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Penile sensitivity in men with premature ejaculation

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Premature ejaculation is the most prevalent form of male sexual dysfunction, but its cause has not been well established. Recent studies have indicated that in men with premature ejaculation, penile sensitivity is increased. To investigate whether penile hypersensitivity is a cause of premature ejaculation, we prospectively evaluated the penile sensitivity of 18 patients with a lifelong history of premature ejaculation from the first coital experience and 15 controls, both in the flaccid and erect state. We used an SMV-5 vibrometer (Suzuki-Matsuoka, Teknologue, Tokyo, Japan), which automatically controls stimulatory strength; its precision and reproducibility are thus higher than analogue-type biothesiometers. At the styloid process of the ulna and medial maleolus of the tibia, there was no significant statistical difference in vibratory threshold between the two groups ($P > 0.05$). Also we did not find significant statistical differences in sensitivity of the glans penis, dorsum of the penile shaft, or frenulum of the penis between the two groups, in either the flaccid or erect state ($P > 0.05$). According to our results, penile hypersensitivity, as measured by an SMV-5 vibrometer, does not appear to be a major factor contributing to premature ejaculation.

Keywords: premature ejaculation; penile sensitivity; vibrometer

Introduction

Premature ejaculation is the most prevalent form of male sexual dysfunction, affecting an estimated 30% of men.[1,2] Premature ejaculation implies that due to an absence of voluntary control during sexual activity, ejaculation/orgasm occurs before the individual desires this. For many clinicians the time that elapses between vaginal penetration and ejaculation is the diagnostic criterion.[3] Some authorities define premature ejaculation as occurring when male orgasm occurs within 1 min of penetration, while others define it as ejaculation occurring before ten thrusts. At the other extreme, Kaplan defines it as the absence of voluntary control over the ejaculation reflex, disregarding time, the number of thrusts or orgasm by the partner.[3] The American Psychiatric Association's Diagnostic and Statistical Manual (DSM-IV) defines premature ejaculation as 'persistent or recurrence of ejaculation with minimal stimulation before, on, or shortly after penetration and before the person wishes it.'[4] Despite disagreement in the definition, there is no difficulty in recognizing and diagnosing premature ejaculation on the basis of a patient's history.

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Because premature ejaculation was traditionally believed to have a psychological etiology, treatment has typically involved a variety of behavioral or other psychotherapeutic modalities. Penile hypersensitivity was also proposed by some researchers as one cause of premature ejaculation,[5–7] but it was not proven to be a definite organic cause yet. Although application of a condom or local anesthetic or penile dorsal nerve ablation delays ejaculation, it does not necessarily follow that penile hypersensitivity is the cause of premature ejaculation. Most studies comparing penile sensitivity in premature ejaculation patients with that of a control group have been during the flaccid state, not the erect state. Ejaculation is a phenomenon that occurs during penile erection, which may change penile sensitivity; we therefore evaluated the penile sensitivity of premature ejaculation patients in the erect state. We used an SMV-5 vibrometer (Suzuki-Matsuoka vibrometer, Teknologue, Tokyo, Japan), which employs a digital system and automatically controls stimulatory strength. Therefore it provides higher precision and reproducibility than analogue type biothesiometers.[8,9]

Patients and methods

We prospectively investigated 18 patients who visited our clinic with a lifelong history of

premature ejaculation from the first coital experience, who satisfied the inclusion and exclusion criteria, and 15 healthy, sexually functional controls. In this study premature ejaculation was defined as involuntary ejaculation during foreplay or within 1 min of penetration. Patients were included if they had one sexual partner like a spouse and had sexual intercourse more than once a week. None of the patients had erectile dysfunction. Furthermore, patients with any risk factors for impotence, including hypertension, diabetes mellitus, neurologic disease, extensive alcohol use or the use of medication known to cause sexual dysfunction were excluded from the study. All men were screened medically and urologically. The mean age of 18 patients with premature ejaculation was 43.5 (range, 29–60 y), and that of 15 controls was 35.7 (range, 24–54 y).

