*, 1984).

Continuous multiple orgasms are reported to occur in adult men as well as in women (Robbins and Jensen, 1978). A previous speculation suggested that only terminative orgasms of women (those followed by satiation and a period of sexual refractoriness) are accompanied by intense uterine contractions, whereas nonterminative (multiple) orgasms are not (Davidson, 1980). Indeed, OT levels were higher during terminative orgasms in mono-orgasmic and multiorgasmic women (Carmichael *et al.*, 1987). In addition to the effect OT may have on the physiological aspects of orgasm, it may indirectly promote the psychological state of sexual satiety by facilitating orgasm.

The main purpose of this study was to determine whether measures of subjective and physiological sexual arousal and orgasm correlated with levels of plasma OT (values of OT reported in Carmichael *et al.*, 1987). The activity of the striated musculature of the pelvic floor of men and women can indicate sexual arousal and is diagnostic of orgasm when quantitatively measured by anal electromyography (cf. Kadefors and Peterson, 1970; Bohlen *et al.*, 1982). Additionally, increased vasocongestion in the pelvic area, as measured by anal photoplethysmography, is indicative of sexual arousal and orgasm in men and women. Use of anal measures allows direct comparisons of the physiological sexual responses between women and men. Systolic blood pressure can be used to assess extragenital autonomic arousal. All these measures were employed concomitantly in this study in order to increase understanding of the psychophysiology of human sexual activity.

METHOD

Subjects

Thirteen women and 10 men completed the study. Subjects were recruited by bulletin board notices in Stanford Medical School and local newspaper advertisements. Volunteers visited the research facilities for further information and informed consent. They completed a short medical history and the following questionnaires: Minnesota Multiphasic Personality Inventory (Hathaway and McKinley, 1943), Spielberger Manifest Trait Anxiety test (Spielberger *et al.*, 1970), Beck Depression Inventory (Beck and Beamesderfer, 1974), and Abramson-Mosher Attitudes towards Masturbation Scale (Abramson and Mosher, 1975). Individuals selected for study were 21–40 years old, in good physical, sexual, and mental health, without major negative attitudes toward masturbation, and without a history of hepatitis or psychoses, or present debilitating disease. Males ranged in age from 21 to 37 ($\bar{x}$ = 28) years and females from 21 to 40 ($\bar{x}$ = 27.5) years. Six women were nulliparous. There were 15 Caucasians, 2 Asians, 3 Hispanics, and 3 Blacks. Each individual was met in privacy and insured of anonymity and confidentiality. All subjects completed a series of desensitization sessions in which tests were simulated but no data collected. Only after the subject was comfortable with the procedures did actual testing begin.

Apparatus

Experiments were conducted in a sound-attenuated room equipped with a reclining chair and television. Recording equipment, video cassette player, and a peristaltic pump for withdrawal of blood samples were located in an adjacent room.

Systolic blood pressure (SBP) recordings were obtained from a standard arm cuff inflated by a Schneider Automated Sphygmomanometer which was programmed for 1 measurement/20 sec. Anal photoplethysmography (APG) and electromyographic activity (EMG) were obtained from an acrylic anal probe (Biotechnologies Inc., 242 Old Eagle School Road, Strafford, PA 19087), containing an infrared light-emitting diode and photosensor for monitoring blood-pulse amplitude, and smooth-surface electrodes for monitoring muscle activity. Venous blood samples were collected from the forearm via an indwelling, 21-gauge butterfly needle attached to a 152 cm heparin/protamine line, with a dead space of 2 ml. A video cassette player projected to the television in the test room for those subjects requesting viewing of erotic films. All leads, including the blood line, passed through the test room wall into the adjoining laboratory.

Recordings were made on a 6-channel Grass Polygraph 7 at a paper chart speed of 10 mm/sec. APG was calibrated at a standard setting of 20 mv/cm and EMG at 300 mv/cm. Subjects were provided with a manually operated foot pedal, attached to a battery-powered buzzer which signaled the beginning and end of subjective orgasm.

Posttest self-ratings of intensity, satisfaction, and pleasure associated with orgasm(s) during testing were obtained on 7-point Likert scales, and personal comments were elicited by essay-type questions on the paper-and-pencil, posttest evaluation form. Men were questioned as to whether they ejaculated with or without orgasm or experienced orgasm without ejaculation.

Procedure

Two desensitization sessions without blood sampling were conducted. In the first, the subject became acquainted with the laboratory and erotic films were shown as requested. One week later, a simulated test session was conducted. The subject was fitted with the blood-pressure cuff and instructed in insertion and removal of the anal device. The subject was then left in privacy to relax and to then self-stimulate to orgasm with the test equipment in place. All subjects completed two test sessions in which the physiological measures were obtained continuously from 6 min prior to self-stimulation to 5 min after end of orgasm. The replication test was conducted 4–5 weeks after the first test, both tests occurring in the same phase of the menstrual cycle for female subjects. Self-stimulation was conducted according to the subject's regular method.

