Relation between flexor spasms, uninhibited detrusor contractions and anal sphincter activity

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SUMMARY The time relation between flexor spasms, detrusor contractions and anal sphincter activity was recorded in a consecutive series of 111 patients with hyperreflexic bladder and flexor spasms. In 76 of the patients flexor spasms were preceded by detrusor contractions. The opposite pattern, namely detrusor contractions preceded by flexor spasms, was seen in only nine patients. The anal sphincter activity was generally increased in relation to detrusor contractions in patients with spinal lesions but decreased in patients with cerebral lesions. The treatment of flexor spasms and uninhibited detrusor contractions is discussed on the basis of this mutual relationship.

The flexor reflex and associated flexor spasms, which are involuntary contractions in the flexor muscles,1,2 can be increased by lesions of suprasegmental levels in the central nervous system owing to disinhibition. In severe cases flexor withdrawal spasms, often seen in patients with complete upper motor neuron lesions, can facilitate development of contractures. In less pronounced cases flexor spasms may impair mobilisation and can be painful. In this group of patients hyperreflexic bladder is also seen. Hyperreflexic bladder is characterised by uninhibited detrusor contraction at subnormal filling of the bladder resulting in frequency, urgency and urge incontinence.3

The striated sphincters are functionally interrelated with the bladder and the lower limb flexor muscles.4–6 This paper presents our studies of the mutual relationship between flexor spasms, uninhibited detrusor contractions and activity in the anal sphincter.

Material and methods

The material was selected from a group of patients with neurological disorder and distinct voiding dysfunction admitted to the Department of Neurology, Aarhus Kommunehospital, Aarhus in the period of 1 January 1981 to 1 December 1983.

The uninhibited detrusor contractions were demonstrated by cystometry using a two-way balloon catheter introduced via the urethra into the bladder. Isotonic saline was infused at the rate of 20 ml per minute and intravesical and intrarectal pressures were continuously recorded. The infusion was stopped during uninhibited contractions to allow isometric conditions. EMG from the anal sphincter was performed by a unipolar needle electrode. Flexor spasms were recorded by their EMG from the tibialis anterior muscle by surface electrodes. The electromyograms were recorded on a multi pen recorder together with the volume of saline infused into the bladder, intravesical pressure, intrarectal pressure and the electronically calculated difference between the two pressures.

One hundred and seventy four patients had detrusor hyperreflexia according to the international criteria based on cystometry,7 but 63 of these patients had no flexor spasms and were therefore excluded. The remaining 111 patients, 57 men and 54 women, had a mean age of 56 ranging from 12 to 76 years. Urge incontinence was reported in 103 patients and in eight patients voiding disturbances were reported as urge and/or voiding disability. In two patients voiding disturbances were only a minor part of the illness but in the remaining patients these disturbances were important or predominant. Twenty three patients had cerebral lesions and 18 had spinal lesions as shown in the table. Seventy patients had multiple sclerosis; the disability scores being shown in fig 1. This rating system has been compared to other systems in a previous report.8

Results

Detrusor contractions preceded flexor spasms in 76 of the 111 patients as illustrated by fig 2. This order was reproduced at each detrusor hyperreflexia in 63% of the patients. In 35 of these 76 patients flexor spasms were also recorded without associated detrusor contractions but during bladder contractions these flexor spasms were increased in 12 patients or became synchronous with bladder contractions in the remaining 23 patients.

In four of the 111 patients detrusor contractions
were followed by relaxation of flexor spasms. Flexor spasms preceded detrusor contractions in nine patients, (fig 3) one of whom also demonstrated detrusor contractions without flexor spasms. In 18 patients no fixed pattern was presented and in the last four patients anal sphincter spasms preceded detrusor contractions and flexor spasms, (fig 4).

EMG activity was unchanged or decreased during uninhibited detrusor contractions in 37 patients as shown in fig 2. Three of these patients also showed relaxation some seconds before the detrusor contractions as illustrated in fig 5. The same figure shows decreased sphincter activity with clonic pattern interspersed throughout the detrusor contractions. All in all this pattern was found in 32 patients.

In 15 of the 18 patients who had lesions only in the spinal cord, sphincter EMG activity was increased throughout detrusor contractions, whereas all 23 patients with cerebral lesions showed relaxation during detrusor contractions. The multiple sclerosis patients contributed to both groups. Further it should be mentioned that in the period of observation five patients without detrusor contractions demonstrated an increase by more than 100% of flexor spasms during bladder filling.

**Discussion**

The present investigation demonstrated that, in the majority of the cases, flexor spasms were preceded by detrusor contractions but the opposite pattern, where flexor spasms were followed by detrusor contractions,
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Fig 3 Flexor spasms preceding detrusor contractions. Increased sphincter activity during bladder contraction.

Fig 4 Anal sphincter spasms preceding detrusor contractions and flexor spasms.

Fig 5 Sphincter relaxation preceding detrusor contraction which again precedes flexor spasms. Clonic pattern of sphincter activity interspersed throughout bladder contraction.


14. deGroat WC, Ryall RW. Reflexes to sacral parasym-
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