All patients were evaluated by one examiner (Figure 1). Vibratory threshold recorded at the glans penis, penile shaft, and frenulum of the penis in the flaccid and erect state induced by intracavernous injection of PGE_1 10 μg. The patient was asked to inform the examiner of the first sensation of vibration as the amplitude of this changed; the test was repeated several times until the threshold value of a reproducible perception of vibration was obtained. As an internal control, sensory threshold was measured at the styloid process of the ulna and medial maleolus of the tibia.

Comparison of thresholds among groups was carried out using the *t*-test and Wilcoxon rank-sum test. And paired *t*-test was performed in order to assess differences in the flaccid state and erect state. A value of $P < 0.05$ was considered statistically significant. This study was approved by the institutional review board of Seoul National University Hospital.

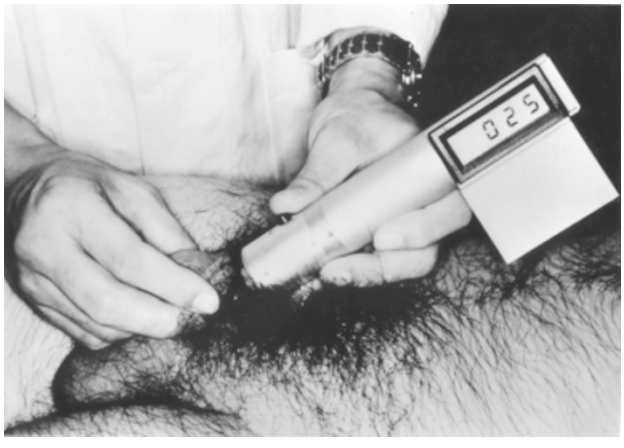

Figure 1 Evaluation of vibratory sensory threshold by SMV-5 vibrometer.

Results

Average vibratory thresholds in patients with premature ejaculation *vs* normal men are shown in Figure 2. There was no significant statistical difference between the two groups in vibratory threshold at the styloid process of the ulna or medial maleolus of the tibia ($P > 0.05$), nor did we find significant statistical differences in sensitivity of the glans penis, dorsum of the penile shaft, or frenulum of the penis between the two groups, in either the flaccid or erect state ($P > 0.05$) (Figure 2).

Average vibratory thresholds in the flaccid versus erect state are shown in Figure 3. There were no significant statistical difference between the flaccid and erect state in vibratory threshold at the styloid process of the ulna or medial maleolus of the tibia ($P > 0.05$), nor did we find significant statistical

