UNIVERSITAS MUHAMMADIYAH JAKARTA
FAKULTAS KEDOKTERAN DAN KESEHATAN

SURAT TUGAS
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Untuk membuat **Penyusunan Rencana Pembelajaran Semester Blok 1.3 Siklus Hidup Manusia 1.**
Demikian surat tugas ini diberikan kepada yang bersangkutan untuk dilaksanakan sebagai amanah.

Jakarta, 30 Oktober 2022
Dekan Fakultas Kedokteran dan Kesehatan UMJ

Dr. dr. Muhammad Fachri, Sp.P, FAPSR, FISR
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SUPLEMEN FARMAKOLOGI SHM1 MODUL 3
PELEMAS OTOT
Rina Nurbani

Comparison of Sympathetic, Parasympathetic, and Motor Nerves Differences

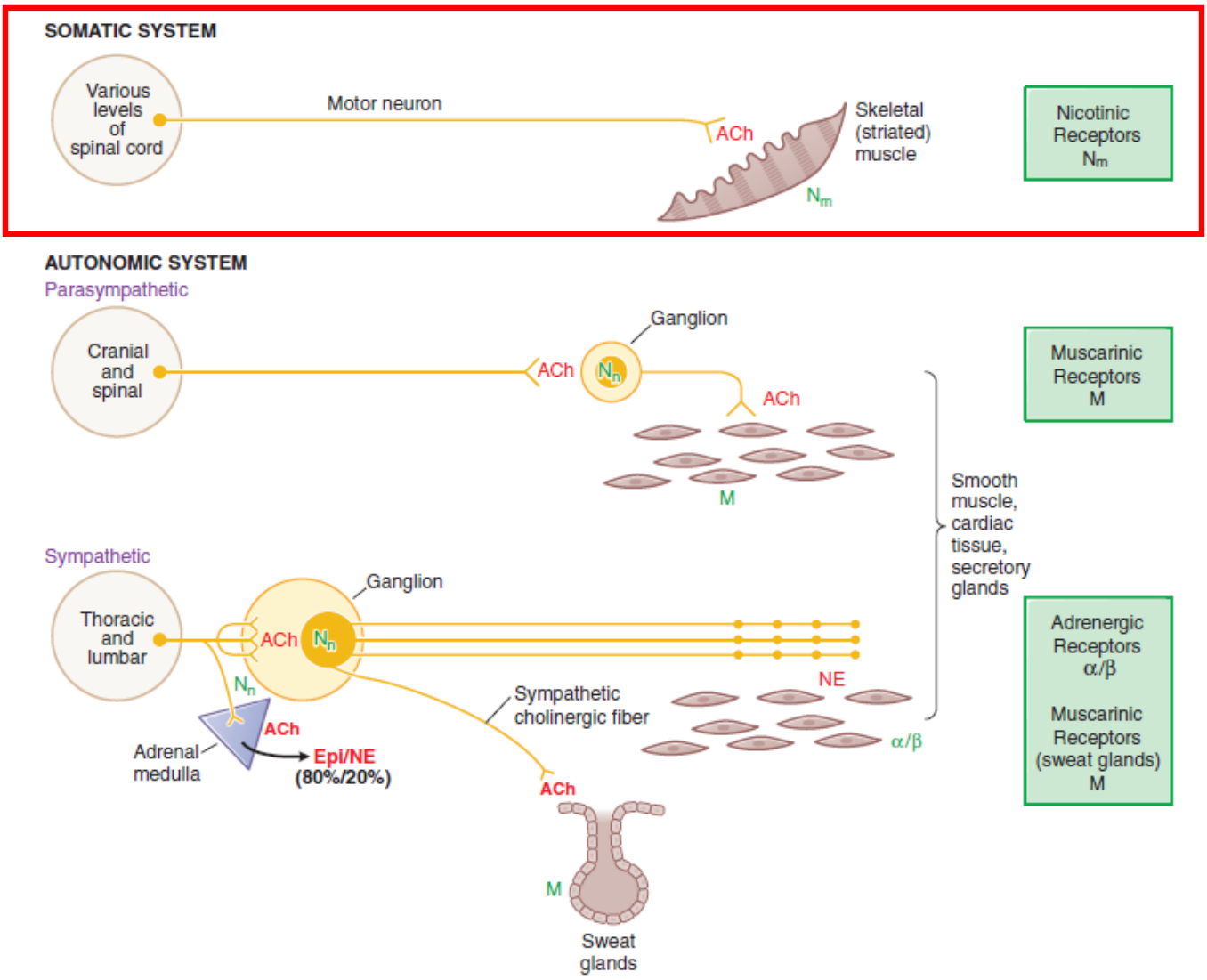

Figure 8–2 *Comparative features of somatic motor nerves and efferent nerves of the autonomic nervous system.* The principal neurotransmitters, ACh and NE, are shown in *red*. The receptors for these transmitters, nicotinic (N) and muscarinic (M) cholinergic receptors, α and β adrenergic receptors, are shown in *green*. Somatic nerves innervate skeletal muscle directly at a specialized synaptic junction, the motor end plate, where ACh activates N_m receptors. Autonomic nerves innervate smooth muscles, cardiac tissue, and glands. Both parasympathetic and sympathetic systems have ganglia, where ACh is released by the preganglionic fibers; ACh acts on N_n receptors on the postganglionic nerves. ACh is also the neurotransmitter at cells of the adrenal medulla, where it acts on N_n receptors to cause release of EPI and NE into the circulation. ACh is the dominant neurotransmitter released by postganglionic parasympathetic nerves and acts on muscarinic receptors. The ganglia in the parasympathetic system are near or within the organ being innervated, with generally a one-to-one relationship between pre- and postganglionic fibers. NE is the principal neurotransmitter of postganglionic sympathetic nerves, acting on α or β adrenergic