The purpose of this chapter is to review the peripheral and central effects of excitation of efferent neurons innervating the lower urinary tract. The anatomy was reviewed in Section I, and our main concern in this chapter will be with the physiology and pharmacology of ganglionic and/or neuro-effector transmission in sympathetic, parasympathetic and somatic efferent pathways. There are many excellent reviews on this subject and the reader may find monographs by Appenzeller (1982), Gabella (1976), Nilsson (1983) and Johnson and Spalding (1974), and reviews by Burnstock (1981, 1986), Morrison (1982), de Groat et al. (1981), de Groat (1975) and Andersson and Sjögren (1982) helpful for further study. The physiology of smooth muscle and of neuromuscular transmission in the bladder is dealt with in Chapter 6. However, the starting point of this chapter will deal with an effect which appears to be mediated by parasympathetic efferent neurons within the central part of their course, viz. the phenomenon of recurrent inhibition.

Preganglionic Parasympathetic Efferents

Recurrent Inhibition in the cord

In 1968, de Groat and Ryall observed that bladder contractions in the cat could be inhibited by stimulation of the central cut end of a sacral ventral root; the firing of contralateral preganglionic parasympathetic neurons whose discharges correlated with the occurrence of bladder contractions was also depressed. The hypothesis was that the depression of activity in the parasympathetic nerves to the bladder, and of bladder motility, was due to a recurrent inhibitory pathway, similar to that involved in the inhibition of somatic motoneurons; axon collaterals of parasympathetic efferent neurons, however, have not been detected in labeling experiments (Nadelhaft et al. 1980), and axons projecting into the cord via the ventral roots have...
been regarded as ventral root afferents rather than recurrent axon collaterals by Morgan et al. (1981). The anatomical basis for this phenomenon is therefore unclear. The recurrent inhibition of α-motoneurons is known to be mediated by recurrent axon collaterals synapsing on Renshaw cells (Eccles et al. 1954) and differs in a number of aspects from the inhibition observed by de Groat and Ryall (1968). Unlike the recurrent inhibition of somatic motoneurons, the effects of ventral root stimulation on bladder motility were prolonged, and often lasted for several minutes.

The inhibitory effects of ventral root stimulation were seen only when the central cut end of a ventral root which contained parasympathetic efferent fibres was stimulated at intensities which excited these or other fibres of similar diameter, and at rates greater than 10 s⁻¹. The effects were crossed, being relayed to the bladder via the parasympathetic efferents on the opposite side of the cord, and were seen in chronic spinal animals as well as chloralose-anesthetized and decerebrate preparations. The phenomenon was still present when the bladder was deafferented, so the effects were not due to ventral root afferents (de Groat 1976). The recurrent inhibition was seen following section of the hypogastric nerves; the conclusion that it was not dependent on sympathetic efferent pathways may not be absolutely valid, because about 10% of the sympathetic fibres running to the pelvic viscera travel in the pelvic nerves (see Chap. 3, p. 79), and in addition, the ventral roots may contain sympathetic efferent fibres that innervate pial blood vessels and possibly other structures (Risling et al. 1984).

Recurrent inhibitory effects (Fig. 5.1) were most effective at low bladder pressures, and were relatively weak when afferent inputs from the bladder were activated by higher levels of intravesical pressure; in contrast, high bladder pressures did not influence the firing of Renshaw cells. These recurrent inhibitory effects were explained by the presence of interneurons in the sacral autonomic cell columns that received recurrent axon collaterals from the parasympathetic efferents. de Groat and Ryall (1968) recorded from some cells which were synaptically activated by antidromic stimulation of the parasympathetic efferents in the ventral roots, at rather longer latencies than were observed in Renshaw cells, as one might expect from the low conduction velocities of the parasympathetic axons. They fired at high rates of discharge (600–800 spikes per second) for the first few spikes; the maximum number of spikes in a burst was seven, and the duration of the burst was 10–80 ms. This discharge is not as marked as seen in Renshaw cells in the same animals. These neurons followed frequencies of up to 40 per second. The interneurons that could be synaptically activated from a ventral root were not excited by stimulation of adjacent or contralateral ventral roots, but could be fired by stimulation of various ipsilateral leg nerves. Thus some form of somatovisceral convergence is seen even in this system. The recurrent inhibition was unaffected by intravenous injection of dihydor-β-erythrodine, which blocks the antidromic activation of Renshaw

![Pelvic nerve stimulation](image)

Fig. 5.1a,b. Recurrent inhibition of spontaneous bladder contractions by stimulation of the central end of a transected pelvic nerve. Stimulation applied at a frequency of 20 Hz during period indicated by bar. a Obtained with ipsilateral sacral dorsal roots transected, but the ventral roots intact; b after transection of the ipsilateral sacral ventral roots. (de Groat 1976)