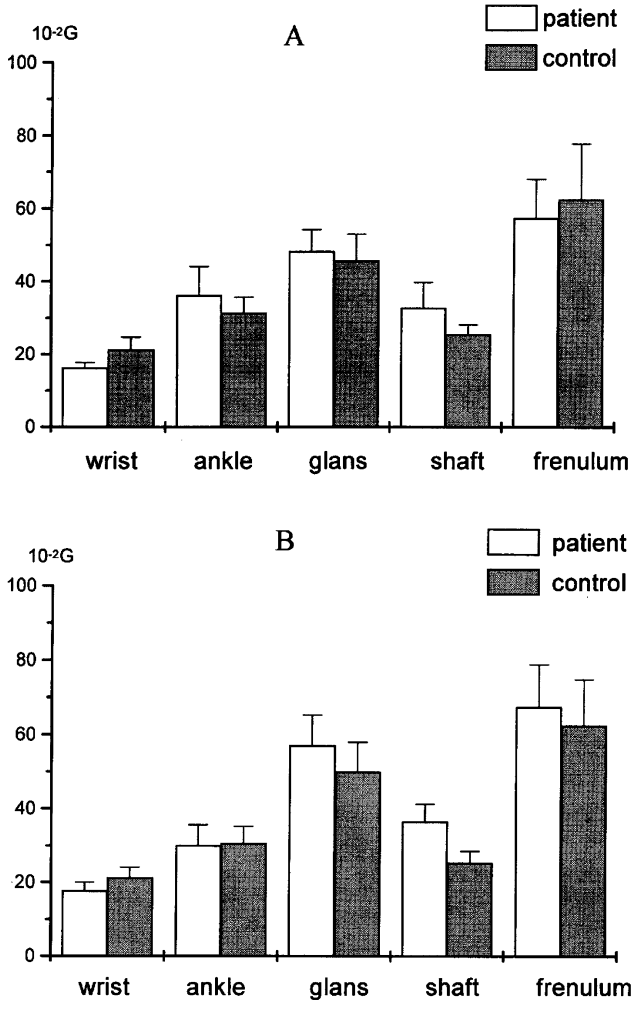

Figure 2 Comparison of penile sensitivity between patient and control group. (A) Comparison of patients group and control group in flaccid state ($P > 0.05$), (B) Comparison of patient group and control group in erect states ($P > 0.5$).

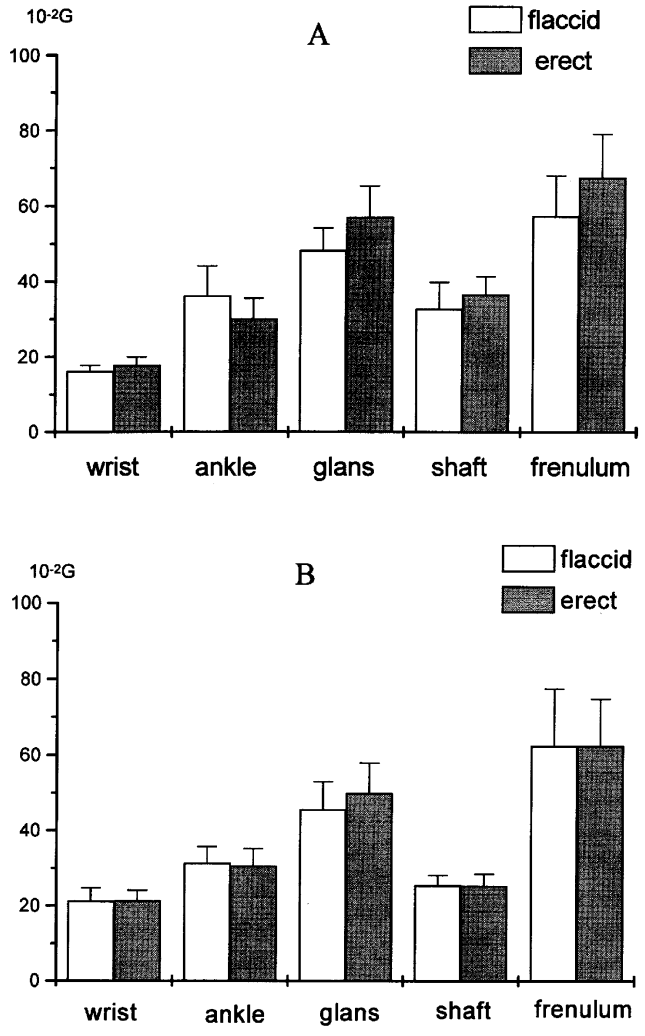

Figure 3 Comparison of penile sensitivity in flaccid and erect state. (A) patients group ($P > 0.05$), (B) control group ($P > 0.5$).

differences in sensitivity of the glans penis, dorsum of the penile shaft, or frenulum of the penis between the flaccid and erect state, in either the patient or control group ($P > 0.05$) (Figure 3).

Discussion

In most cases, the cause of premature ejaculation is unknown. Organic conditions such as urinary tract infection, prostatitis, alcoholism, diabetes mellitus, atherosclerosis and cardiovascular disease are not common causes of this disorder.[10] However, most authorities believe that men with this dysfunction have an underlying psychological problem of a performance anxiety type, a theory that is difficult to prove.[11,12]