Blood sampling and plasma oxytocin measurement by radioimmunoassay are described in detail elsewhere (Carmichael *et al.*, 1987). The sensitivity of the hormone assay was 0.28 ± 0.21 pg/assay tube. Each tube was extracted from a 10 ml/min blood sample identified in chronological order during testing, as follows: *baseline* (5 min prior to self-stimulation); *early SS* (2 min after onset of self-stimulation); *mid SS* (halfway through the duration of self-stimulation for those individuals self-stimulating for more than 8 min prior to orgasm); *late SS* (the last 10 ml sample collected prior to orgasm); *O-0* (immediately after the subject signaled orgasm and the dead space of the blood line was cleared); *post-O* (2 and 5 min after the subject signaled end of orgasm).

Data Analysis

Since there were no statistically significant differences between first and second test results (*t* tests for correlated means), they were averaged for subsequent data analyses. Data of multiorgasmic women were analyzed

separately from those of mono-orgasmic women and men. One man who was mono-orgasmic during one test session was multiorgasmic in another. He was given an additional test session during which he was multiorgasmic. Due to technical difficulties, no blood samples were obtained for this man. Data from a separate analysis of his psychophysiological data, collected during the multiorgasmic sessions, are reported.

Statistical significance of changes in SBP, EMG, and APG in all mono-orgasmic subjects was tested by analysis of variance (ANOVA) for repeated measures. Student's *t* tests were performed to examine possible differences between mono-orgasmic and multiorgasmic groups and possible gender differences of mono-orgasmic subjects. Statistical correlations were calculated among APG, EMG, and SBP; and between the per person levels of OT and the mean duration of self-stimulation; duration of orgasm; number of contractions during orgasm; mean EMG, APG, and SBP values before, during, and after orgasm.

Systolic Blood Pressure. The mean and SEM were calculated for six SBP readings (3 SBP readings × 2 test sessions) covering the time of collection of each 10-ml blood sample. Linear regression analyses were performed using the mean values of SBP obtained during baseline, self-stimulation, orgasm, and postorgasm for each subject. Statistical significance of the slope of the line was tested by two-tailed *t* test for zero linear regression.

Electromyography. For each blood sampling period, the last 30–35 sec were selected for EMG analysis. The two highest recorded amplitudes in each 1 sec segment were measured and a mean was computed for the 10 measures obtained from each 5-sec segment sampled during baseline, self-stimulation, and postorgasm. During orgasm, the highest amplitude of each discrete EMG spindle, occurring with each contraction, was measured and a mean computed from the total number of spindles for each subject.

Anal Photoplethysmography. The first 10 blood–pulse wave forms recorded at the beginning of the last 30 sec of each blood sampling were selected for analysis, with the exception of orgasm when the 10 blood–pulse wave forms recorded from the start of the first contraction of orgasm were used. The amplitudes were measured and a mean calculated for each 10 data points.

Posttest Subject Evaluations. The subjective ratings of intensity, pleasure, and satisfaction of orgasm, scored from a 7-point Likert scale, were tested for statistical correlation with the number of contractions recorded during orgasm; with the mean values of EMG, APG, SBP, and OT levels during orgasm, respectively; and with the objectively recorded, as well as the subjectively signaled, durations of orgasm.

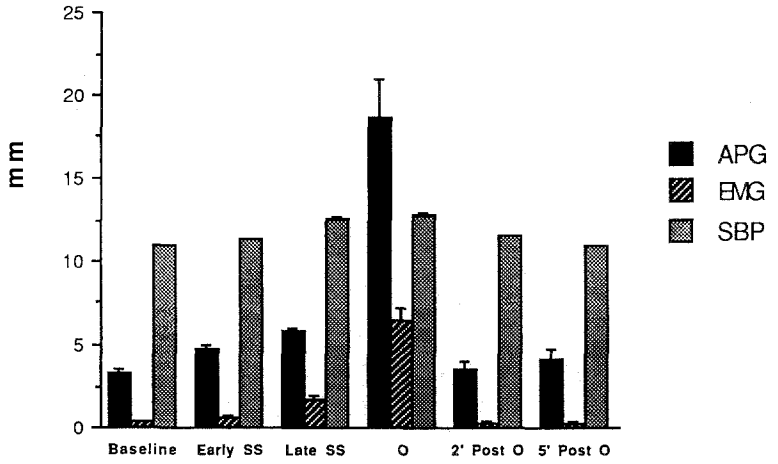

Fig. 1. Psychophysiological Measures: $\bar{x}$ (+SEM) values of APG and anal EMG in mm amplitude of pen deflection and SBP in mm Hg/10 at baseline and at various stages before, during, and after self-stimulation (SS) to orgasm (O). See text (Methods, Procedures) for descriptions of stages and abbreviations in this and other figures.

