

Uroepithelial dysfunction - OAB

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Pathophysiology of OAB

- The myogenic hypothesis
- The neurogenic hypothesis
- The afferent mechanisms hypothesis
 - Urothelial cells possess sensory and signaling properties that allow them to respond to their chemical and physical environments and to communicate with subjacent structures

*Decreased capacity
to handle
afferent information*



*Decreased suprapontine
inhibition*

*Mirabegron
Tadalafil
BoNT/A*



*Increased afferent
activity*

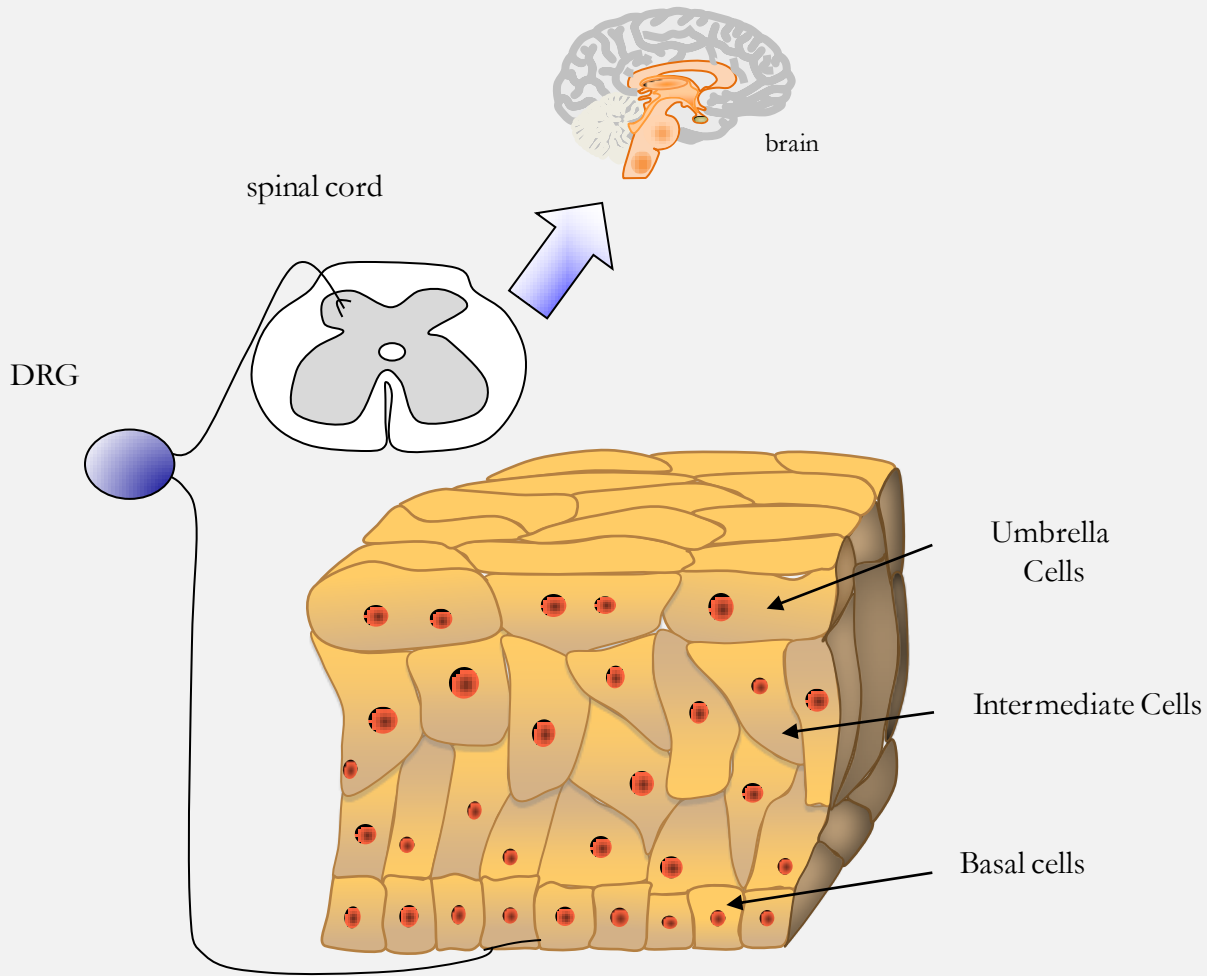


*Myogenic activity and
influence of local factors
Urothelial signaling*

Urothelium - multifunctional tissue

- not only acts as a barrier
- but also acts as a sensory organ by transducing physical and chemical stresses to the attendant afferent nervous system and underlying smooth muscle