Could an increase in penile sensitivity be a possible cause of premature ejaculation? A number of studies have attempted to attribute premature ejaculation to a simple organic cause-increased penile sensitivity,[5–7,13] although studies including neurological examinations (somatosensory evoked potential or biothesiometry, for example) have, as yet, reached no coherent conclusion. Xin *et al*[5] investigated 120 patients with premature ejaculation and 66 normal potent volunteers and concluded that patients with primary premature ejaculation had penile hypersensitivity. However, Rowland *et al*[7] showed significantly elevated threshold in men with erectile dysfunction, not in men with premature ejaculation. They have suggested that penile dermal hypersensitivity does not appear to be a major factor contributing to short ejaculation latencies and cognitive-affective factors may play the more salient role in this dysfunction. Ejaculation is a neural reflex stimulated by sensory input to the penis and mediated by smooth muscle and striated muscle contractions which produce seminal emission and expulsion. The afferent stimuli for the ejaculatory pathways are processed at the cerebral or spinal cord levels or both. It is conceivable that individual differences in the neurophysiological properties at any levels partially account for variance in the ease and speed of the ejaculatory reflex. Therefore, premature ejaculation would not be explained by a singular cause.

Studies on penile sensitivity have, until now, used a biothesiometer to provide a measurement of penile sensory threshold in the flaccid state. Biothesiometer is a simple, economical, and non-invasive instrument that can be utilized easily and is generally used in clinical research,[14] but it allows examiners' subjectivity to influence the measuring process. In addition, studies have been limited to evaluating penile sensitivity in the flaccid state, with no evaluation during erection. The erect state is likely to reflect the actual situation; if the cause of premature ejaculation is to be determined, the evaluation of sensitivity in the erect rather than the flaccid state therefore appears to be essential. Accordingly, to determine whether actual penile sensitivity increases in premature ejaculation patients, we measured penile sensory threshold during erect state and compared to that during the flaccid state. To measure this threshold we used the SMV-5 vibrometer, since this employs a digital system which automatically controls the degree of stimulation and therefore provides higher precision and reproducibility than analogue type biothesiometers.[8,9]

The results of this study showed that with regard to vibratory stimulation during both the flaccid and erect state, there were no significant statistical differences between the patient group and the control group. There might be some possible reasons for the negative results of this study: a small number of subjects and variation caused by age differences. But recruitment of the control group was very

difficult due to a precondition that in order to measure penile sensitivity during the erect state, artificial erection was to be induced by an injection in the penis. For the same reason age-matched control study was not performed. The mean age of controls was 8 y younger than that of patients. However, there was no statistical difference in age in both groups. From our experience in a large number of patients the test figures of penile sensitivity are in the broad range. Therefore even if a large number of control subjects were recruited, we would hardly expect significant statistical differences between the patient group and the control group.

Penile sensitivity was not significantly different between the flaccid and erect state. This lack of difference between the flaccid and erect state was contrary to our expectation. It is difficult to provide a precise explanation for such a result, though, Bemelmans *et al* contended that vibratory stimulation is inappropriate for penile sensory evaluation, and this seems a very reasonable explanation.[15]

According to our results, penile hypersensitivity does not appear to be a major factor contributing to premature ejaculation. Delaying ejaculation by decreasing penile sensitivity with a condom or local anesthetic is a method already in clinical use; this does not, however, imply that penile sensitivity increases in patients with premature ejaculation, since the method is also used by the men for whom premature ejaculation is not a problem. Furthermore penile sensitivity, evaluated by many researchers including ourselves, does not represent the sensory threshold that finally induces ejaculation. Little is known about the intensity of stimulus required to induce ejaculation, or the influence of the cerebral cortex on the ejaculatory reflex, though the peripheral neural pathway involved in ejaculation is fairly well understood. Many studies showed pharmacotherapy using selective serotonin reuptake inhibitor antidepressants for patients with premature ejaculation is very promising.[16–18] We believe further studies are needed especially about the mechanism of cerebral modification on the ejaculatory reflex.

Conclusions

Employing newly-developed instrument, the SMV-5 vibrometer, we compared penile sensitivity in premature ejaculation patients to that in a normal control group. During the flaccid as well as the erect state, differences of sensitivity of the glans, penile shaft, and frenulum of the penis between the two groups were not observed. In addition, significant changes in penile sensitivity during the flaccid and erect state were not observed in either the patient group or control group.

In order to investigate the organic causes of premature ejaculation, continuing psychological and neurological research is needed, along with studies of the action mechanisms of effective drugs such as selective serotonin reuptake inhibitors.

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