The Somatic and Autonomic Nervous Systems[1]

I. Somatic Nervous System:

A. General Characteristics:

1. Target and action: control of "**voluntary" movement** -- controls **skeletal muscles**.

2. Somatic nerves contain **both sensory (afferent) and control (efferent) neurons.**

! Note that a **nerve is a collection of axons**. It does not usually contain soma or dendrites and most nerves are made of large numbers of axons that pass information in both the afferent and efferent direction. **Be certain you know the difference between a neuron and a nerve.**

3. **Control centers are entirely within the CNS** -- both in the brain and spine. These centers are called **nuclei** and are sites of information processing, relay and decision-making.

B. Efferent Synapse Chemistry: In the peripheral parts of the system, all efferent neurons:

1. Release **Acetylcholine (Ach) as a neurotransmitter (NT).**

2. The Ach binds on the target cell to a special type of Ach receptor called the **nicotinic neuromuscular receptor (NNMR** also sometimes called the neuromuscular nicotinic receptor or NMNR -- sorry but it is give both ways)

! A bit of pharmacology. The receptor is described as nicotinic because it has been found that the drug nicotine will bind to this receptor and at certain concentrations cause the receptor to undergo its normal allosteric change. A pharmacological agonist such as nicotine on the NNMR thus has an effect that is similar to the normal **agonist** Ach. Both Ach and nicotine are agonists of the nicotinic neuromuscular receptor. However, the similar effects are not experienced at identical agonist concentration levels -- far more nicotine is required to get an effect. On the other hand, another NNMR agonist, **carbachol**, see below can bind to the NNMR at concentrations similar to Ach. One final note: just because something is an agonist, it does not mean it exactly mimics the effects of the normal (produced by the body) agonist. For instance, nicotine will initially stimulate NNMRs but then inhibits them.

3. Other terms for such a system are **cholinergic** (meaning that Ach is the NT) or more specifically **nicotinic** (meaning that the receptor is the nicotinic receptor (which means that Ach is the transmitter since nicotinic receptors always are also agonized by Ach).

C. Somatic Effector Activity Levels: The **somatic effectors (skeletal muscles) show no activity on their own**.

1. Thus, **if** there is no input from the somatic system (or some pharmacological agent that acts on the system's receptors), the effectors are totally inactive. Activity level itself can be adjusted only by adjusting the rate of somatic ns input to the muscles at the neuromuscular junction (with a few other ways to slightly affect performance).

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We will discuss this in detail when we consider the operation of skeletal muscles in the next unit of study.

2. Put another way, there is **no tonic level of activity** of the effectors nor is there any such level of activity in the terminal neurons of the system. **Tonus** refers to a **non-zero baseline level of activity whether a neural firing rate, contractile force or secretory rate**.

3. Thus, injuries to somatic nerves may result in total loss of function in the affected muscle.

3. One final comment -- as we will see, effector activation in the somatic ns is graded by a number of different methods, but each individual effector (muscle fiber) acts in an all-or-none fashion. This also is rather different than what happens in the autonomic ns.

II. Autonomic Nervous System (ANS): The ANS controls a wide range of **"involuntary" responses**, associated with the general physiological status of the individual.

A. General:

1. The ANS is more diffuse than the somatic -- in the somatic there are no centers of neural integration outside of the central nervous system (CNS). By contrast, in the autonomic there are a series of **ganglia** that are located outside of the CNS. A ganglion is a collection of neural cell bodies and to some degree it usually represents a processing location. Anatomically the ANS will always have a **preganglionic neuron** that links to the ganglion and then a **postganglionic neuron** that links to the effector. This is sometimes referred to as a **neural chain** (since there are two links in it between the CNS and effector).

2. The **effectors are either:**

(a) smooth muscles, for instance those lining the vascular system or the GI tract.

(b) cardiac muscle and cardiac tissue derived from muscle (the heart's conduction system),

(c) certain types of endocrine tissues (mainly the **adrenal medulla**).

B. There are **two branches of the autonomic ns**:

1. **Sympathetic** -- the neurons for this system emerge segmentally from the central portion of the spine (i.e., the non-cranial and non-caudal regions). Generally, increased activity of the sympathetic ns readies an organism for increased physical activity ("fight or flight"). Examples include

a. increasing the perfusion of skeletal muscles at the expense of blood flow to the viscera. The way this happens is rather complex and we will consider it more detail when we get to the circulation.

b. increased surface vasodilatation (*i.e.*, blood vessels near the surface open wider)

c. increased cardiac output by increasing both the heart rate and force of contraction (which will generally result in an increase in blood pumped per beat)

2. **Parasympathetic** -- the nerves for this system emerge at either end of the spine -- e.g., some of them are termed **cranial nerves** (for instance the **X^{th}Cranial** Nerve, a.k.a. the **Vagus Nerve**) **or they are among the first and last spinal nerves**. Therefore, they surround the sympathetic nerves of the autonomic nervous system.

