Parasympathetic N.S.

Sympathetic N.S.
Figure 9.35
The preganglionic fibers of the parasympathetic division of the autonomic nervous system arise from the brain and sacral region of the spinal cord.
Parasympathetic and sympathetic pathways to the ciliary muscle. The major innervation to the ciliary muscle is parasympathetic and follows the pathway shown by the thick solid bold lines. The parasympathetic pathway originates in the Edinger-Westphal nucleus and courses with the third nerve, whence the fibers travel to and synapse in the ciliary ganglion. The majority of the postganglionic parasympathetic fibers travel to the ciliary muscle via the short ciliary nerves, but some postganglionic fibers (double asterisk) also travel with the long ciliary nerves. There is also evidence for a direct pathway of uncertain functional significance (single asterisk) to the internal eye structures from the Edinger-Westphal nucleus. The sympathetic supply to the ciliary muscle (thin solid lines) originates in the diencephalon and travels down the spinal cord to the lower cervical-upper thoracic segments, to synapse in the spinociliary center of Budge in the intermediolateral tract of the cord. From there second-order nerves leave the cord by the last cervical and first two thoracic ventral roots; these preganglionic fibers run up the cervical sympathetic chain to synapse in the superior cervical ganglion. The third-order fibers continue up the sympathetic carotid plexus and enter the orbit with the first division of the trigeminal nerve (following the nasociliary division) or independently, whence they join the long and short ciliary nerves, in the latter instance passing through the ciliary ganglion without synapsing. (Adapted from Duke-Elder S, in System of ophthalmology, vol XII, St Louis, CV Mosby, 1971, pp 600, 602; Parulman JJ, Fay MT, Burde RM, Trans Am Ophthalmol Soc 82:371, 1984; Warwick R, in Eugene Wolff's anatomy of the eye and orbit, Philadelphia, WB Saunders, 1976, pp 308-312.)
Figure 12

Schematic illustration of a generalized cholinergic junction (not to scale). Choline is transported into the presynaptic nerve terminal by a sodium-dependent carrier (A). This transport can be inhibited by hemicholinium drugs. ACh is transported into the storage vesicle by a second carrier (B) that can be inhibited by vesamicol. Peptides (P), ATP, and proteoglycan are also stored in the vesicle. Release of transmitter occurs when voltage-sensitive calcium channels in the terminal membrane are opened, allowing an influx of calcium. The resulting increase in intracellular calcium causes fusion of vesicles with the surface membrane and exocytotic expulsion of ACh and cotransmitters into the junctional cleft. This step is blocked by botulinum toxin. Acetylcholine's action is terminated by metabolism by the enzyme acetylcholinesterase. Receptors on the presynaptic nerve ending regulate transmitter release.
(a) A catechol group and (b) the catecholamine neurotransmitters.

The synthesis of catecholamines from tyrosine. The catecholamine neurotransmitters are in boldface type.
Schematic diagram of a generalized noradrenergic junction (not to scale). Tyrosine is transported into the noradrenergic ending or varicosity by a sodium-dependent carrier (A). Tyrosine is converted to dopamine (see Figure 6–5 for details), which is transported into the vesicle by a carrier (B) that can be blocked by reserpine. The same carrier transports norepinephrine (NE) and several other amines into these granules. Dopamine is converted to NE in the vesicle by dopamine-β-hydroxylase. Release of transmitter occurs when an action potential opens voltage-sensitive calcium channels and increases intracellular calcium. Fusion of vesicles with the surface membrane results in expulsion of norepinephrine, cotransmitters, and dopamine-β-hydroxylase. Release can be blocked by drugs such as guanethidine and bretylium. After release, norepinephrine diffuses out of the cleft or is transported into the cytoplasm of the terminal (uptake 1 [1]), blocked by cocaine, tricyclic antidepressants or into the postjunctional cell (uptake 2 [2]). Regulatory receptors are present on the presynaptic terminal.
The nicotinic acetylcholine receptor, a ligand-gated ion channel. The receptor molecule is depicted as embedded in a rectangular piece of plasma membrane, with extracellular fluid above and cytoplasm below. Composed of five subunits (two $\alpha$, one $\beta$, one $\gamma$, and one $\delta$), the receptor opens a central transmembrane ion channel when acetylcholine ($ACh$) binds to sites on the extracellular domain of its $\alpha$ subunits.
Transmitter actions at G-protein-coupled receptors. The binding of neurotransmitter to the receptor leads to activation of G-proteins. Activated G-proteins activate effector proteins, which may be (a) ion channels, or (b) enzymes that generate intracellular second messengers.
**Modulation by the NE β receptor.** (1) The binding of NE to the receptor activates a G-protein in the membrane. (2) The G-protein activates the enzyme adenylyl cyclase. (3) Adenylyl cyclase converts ATP into the second messenger cAMP. (4) cAMP activates a protein kinase. (5) The protein kinase causes a potassium channel to close by attaching a phosphate group to it.