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# DO SACRAL/PERIPHERAL LESIONS CONTRIBUTE TO DETRUSOR-SPHINCTER DYSSYNERGIA?

Hypothesis / aims of study
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.

Study design, materials and methods
We had 144 patients with diverse neurologic etiologies, who 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 the 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 our 144 patients, respectively. The incidence of DSD was the same in SPL positive group (31%) and SPL negative group (30.1%). By contrast, within the subgroup of patients without DO, the incidence of DSD was significantly more common in SPL positive group (41.4%) than SPL negative group (25.0%) ($p<0.05$). In 53 of SPL positive group who were able to void, post-void residual >100 ml was more common in patients with DSD (not statistically significant).

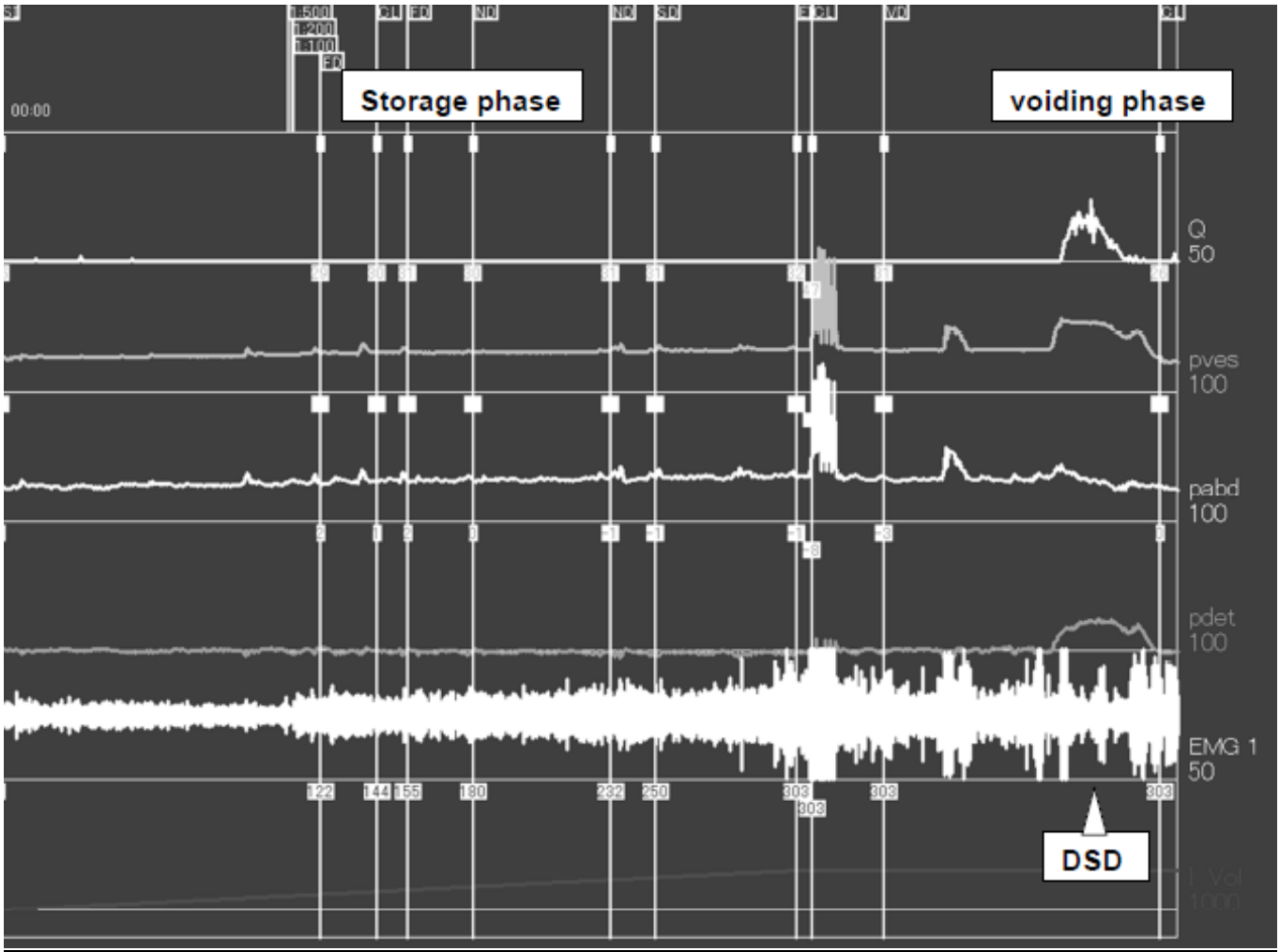


**Figure 1 Typical trace of DSD in sacral/peripheral lesion.**
DSD: detrusor-sphincter dyssynergia. Q: uroflow (ml/sec), Pves: vesical pressure ($cmH_2O$), Pabd: abdominal pressure ($cmH_2O$), Pdet: Pves- Pabd, detrusor pressure ($cmH_2O$), EMG: sphincter electromyography.