receptors. Autonomic nerves form a diffuse pattern with multiple synaptic sites. In the sympathetic system, the ganglia are generally far from the effector cells (e.g., within the sympathetic chain ganglia). Preganglionic sympathetic fibers may make contact with a large number of postganglionic fibers.

Cholinergic Receptors

Nicotinic receptors are ligand-gated ion channels whose activation causes a rapid (millisecond) increase in cellular permeability to Na+ and Ca2+, depolarization, and excitation.

Muscarinic receptors are GPCRs. They may be either excitatory or inhibitory, and not linked to changes in ion permeability.

Subtypes of nAChRs.

The nAChRs exsist at the skeletal NMJ, autonomic ganglia, adrenal medulla, CNS and in nonneuronal tissues

- Muscle type (Nm), found in the vertebrate skeletal muscle, where they mediate transmission at the NMJ
- Neuronal type (Nn), found mainly throughout the peripheral nervous system, CNS, and nonneuronal tissues

Pharmacological Considerations

Each step involved in neurotransmission is a potential point of pharmacological intervention. The diagram of cholinergic terminals and their postjuctional site (Figure 8-6 and 11-4) show its point of intervention. Drugs that affect processes involved in the steps of transmission at cholinergic junction is summarized in Table 8-2 and 8-7.

TABLE 8–2 ■ CHARACTERISTICS OF SUBTYPES OF NICOTINIC ACETYLCHOLINE RECEPTORS (NACHRS)

RECEPTOR (Primary Receptor Subtype)[a]	MAIN SYNAPTIC LOCATION	MEMBRANE RESPONSE	MOLECULAR MECHANISM	AGONISTS	ANTAGONISTS
Skeletal Muscle (N_m) $(\alpha 1)_2\beta 1\varepsilon\delta$ adult $(\alpha 1)_2\beta 1\gamma\delta$ fetal	Skeletal neuromuscular junction (postjunctional)	Excitatory; end-plate depolarization; skeletal muscle contraction	Increased cation permeability (Na^+; K^+)	ACh Nicotine Succinylcholine	Atracurium Vecuronium *d*-Tubocurarine Pancuronium α-Conotoxin α-Bungarotoxin