Sensory Function

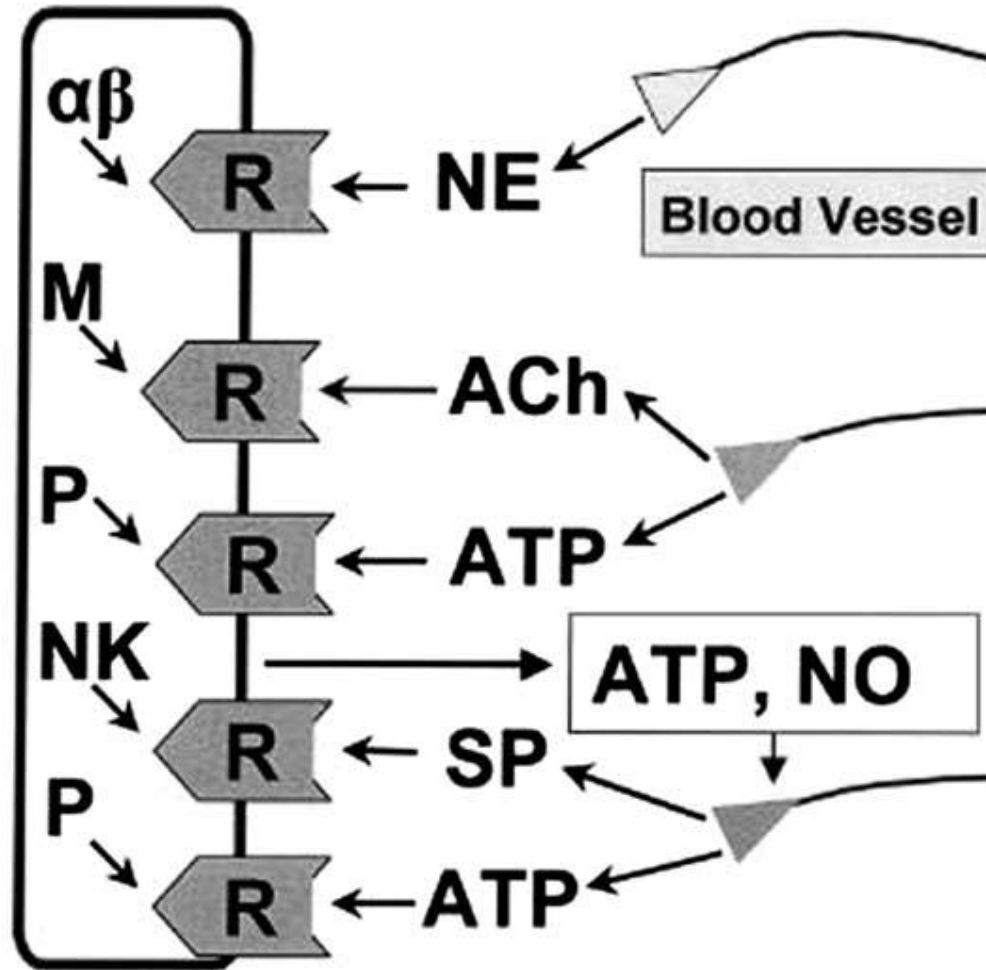


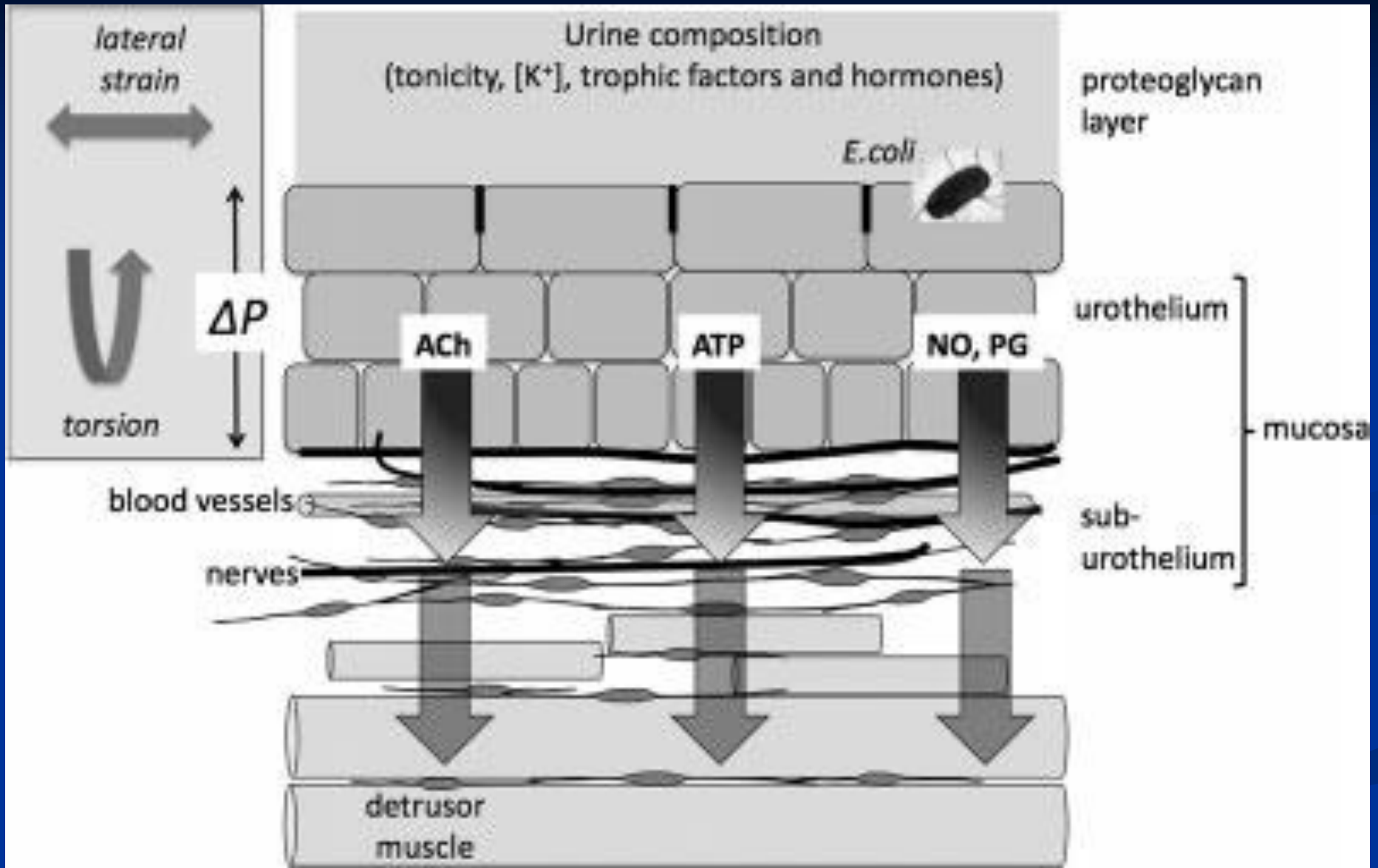
Urothelium

Adrenergic Neuron

Cholinergic Neuron

Sensory Neuron





Muscarinic mechanisms for afferent activation

- Antimuscarinics block MR on the efferent in the detrusor muscle and reduce the ability of the detrusor to contract
- Dose recommended for treatment of OAB, there is little evidence for a significant reduction of the voiding contraction

Urothelium-related mechanisms

- Urothelium release signaling molecules (ATP, prostaglandins, etc.)
→ activate C-fiber afferent in the suburothelial layer
- Suburothelial layer of myofibroblasts (interstitial cells) → close appositions to unmyelinated nerves (afferent C-fiber nerves)