RESULTS

Significant increases were recorded for APG, $F(5, 85) = 38.84$, $p < 0.0005$; EMG, $F(5, 85) = 47.63$, $p < 0.0005$; and SBP, $F(5, 85) = 6.6$, $p < 0.05$, during testing, reaching maximum values during orgasm and declining thereafter (Fig. 1). There was no statistical difference between the subjectively defined duration of orgasm and the objectively measured duration of APG and EMG. The mean and SEM of durations of self-stimulation and orgasm and the mean number and SEM of contractions during orgasm are reported in Table I. Intercontraction intervals during orgasm increased over time for most men and women. There were no statistically significant differences between men and women or between mono-orgasmic and multiorgasmic women in duration of self-stimulation and orgasm or number of contractions during orgasm. Statistically significant positive correlations among the various measures are shown in Table II.

Oxytocin Levels

Oxytocin levels increased from baseline levels through orgasm (see Carmichael *et al.*, 1987). As shown in Table II, levels of OT correlated positively with increases in SBP and EMG. In addition, the percentage change

in OT levels from baseline to and through orgasm correlated positively with EMG intensity during orgasm in all subject groups (mono- and multiorgasmic women: both $r = 0.81$; mono-orgasmic men: $r = 0.73$; all $p < 0.05$). There were no significant correlations between OT levels and the mean duration of self-stimulation, duration of orgasm, number of contractions during orgasm, mean APG values before, during, and after orgasm, SBP after orgasm or with the presence or absence of the bulbocavernosus reflex. Oxytocin levels during orgasm correlated significantly with the pattern of orgasmic contractions as described below.

Systolic Blood Pressure

As expected, men had higher SBP during baseline than women: 117 vs. 102 mm Hg, respectively (Fig. 2). For all groups, SBP increased from baseline through orgasm. This increase was significant for both mono-orgasmic men and women during late SS ($t = 1.97, p < 0.05$ and $t = 4.08, p < 0.005$, respectively) and for mono-orgasmic women during orgasm ($t = 3.53, p < 0.01$). The changes in SBP for multiorgasmic women did not reach significance, possibly due to low power resulting from the small number of multiorgasmic female subjects. In general, SBP declined following orgasm but this change was significant only in mono-orgasmic women from orgasm to 5 min postorgasm ($t = 3.53, p < 0.01$). For the single multiorgasmic man, mean SBP was greater during ejaculation than during prior orgasms without ejaculation (159 vs. 117 mm Hg, respectively). There were no differences in SBP during first and second orgasms for multiorgasmic women. There were no other statistically significant group or gender differences in SBP throughout self-stimulation, orgasm, and postorgasm. Although there was a significant correlation between SBP and EMG from baseline to beginning of orgasm (Table II), SBP did not correlate significantly with APG or EMG amplitude during or after orgasm.

Electromyography

Anal-EMG amplitude for all subjects was less than 0.5 mm during baseline measures, and there were no significant differences among groups in resting values. The amplitude increased significantly from baseline through orgasm for women ($t = 8.9, p < 0.001$) and men ($t = 5.68, p < 0.001$). Men had a significantly higher mean EMG amplitude during orgasmic contractions than women with means of 8.5 mm and 4.4 mm, respectively; $t = 2.63$, $p < 0.02$ (Fig. 3). During male and female orgasm, EMG amplitude increased with each contraction until a point midway to late in the duration of the orgasm whereupon the amplitude began to decrease.

Table I. Parameters of Self-Stimulation and Orgasm: Mean ± SEM of Duration of Stimulation, Duration of Orgasm, and the Frequency of Contractions During Orgasm

Subjects	*n*	Self-stimulation duration (min)	Orgasm	
			Duration (sec)	Contraction frequency
Mono-orgasmic				
Women	9	15.4 ± 4.24	21.9 ± 6.40	13.7 ± 2.64
Men	9	12.6 ± 2.87	21.8 ± 3.46	15.8 ± 1.73
Multi-orgasmic				
Women	4			
1st orgasm		4.7 ± 0.54	25.8 ± 0.25	18.8 ± 6.77
2nd orgasm		6.9 ± 1.15	26.0 ± 2.31	15.0 ± 1.00
Man	1			
1st orgasm		11.8 ± 1.76	20.5 ± 10.53	25.0 ± 15.04
2nd orgasm		4.0	47.0	28.0
3rd orgasm		6.5	35.0	22.0
4th orgasm		3.0	25.0	22.0
Ejaculation		6.8 ± 6.27	32.0 ± 2.01	19.5 ± 1.50

Table II. Positive Correlations between Physiological, Hormonal and Subjective Measures of Sexual Activity in Mono- and Multiorgasmic Women and Men