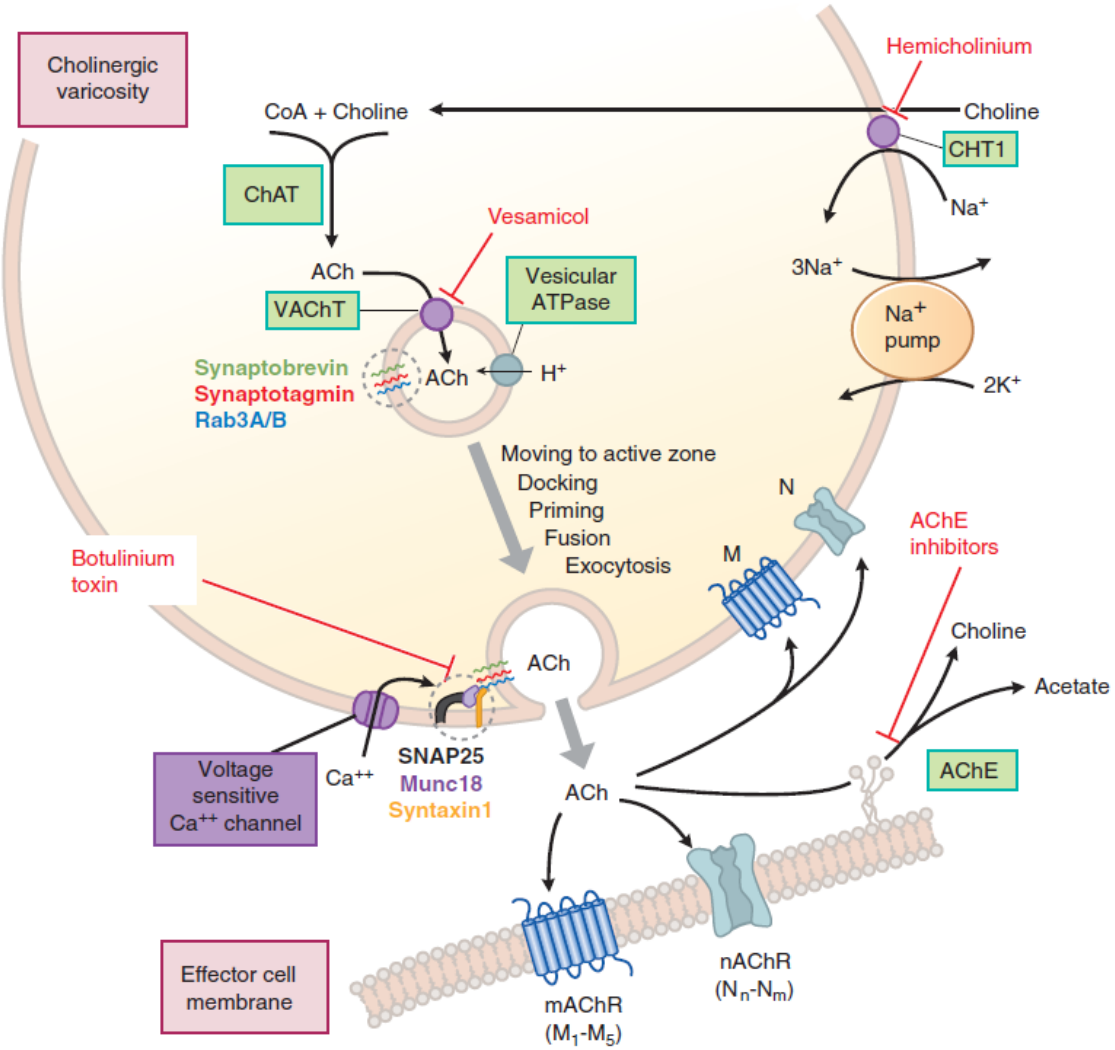

Figure 8–6 *A typical cholinergic neuroeffector junction.* The synthesis of ACh in the varicosity depends on the uptake of choline via a Na^+-dependent carrier, CHT1, that hemicholinium can block. The enzyme ChAT catalyzes the synthesis of ACh from choline and the acetyl moiety of acetyl CoA. ACh is transported into the storage vesicle by VAChT, which can be inhibited by vesamicol. ACh is stored in vesicles (along with other potential cotransmitters, such as ATP and VIP, at certain neuroeffector junctions). Release of ACh and any cotransmitters occurs via exocytosis (the stages are itemized along the gray arrow), triggered by Ca^{2+} entry via a voltage-sensitive Ca^{2+} channel in response to membrane depolarization, as described in Figures 8–3, 8–4, and 8–5. Exocytotic release of ACh at the NMJ can be blocked by botulinum toxins, the active fragments of which are endopeptidases that cleave synaptobrevin, an essential member of the SNARE proteins that mediate docking/priming/exocytosis. Once released, ACh can interact with the muscarinic receptors (M), which are GPCRs, or nicotinic receptors (N), which are ligand-gated ion channels, to produce the characteristic response of the postsynaptic cell. ACh also can act on presynaptic mAChRs or nAChRs to modify its own release. The action of ACh is terminated by extracellular metabolism to choline and acetate by AChE, which is associated with synaptic membranes.

