The bladder has somatic, parasympathetic, and sympathetic innervation. In other words, it has input from higher centres (brain) provided that the pathways via the spinal cord are undamaged, and also autonomic control.

The **pudendal nerve** is the somatic component of bladder innervation and innervates the external sphincter. When stimulated, it produces contraction of the external urethral sphincter, which is only able to remain tightly contracted for a short period of time.

This sphincter normally contracts with transient increases in abdominal pressure such as when there is coughing sneezing, and laughing.

The parasympathetic nerve fibers arise from sacral segments S2-4, innervating the detrusor muscle, when the individual desires micturition.

When stimulated by a need to empty the bladder (voluntary control), the detrusor contracts resulting in raised pressure within the bladder.

The internal urinary sphincter is innervated by the sympathetic nervous system, nerves originating from the thoracolumbar region. When stimulated, the internal sphincter relaxes.

As urine fills the bladder via the ureters (tubes which come down from the kidneys), the bladder wall muscle (detrusor) stretches allowing the bladder to expand; as the bladder fills, stretch receptors within the bladder wall are stimulated, sending the brain information as to the amount of urine in the bladder.

Approximately 300 cc of urine within the bladder is necessary before the pressure within the

bladder rises enough for the brain to recognize a sense of bladder fullness. With low bladder volumes, the sympathetic nervous system is stimulated and parasympathetic system is inhibited resulting in internal sphincter contraction and detrusor relaxation.

When the bladder is full and micturition is desired, the inhibitory signals from the brain are replaced by impulses which stimulate the parasympathetic system resulting in detrusor contraction, and inhibit the sympathetic system resulting in internal sphincter relaxation.

The bladder pressure then rises to a point at which it exceeds the resistance within the urethra (the tube through which the urine leaves the bladder), and urine flows out.

Once the bladder has emptied, the brain again sends impulses restoring parasympathetic inhibition and sympathetic stimulation resulting in detrusor relaxation and internal sphincter contraction.

A severe lesion in the **cauda equina** (at the lower end of the spinal cord) affects bladder and bowel function as a result of damage to the **sacral parasympathetic outflow**. The detrusor muscle of the bladder is affected and reflex connection no longer occurs in response to distension.

The bladder wall itself has a certain amount of elasticity, and with rising bladder pressure this forces some urine into the urethra.

However, the unopposed sympathetic supply to the sphincter muscle keeps it contracted and closed, and dribbling incontinence occurs. A similar situation arises with regard to the bowel and anal sphincter.

Damage further up the spinal cord , in which voluntary control of the bowel and bladder is affected, but which does not affect the parasympathetic outflow leads to an automatic bladder.

Any voluntary aspect to the control of the bladder is lost, leading to "accidents".

Note that certain medications used in arachnoiditis patients may affect bladder function.

For instance, antidepressants such as amitriptyline have anticholinergic effects, inhibiting detrusor muscle function.

This may lead to some difficulty in initiating urination and possibly in emptying the bladder fully.

Further details on bladder problems are available in other articles available from the Arachnoiditis Support Groups