Objective: While detrusor-sphincter dyssynergia (DSD) occurs in conjunction with lesions between the brainstem and the sacral cord, it is not well known whether sacral/peripheral lesions contribute to DSD. We studied the relationship between DSD and sacral/peripheral lesions.

Methods: One hundred and forty-four patients with diverse neurologic etiologies underwent urodynamic study and analysis of motor unit potentials in the external sphincter muscles, 117 of whom were able to void during a urodynamic test. Sacral/peripheral lesion (SPL) is defined as neurogenic change in motor unit potentials. Detrusor overactivity (DO) is defined as involuntary detrusor contractions during the filling phase, which commonly occurs in lesions above the sacral cord. We considered DO as a putative indicator of supra-sacral lesion.

Results: DSD was found in 44 (30.6%), SPL in 71 (49.3%), and DO in 83 (57.6%) of 144 patients, respectively. The incidence of DSD was the same in the SPL positive group (31%) and the SPL negative group (30.1%). By contrast, within the subgroup of patients without DO, the incidence of DSD was significantly more common in the SPL positive group (41.4%) than in the SPL negative group (25.0%) (P < 0.05). In 53 of the SPL positive group who were able to void, postvoid residual >100 mL was more common in patients with DSD (not statistically significant).

Conclusion: The results of the present study suggest that not only suprasacral pathology, but also sacral/peripheral lesions can produce DSD. In light of the previous reports, DSD might also result from partial lesions in peripheral branches of the sphincter circuit.

Key words: detrusor-sphincter dyssynergia, neuropathy, sacral Onuf’s nucleus.

1. INTRODUCTION

Detrusor-sphincter dyssynergia (DSD) is a condition of detrusor contraction concurrent with an involuntary contraction of the urethral and/or periurethral striated muscle.1 DSD indicates disturbed coordination of pudendal and pelvic nerve function, and is one of the major causes of disturbed evacuation in neurologic patients.2 DSD typically occurs in conjunction with lesions between the brainstem structure and the sacral spinal cord.3,4 By contrast, it is not well known whether sacral/peripheral lesions might contribute to DSD.5,6 We herein studied the relationship between DSD and sacral/peripheral lesion.

2. METHODS

One hundred and forty-four patients underwent both electromyography (EMG)-cystometry and analysis of motor unit potentials in the external anal sphincter muscles at our urodynamic laboratory. Most patients were referred from the neurology department and had either central (stroke, Parkinson’s disease, multiple sclerosis) or peripheral (lumbar spondylosis, diabetic neuropathy) neurologic etiologies. The patients were 71 men and 73 women with a mean age of 65.5 years, ranging from 14 to 87 years. We performed standard EMG-cystometry by urodynamic computer, using the methods and definitions of the International Continence Society.1 After free flowmetry, we measured postvoid
residual by transurethral catheterization, and the volumes were regarded normal at <30 mL. We performed standard electromyography (EMG)-cystometry (medium-fill [50 mL/min], liquid [saline]) using a urodynamic computer (Urovision; Lifetech Inc., Houston, TX, USA) and an EMG computer (Neuropack M2; Nihon Kohden Inc., Tokyo, Japan). Normal values for first sensation and bladder capacity are estimated as 100–300 and 200–600 mL in our urodynamic laboratory. We performed pressure-flow analysis in all subjects using Schäfer’s nomogram. One hundred and seventeen of 144 patients were able to void. Regarding the difficulty diagnosing DSD, it is not standardized and there is still a subjective aspect, but steps were taken to exclude EMG activity elicited by movement or straining as follows. If a patient could not contract the bladder at all, or if they strained, we did not diagnose DSD. Even in patients who were unable to void, if the patient had voiding detrusor contraction together with augmented sphincter EMG activity, we diagnosed the patient with DSD, only if they did not strain. Detrusor overactivity (DO) is defined as involuntary detrusor contractions during the filling phase, which commonly occurs in lesions above the sacral cord. After inserting a concentric needle electrode in the external anal sphincter muscles, we performed single-motor unit potential analysis using an EMG computer. We sampled at least 10 single motor unit potentials per patient, manually examining the automatically sampled waves to assure single motor unit potentials. Sacral/peripheral lesions (SPL) are defined as the presence of neurogenic change in the motor unit potentials, which was diagnosed when at least one of the following abnormalities was seen: (i) a percentage of motor unit potentials with a duration >10 msec greater than 20%, or (ii) an average motor unit potentials duration >10 msec, particularly including the late components. All patients gave informed consent before entering the study. Statistical analysis was performed using Student’s t-test.