	Mono-orgasmic men	Mono-orgasmic women	Multiorgasmic women
Baseline to beginning of orgasm			
EMG and plasma oxytocin	.99[b]	.96[a]	.88[a]
EMG and SBP	.98[a]	.97[b]	ns
Changes from baseline through orgasm			
Plasma oxytocin and SBP	.96[b]	.94[b]	ns
EMG and plasma oxytocin	.77[a]	.82[a]	.92[a]
EMG and anal photoplethysmography	.99[b]	.98[b]	.83[a]
Number of anal contractions and duration of orgasm	.86[c]	.91[c]	.98[a]
Subjective report of orgasm intensity and			
Levels of oxytocin	ns	ns	.94[a]
EMG during orgasm	ns	ns	.72[a]
Pleasure	.73[b]	.75[a]	.60[b]
Satisfaction	ns	.85[b]	.48[a]
Terminative orgasm to 5 min postorgasm			
EMG and plasma oxytocin	ns	.99[c]	.93[a]
EMG and anal photoplethysmography	.99[c]	.99[a]	.99[a]

[a] $p < 0.05$.
[b] $p < 0.01$.
[c] $p < 0.001$.

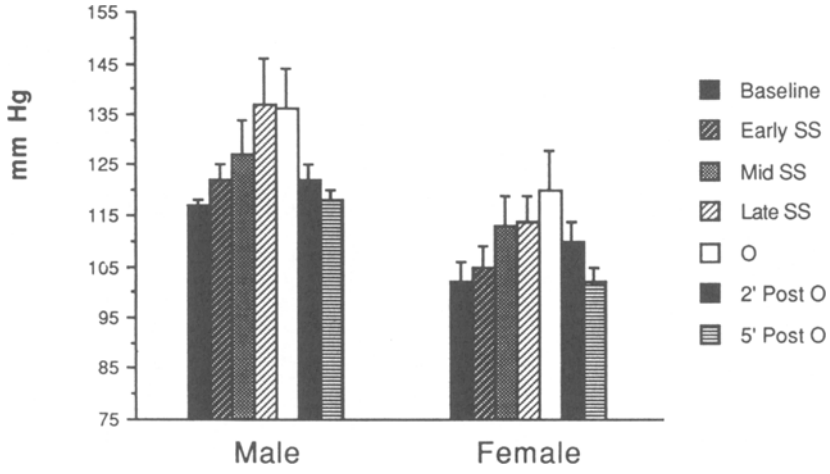

Fig. 2. Systolic blood pressure: $\bar{x}$ (+SEM) SBP (mm HG) for mono-orgasmic men (n = 9) and women (n = 9) at baseline and at various stages before, during, and after self-stimulation to orgasm.

Multiorgasmic women did not differ significantly from mono-orgasmic women in the rate of increase in EMG amplitude during self-stimulation to first orgasm. In multiorgasmic women, a maximum increase in EMG amplitude occurred during each orgasm; $t = 7.98$, $p < 0.05$ (first orgasm); $t = 12.31$, $p < 0.005$ (second orgasm); and the increased value during either orgasm did not differ significantly from that of mono-orgasmic women during their single orgasm (see Fig. 3). In the multiorgasmic man, the EMG amplitude during orgasms without ejaculation was less than the amplitude during orgasms with ejaculation (means of 6.2 and 14.5 mm, respectively). The most noticeable difference between the data of multiorgasmic women (Fig. 3) and those of the multiorgasmic man is the greater EMG amplitude recorded during the man's terminative orgasm (with ejaculation), compared to the EMG amplitude of the women during their terminative orgasm. EMG decreased significantly in both men ($t = 5.69$, $p < 0.001$) and women ($t = 4.23$, $p < 0.001$) by 2 min postorgasm.

Patterns of Orgasmic Contractions

EMG recordings revealed two discernible patterns of contractions during orgasm. While Type A is characterized by a continuous series of

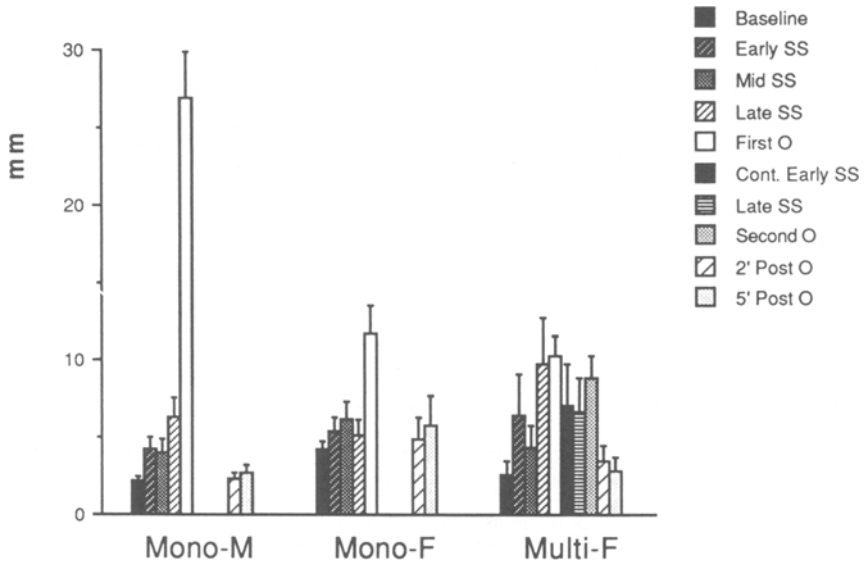

Fig. 3. Electromyography: $\bar{x}$ (+SEM) mm amplitude of pen deflection during anal EMG measures for mono-orgasmic men (n = 9), mono-orgasmic women (n = 9) and multiorgasmic women (n = 4) at baseline and at various stages before, during, and after self-stimulation to orgasm.