TABLE 8–7 ■ REPRESENTATIVE AGENTS ACTING AT PERIPHERAL CHOLINERGIC AND ADRENERGIC NEUROEFFECTOR JUNCTIONS

MECHANISM OF ACTION	SYSTEM	AGENTS	EFFECT
1. Interference with synthesis of transmitter	Cholinergic	Choline acetyl transferase inhibitors	Minimal depletion of ACh
	Adrenergic	α-Methyltyrosine (inhibition of tyrosine hydroxylase)	Depletion of NE
2. Metabolic transformation by same pathway as precursor of transmitter	Adrenergic	Methyldopa	Displacement of NE by α-methyl-NE, which is an α_2 agonist, similar to clonidine, that reduces sympathetic outflow from CNS
3. Blockade of transport system at nerve terminal membrane	Cholinergic	Hemicholinium	Block of choline uptake with consequent depletion of ACh
	Adrenergic	Cocaine, imipramine	Accumulation of NE at receptors
4. Blockade of transport system of storage vesicle	Cholinergic	Vesamicol	Block of ACh storage
	Adrenergic	Reserpine	Destruction of NE by mitochondrial MAO and depletion from adrenergic terminals
5. Promotion of exocytosis or displacement of transmitter from storage sites	Cholinergic	Latrotoxins	Cholinomimetic followed by anticholinergic
	Adrenergic	Amphetamine, tyramine	Sympathomimetic
6. Prevention of release of transmitter	Cholinergic	Botulinum toxin (BTX, endopeptidase, acts on synaptobrevin)	Anticholinergic (prevents skeletal muscle contraction)
	Adrenergic	Bretylium, guanadrel	Antiadrenergic
7. Mimicry of transmitter at postjunctional sites	Cholinergic		
	Muscarinic[a]	Methacholine, bethanachol	Cholinomimetic
	Nicotinic[b]	Nicotine, epibatidine, cytisine	Cholinomimetic
	Adrenergic		
	α_1	Phenylephrine	Selective α_1 agonist
	α_2	Clonidine	Sympathomimetic (periphery); reduced sympathetic outflow (CNS)
	α_1, α_2	Oxymetazoline	Nonselective α adrenomimetic
	β_1	Dobutamine	Selective cardiac stimulation (also activates α_1 receptors)
	β_2	Terbutaline, albuterol metaproterenol	Selective β_2 receptor agonist (selective inhibition of smooth muscle contraction)
	β_1, β_2	Isoproterenol	Nonselective β agonist
8. Blockade of postsynaptic receptor	Cholinergic		
	Muscarinic[a]	Atropine	Muscarinic blockade
	Nicotinic (N_m)[b]	*d*-Tubucurarine, atracurium	Neuromuscular blockade
	Nicotinic (N_n)[b]	Trimethaphan	Ganglionic blockade

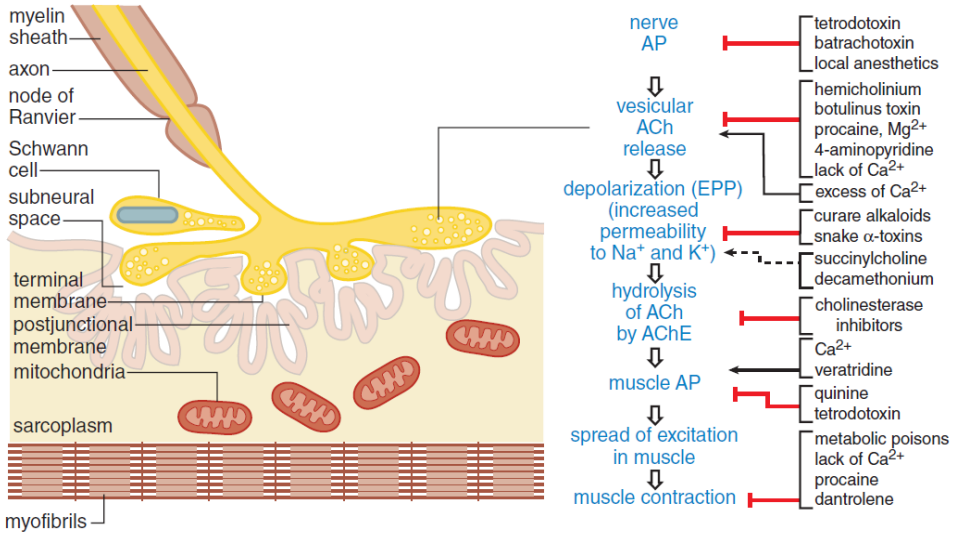

Figure 11–4 *A pharmacologist's view of the motor end plate.* The structures of the motor end plate (left side of figure) facilitate the series of physiological events leading from nerve action potential (AP) to skeletal muscle contraction (center column). Pharmacological agents can modify neurotransmission and excitation-contraction coupling at myriad sites (righthand column). ⟵, enhancement; ⊣, blockade; ⟵ - - -, depolarization and phase II block.