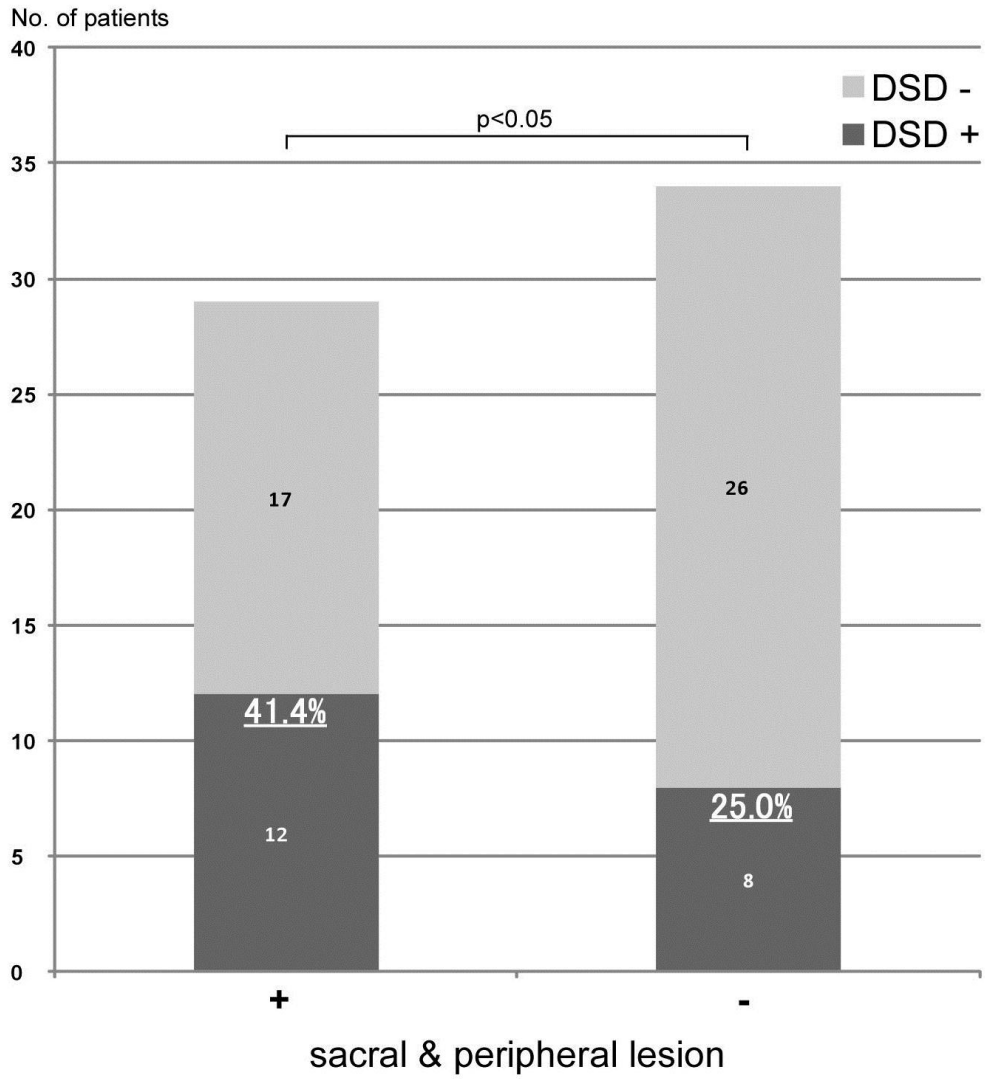


**Figure 2 Relation between detrusor-sphincter dyssynergia with sacral/peripheral lesion in a subgroup of patients without detrusor overactivity.**
The incidence of detrusor-sphincter dyssynergia (DSD) was significantly more common in sacral/peripheral lesion (SPL) positive group (12 of 29, 41.4%) than SPL negative group (8 of 34, 25.0%) ($p<0.05$).

Interpretation of results

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 etc., may lead to decrease in pain sensation as well as spontaneous numbness and pain[1]. A rat model of neuropathic pain showed spontaneous nerve firing. It also 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[2]. Further, animal models showed that this process may influence spinally mediated motor behaviors such as withdrawal reflexes[3].

Concluding message

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.

References

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Disclosures

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