contractions whose frequency decreases with the passage of time, Type B is defined by one or more quiescent period(s) in the series (Fig. 4). The presence of a quiescent period was the primary criterion used by independent raters blind to each others ratings for classifying the pattern of orgasmic contraction. The break lasted 2–3 sec in which no contractions occurred before resumption of the rhythmic contractions or several irregular contractions.

Most individuals (70%) showed a similar pattern of orgasmic contractions during each test. As shown in Table III, the types tended to differ in the number of contractions during orgasm, with a greater mean for Type B than Type A (t = 1.71, $p < 0.1$). The duration of orgasm for Type B was significantly greater than for Type A (t = 2.13, $p < 0.05$). Duration of self-stimulation to orgasm did not differ significantly for the different patterns of orgasm. Different levels of OT were correlated with the pattern of orgasm (r = –0.44, $p < 0.02$); higher levels of OT were correlated with Type A than with Type B (mean OT levels of 3.5 and 2.7 pg/ml, respectively).

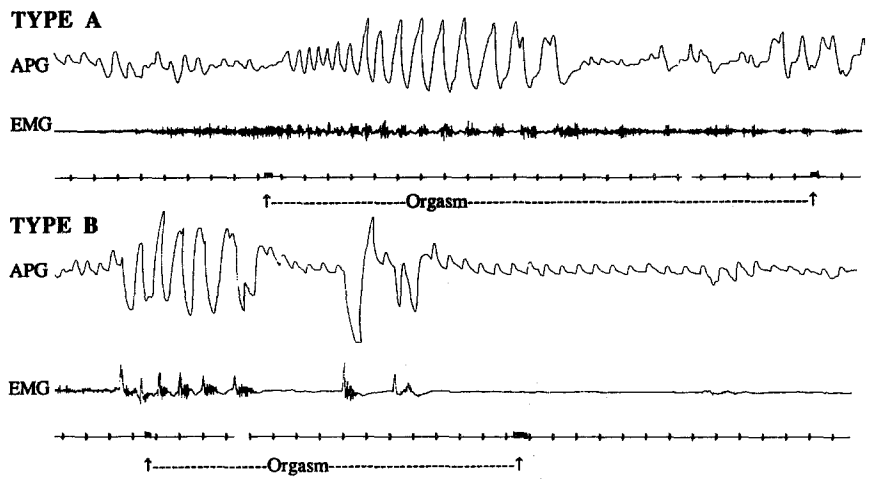

Fig. 4. Patterns of orgasmic contractions: Segments of typical polygraph recordings showing APG trace, anal EMG trace, and a time line (1 sec per notch) indicating subjective onset and offset of orgasm (rectangular notches) for Type A and Type B patterns of contraction during self-stimulation to orgasm.

A low-amplitude tonic burst of EMG activity immediately preceding the onset of orgasm was observed. In some individuals this activity manifested as a marked reduction in APG amplitude sustained for at least 3 sec immediately preceding orgasm (cf. Gillan and Brindley, 1979). Eight of the nine mono-orgasmic men had this activity on each test; the ninth showed no evidence of it. In the multiorgasmic man, the activity appeared in one of three ejaculations and in two of six orgasms without ejaculation. Two of the eight mono-orgasmic women and three of the four multiorgasmic women had the activity on at least one test occasion. There was no correlation between pattern of orgasm and presence or absence of the activity. There was a trend for EMG amplitude during orgasm to correlate with the pattern of orgasmic contractions ($r = .32$, $p < 0.10$). EMG amplitude tended to be higher in Type A than in Type B orgasms ($\bar{x} = 7.33$ and 4.93 mm, respectively). There was also a trend for APG amplitude during orgasm to correlate with the pattern of orgasm ($r = .35$, $p < 0.10$); to be higher in Type A orgasms than in Type B ($\bar{x} = 21.3$ and 13.3 mm, respectively).