3. RESULTS

DSD was found in 44 (30.6%), SPL in 71 (49.3%), and DO in 83 (57.6%) of the 144 patients, respectively. The patterns/forms of DSD in SPL were not different from those usually seen in suprasacral lesions, e.g., sphincter EMG persisted involuntarily, which normally disappears completely during detrusor contraction on voiding (Fig. 1). The incidence of DSD was the same in the SPL positive group (22 of 71, 31%) and the SPL negative group (22 of 73, 30.1%). By contrast, within the subgroup of

---

**Fig. 1** Typical trace of DSD in sacral/peripheral lesion. DSD, detrusor-sphincter dyssynergia; Q, uroflow (mL/sec); Pves, vesical pressure (cmH2O); Pabd, abdominal pressure (cmH2O); Pdet, Pves-Pabd; detrusor pressure (cmH2O); EMG, sphincter electromyography.
patients without DO during the filling phase, the incidence of DSD was significantly more common in the SPL positive group (12 of 29, 41.4%) than in the SPL negative group (8 of 34, 25.0%) (P < 0.05). In 53 of the SPL positive group who were able to void, maximum urinary flow <10 mL/sec was the same in 5/14 (35.7%) with DSD and 15/39 (38.5%) without DSD. By contrast, postvoid residual urine >100 mL was more common in 10/14 (71.4%) with DSD than 20/39 (51.3%) without DSD (not statistically significant).

4. DISCUSSION

DSD is a condition of detrusor contraction concurrent with an involuntary contraction of the sphincter.1 DSD commonly occurs in supra-sacral lesion (e.g. between the brainstem and the sacral cord).2 Whereas detailed mechanism of DSD remains uncertain, in cerebral lesion, DSD is significantly frequent in patients with disturbed deep sensation and pyramidal signs, indicating that dorsal and lateral spinal pathways may play a role.3 Gamma-amniobutyric acid (GABA), opioids, glycine, and nerve growth factors appear to be involved in DSD due to spinal cord lesions.4–7 By contrast, in the present study, to clarify the relationship between DSD and sacral/peripheral lesion, we considered DO as a putative indicator of DSD (not statistically significant).

The underlying mechanism of the ‘sacral/peripheral-type’ DSD remains obscure. DSD is a motor phenomenon. Peripheral branch of the neural circuit controlling the external sphincter are afferent sensory fibers and pudendal efferent fibers. Partial peripheral nerve lesions (e.g. painful neuropathy) may lead to decrease in pain sensation as well as spontaneous numbness and pain.8 A rat model of neuropathic pain showed spontaneous nerve firing.9 In contrast to sensory disorders, little is known about the role of peripheral nerve lesions in the development of motor disorders such as dystonia. However, Yamaguchi et al. reported that diabetic cystopathy showed not only bladder weakness, but also DO, although the frequency of DO in this condition is uncommon.10 It appears that experimental diabetes induces irritation of the pelvic nerve fibers or ephaptic transmission, due in part to intra-neuronal metabolic changes and impaired axonal transport, particularly of nerve growth factors in experimental diabetes.11,12 Further, experimental lesions of the sciatic and tibial nerves led to clawed postures in rats.13 Animal models showed that this process may also influence spinally mediated motor behaviors, such as withdrawal reflexes.14

A limitation of the study was that whereas SPL in our patients was strictly determined by the presence of neurogenic change in sphincter EMG, absence of DO does not completely exclude upper motor neuron lesion. However, in light of the present study findings, sacral/peripheral lesions might also affect peripheral branch of the sphincter circuit by nerve irritation or ephaptic transmission, possibly leading to DSD.

5. CONCLUSION

The results of the present study suggest that not only suprasacral pathology, but also sacral/peripheral lesions can produce DSD. In light of the previous reports, DSD might also result from partial lesions in peripheral branch of the sphincter circuit.

Disclosure

All authors have no conflict of interests.

REFERENCES