Table III. Patterns of Orgasm

	Orgasm ($\bar{X}$ ± SEM)		Subjects (n)			
	No. of contractions	Duration (sec)	Mono-W	Mono-M	Multi-W	Multi-M
Type A	12.5 ± 1.41	16.3 ± 2.72	4	6	—	—
Type B	16.7 ± 2.05	26.9 ± 4.28	2	1	3	—
A and B	—	—	2	2	1	1
Unclassified	—	—	1	—	—	—

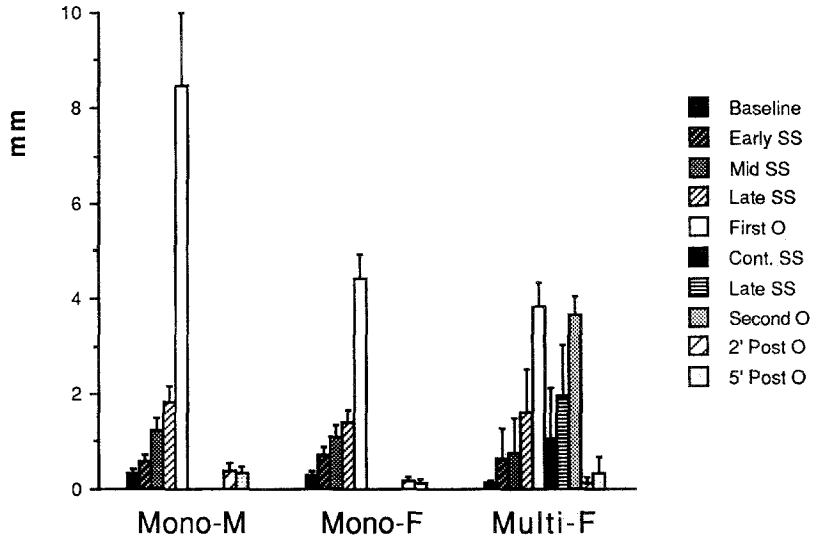

Fig. 5. Anal photoplethysmography: $\bar{x}$ (+SEM) mm amplitude of pen deflection during APG measures for mono-orgasmic males (n = 9), mono-orgasmic females (n = 9) and multiorgasmic females (n = 4) at baseline and at various stages before, during, and after self-stimulation to orgasm.

Anal Photoplethysmography

Women had significantly higher mean APG amplitude during baseline than men (4.2 vs. 2.2 mm, respectively; t = 3.18, $p < 0.01$; Fig. 5). Linear regression analysis revealed significant increases from baseline through orgasm for women (t = 3.15, $p < 0.02$) and men (t = 8.05, $p < 0.001$). During orgasm, however, men had significantly higher APG amplitude than women (26.9 vs. 11.7 mm, respectively; t = 4.16, $p < 0.001$). After orgasm, APG amplitude declined significantly in men (t = 5.04, $p < 0.001$). Multiorgasmic women did not differ significantly from mono-orgasmic women in APG amplitude values during baseline, self-stimulation to first orgasm, and first orgasm. There was no significant difference in APG amplitude between the first and second orgasms of multiorgasmic women ($\bar{x}$ = 10.3 and 8.9 mm, respectively). APG amplitude declined to baseline value within 5 min after the second orgasm. For the multiorgasmic man, there was no significant difference in APG amplitude between ejaculatory or nonejaculatory orgasms ($\bar{x}$ = 22.4 and 26.96 mm, respectively).

MMPI Personality Profiles

There were no correlations between personality profiles and the other measures taken in this study.

Posttest Subject Evaluations

Subjective posttest evaluations of intensity of orgasm correlated significantly with increased levels of OT and with EMG intensity in the multiorgasmic women but not in the mono-orgasmic subjects (Table II).

No significant correlations were found among the subjective ratings of intensity, pleasure, or satisfaction of orgasm and the number of contractions during orgasm, duration of orgasm, APG amplitude during orgasm, SBP during orgasm, or pattern of orgasm.

DISCUSSION

The psychophysiology of human orgasm is presently very poorly understood, despite its potential clinical relevance to fertility and various important sexual dysfunctions such as female anorgasmia and premature ejaculation (Gebhard, 1970; Swieczkowski and Walter, 1978; Levin, 1981). Relatively few attempts have been made to correlate hormone levels with sexual activity, and most of these have reported negative results. In humans, plasma levels of OT have been shown to rise after ejaculation (Ogawa *et al.*, 1980) and during sexual arousal in men and women with greater increases during orgasm (Carmichael *et al.*, 1987; Murphy *et al.*, 1987). This periorgasmic rise in OT can be blocked by naloxone administration in men (Murphy *et al.*, 1990). In multiorgasmic subjects, OT peaks immediately prior to and during terminative orgasm; i.e., coinciding with sexual satiation. Could periorgasmic OT release in both sexes contribute to reproductive tract smooth muscle contractions? The nonpregnant human uterus contracts, albeit weakly, to low concentrations of OT (less than 10 mU/ml), whereas its spontaneous myogenic activity is inhibited by concentrations higher than 10 mU/ml or more (Wikland *et al.*, 1982). Nevertheless, it remains to be determined whether the uterus contracts rhythmically or tonically during orgasm and whether OT levels correlate with the presence or absence of uterine contraction. In males, administration of exogenous OT can stimulate the vas deferens, epididymis, seminiferous tubules, prostate, anococcygeus muscle, and rectum to contract (Melin, 1970; Knight, 1972; Hib, 1974; Mishra and Raviprakash, 1982; Peeters *et al.*, 1983; Gibson *et al.*, 1984). Furthermore, inhibition of OT release in male rabbits reduced the

number of sperm ejaculated by 45% (Sharma and Hays, 1973). However, studies in other animal species have produced conflicting results regarding whether mating and/or ejaculation is associated with increases in plasma OT levels (see Schams *et al.*, 1982; Peeters *et al.*, 1983; Stoneham *et al.*, 1985).

In a previous review, the potential usefulness of studying the oxytocic reflex in relation to sexual psychophysiological events was pointed out (Davidson, 1980). The purpose of the present study was to investigate relationships among physiological correlates of orgasm, particularly with reference to increases in OT levels. The results showed that the intensity of orgasmic contractions, but not their duration, correlated positively and significantly with increases in OT levels. For multiorgasmic women, the amount of OT increase also correlated positively with subjective ratings of orgasmic intensity. No such significant correlations were found in mono-orgasmic men or women. A speculative explanation is offered: perhaps the discrepancy occurred because the multiorgasmic women were able to make within-test comparisons of the orgasm intensity, whereas mono-orgasmic subjects may have rated their single test orgasm by comparison to "at-home" orgasms. Murphy *et al.* (1990) reported that while administration of naloxone to men prior to self-stimulation to orgasm did not affect heart rate or blood pressure, it did block the periorgasmic rise in OT. Subjects receiving naloxone also reported a decrease in the level of subjective arousal and pleasure at orgasm. Oxytocin has been shown to facilitate sexual behavior in male and female rodents, while oxytocic antagonists disrupt sexual interactions (e.g., Arletti *et al.*, 1990; Caldwell, 1991).

There were no significant differences in levels of OT or EMG amplitudes during baseline testing in men and mono- or multiorgasmic women. In general, during baseline testing, women had higher APG amplitudes than men. In addition, women had higher levels of OT than men during both sexual arousal and orgasm, while men had significantly greater measures of APG and EMG. Kadefors and Petersen (1970) found similar gender differences with voluntary contractions of the external anal sphincter. For all individuals in the present study, intensity of muscle contractions were positively related to the amount of OT released, but men and women differed in the absolute amounts of OT released in relation to the comparative intensity of their orgasmic contractions. Though OT may be instrumental in regulating genitopelvic smooth muscle, the synchronous striated muscle contractions of the pelvic floor, which are stronger in men than women, are the main physiologically identifiable experiential event signaling orgasm in both sexes.

Masters and Johnson (1966) reported that some young "multi-ejaculatory" men experience repeated orgasms with ejaculations. However, it is widely believed that many women but not men can have multiple orgasms (Masters and Johnson, 1966; Kaplan, 1974). Disputing this common belief, Robbins and Jensen (1978) reported on 13 healthy men who had repeated orgasms without ejaculation prior to a terminative orgasm with ejaculation. However, it is not established that the subjective experience is substantially comparable to that of the terminative orgasm. One man in the present study showed this behavior. His quantitative data showed that while APG and EMG increased during each of the multiple orgasms, a greater increase in EMG was observed during the terminative orgasm and that SBP only increased with ejaculation. Based on a very small number of multiorgasmic subjects (one male and four female), there may be a gender difference in this phenomenon. The first and terminative orgasms were not significantly different for women (although there is a possibility of Type II error due to low power). For the multiorgasmic man, however, there was a difference in the terminative orgasm, compared with the preceding, physiologically incomplete orgasms.

The increase in plasma OT during sexual arousal and orgasm may be due to a reflex release induced by initial stimulation of receptors on afferent pelvic nerves, which can also result in the milk ejection reflex during lactation (cf. Peeters and Houvenaghel, 1973). Additionally, because oxytocin can alter neuronal electrical activity, influence cAMP production, and affect catecholamine turnover rates (Sofroniew, 1983), increased levels during sexual arousal may aid in precipitating neuronal activity necessary to orgasm. There is, in all likelihood, an existing interplay between neurogenic sympathetic influences and possible oxytocin effects on orgasm.

The temporal pattern of orgasmic contractions is significantly correlated with levels of oxytocin in both women and men. Bohlen *et al.* (1982) reported three patterns (I, II, and IV) of contraction for women during orgasm. The pattern they designated Type I corresponds to our Type A, which correlated with the highest levels of oxytocin. Their Type II was not evident in our study. Our Type B, which correlated with lower levels of oxytocin, appears to be the same as Type III, which Bohlen *et al.* (1980) found in one male only. These comparisons are, however, somewhat tentative since the Bohlen orgasmic patterns derived from pelvic pressure changes rather than EMG data. Bohlen *et al.* (1982) suggested the potential clinical usefulness of differences in patterns of orgasmic contraction if these patterns were found to correlate with differences in personality characteristics. No significant correlations were found, however, between different personality profiles derived from the MMPI and the Type A and B patterns

of orgasm. Intercontraction intervals during orgasm increased with time for women and men. This result agrees with previously reported findings (Peterson and Stener, 1970; Gillan and Brindley, 1979; Bohlen *et al.*, 1980) as does our number of contractions for mono-orgasmic women (Bohlen *et al.*, 1982) and men (Bohlen *et al.*, 1980).

Interestingly enough, the duration of orgasm was not correlated with positive affect or subjective magnitude. Thus, brief orgasms were reported just as pleasurable, satisfying, and intense as longer orgasms. (See Davidson, 1980, for discussion of experiential phenomena and physiological relationships in sexual arousal and orgasm.)

The bulbocavernosus reflex, which may be evoked by compression of the glans penis or glans clitoris (Lapides and Bobbitt, 1956), is classically described as a reflex contraction of the bulbocavernosus (BC) muscle and striated anal sphincter muscle. Gillan and Brindley (1979) suggested that this would be described more accurately as a "glandipudendal reflex" to account for the involvement of muscles other than the BC. The BC reflex is usually phasic, but a tonic glandipudenal reflex was also described by Gillan and Brindley using intravaginal measurements. In the present study, a 1–3 sec or more tonic, low-amplitude contraction preceded the rhythmical contractions of orgasm in nine men and five women in at least one of their tests. Masters and Johnson (1966) described a similar finding as an initial muscle spasm before rhythmic contractions in some subjects. They stated that the subjective report of orgasm paralleled the spasm rather than the onset of synchronous contractions. This was not the case for our subjects who signaled onset of orgasm just as rhythmic contractions began, shortly after or just at the end of the bulbocavernosus contraction. It should be noted that the male bulbocavernosus reflex need not be restricted to orgasms with ejaculation as the multiorgasmic man showed the reflex preceding two orgasms without ejaculation. This man reported loss of sexual arousal only after the final orgasm of each test session in which ejaculation occurred, in agreement with the report of Robbins and Jensen (1978) that multiple nonejaculatory orgasms of men are terminated by a final orgasm accompanied by emission. This is also consistent with a previous suggestion that smooth muscle activity including uterine contraction and seminal emission and ejaculation are related to postorgasmic satiety (Davidson, 1980). As discussed earlier, SBP and EMG amplitude were significantly higher during orgasms with ejaculation than without ejaculation in the multiorgasmic man. These results differ from previous reports of lack of differences in cardiovascular, respiratory, and pelvic muscle contraction data between nonejaculatory and ejaculatory orgasms (cited in Davidson, 1980).

A positive correlation was found between increased levels of OT and increased SBP in both sexes. The increase in SBP with orgasm has been reported previously by Masters and Johnson (1966) and Fox and Fox (1969) with masturbation or coitus, and by Littler *et al.* (1974), using direct arterial blood pressure measures during coitus. Blood pressure is increased by intracisternal administration of OT in dogs (Montrastruc *et al.*, 1983), suggesting the possible involvement of OT in the central control of blood pressure. Other studies have also pointed to a relation between decreased OT levels in the brain and increased levels in the neurohypophysis and in peripheral blood, with hypertension in the rat (Morris and Keller, 1982). In addition, because OT can be a potent vasodilator in the presence of high degrees of tone in skeletal muscle and other vasculatures (thereby increasing blood flow in several organ regions) and is potent as a contractile agent particularly on umbilical-placental blood vessels (Altura and Altura, 1984), it is possible that OT plays a role in the vasocongestion associated with sexual arousal. Other studies have suggested a regulatory role of OT in the peripheral control of blood pressure (Bohus, 1980) and contractility of vascular smooth muscle (Sofroniew, 1983).

The present report concerns correlations between levels of oxytocin and psychophysiological measures. Although this does not provide evidence for causation, one may speculate as to the mechanisms by which oxytocin influences orgasm. By direct action on smooth muscle in the genital-pelvic area, OT may induce/facilitate contractions. It may also act peripherally to sensitize nerve endings monitoring striated muscle contractions. OT may also serve as a neuromodulator, affecting cerebral neurons responsible for the cognitive feeling of orgasms and/or OT may sensitize cerebral neurons associated with pelvic floor striated muscle contractions. Our study indicates that the psychophysiological changes occurring during sexual arousal and orgasm correspond to changes in levels of OT, and that these changes may be of some functional significance in the human sexual response cycle. At the very least, it is clear that genital and extragenital physiological (including endocrine) events are all part of a precisely coordinated reaction. Mono-orgasmic men and women do not appear to differ qualitatively in the parameters studied, but gender differences can be found in the magnitude of the response measures.

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