(a) It is generally (but not always) true that the physiological effect of increased parasympathetic activity is related to putting the organism in a more relaxed or **energy conservative state** -- for example,

(b) Thus parasympathetic activity tends to increase processes associated with digestion, decrease the heart rate and cardiac output, and to decrease peripheral vasodilatation.

C. **Pharmacology of the Autonomic NS:**

1. In both systems, most of the **preganglionic neurons are cholinergic** and the **pre-ganglionic receptors are nicotinic**. (Note "pre-ganglionic means peripheral neurons that are synapsing with neural somas located within the ganglia mentioned above). This means that efferent neurons from the spinal cord (preganglionic neurons) release Ach onto cells in the ganglion that have nicotinic type receptors.

2. The **postganglionic neurons** (i.e., the neurons that project from the ganglia and synapse with the effector cells) **differ according to the branch of autonomic ns**.

1. **In the Parasympathetic:**

(a) neurotransmitter -- acetylcholine

(b) receptor on the effector -- muscarinic.

! Note that this receptor is not significantly affected by nicotine but instead binds a very different substance, the **mushroom toxin muscarine** (a favorite of Lucretia Borgia of Renaissance Italy fame). Muscarine is also an agonist and can have very pronounced physiological affects -- it kills!

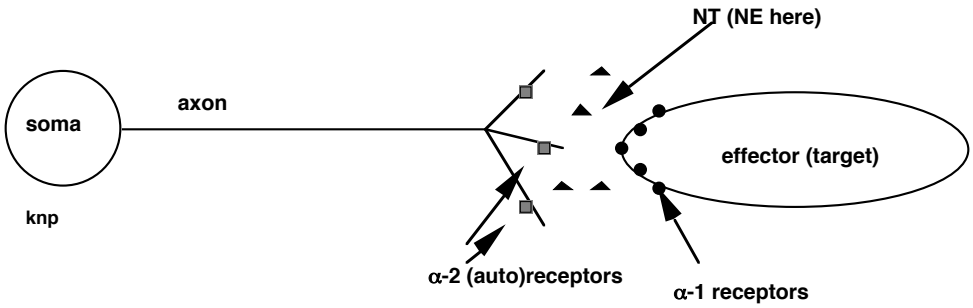
2. **Sympathetic:**

(a) **transmitter: norepinephrine (NE), a.k.a. noradrenalin**

(b) **receptors: alpha 1 or 2 (α_1, α_2),** and to a lesser degree the beta 1 or 2 (β_1, β_2)

(i) **α-1** receptors are the more common type of alpha receptor and are found at all autonomic ns neuromuscular junctions (ex: in the vasculature, on the SA and AV nodes of the heart, in the GI tract).

(ii) **α-2** receptors are termed **autoreceptors** and are examples of an important phenomenon -- they are receptors found on the axonal terminals of the cells releasing the NT (here NE) -- they monitor the levels of NT in the synapse and can be used as part of an important feedback mechanism to decrease the amount of NT released when the levels get too high.



! You are slowly being lead into the main difficulty in understanding the autonomic ns. There are a large number of different receptor types each found in different tissues and which different properties. Further as we will see below, the effects can vary with dose and the transmitters to

some degree cross react -- they act both on their own receptors and on the ones that more commonly use other substances as agonists (if their concentrations get high enough).

(b) **Hormone:** in addition, the **Adrenal Medulla** is a portion of the sympathetic ns. This portion of the "adrenal gland" (it is really two very distinct tissues like the hypophysis -- the other part is called the adrenal cortex) secretes the hormone **epinephrine** (**E**) AKA adrenaline. The **adrenal medulla is nothing more than a sympathetic ganglion** where the cells are specialized for producing and releasing (when ordered) the hormone epinephrine, which chemically is extremely close to the neurotransmitter NE

1. Receptors for epinephrine and some of the places where they are found
   - (a) β_1: heart, adipose tissue
   - (b) β_2: vascular muscles, bronchioles, smooth muscle of GI tract, uterus, etc.
2. Collectively **NE and E along with dopamine** (a CNS neurotransmitter) and several other related compounds are **referred to as catecholamines**. They are all straightforward derivatives of the amino acid tyrosine.
3. The general activity of the sympathetic ns is refereed to as **adrenergic**[2]. However, the exact effects differ according to the location and receptor/NT types.

(c) On the last page there is a listing of the effects of autonomic activity on certain tissues and organs

(d) **Pharmacological agents and their targets** -- the table below lists a number of pharmacological agents that can act relatively specifically on certain receptors by either activating or blocking them. The list is by no means complete and there are additional ways besides agonism or blockage to affect autonomic function.

Note: Please do not feel that you must memorize this table. We will go over many of these effects as we consider various organ systems and you can learn the effects then. For the moment, read it over and ponder it and then use it as a reference.

Type / Agent	Compound and Receptor
1. Sympathetic	epinephrine (natural) **β** norepinephrine **α** **β agonist** isoproterenol (isupryl), ephedrine **β antagonist** propranolol (inderal) **α agonist** phenylepherine **α antagonist** phenoxybenzamine
2. Parasympathetic	acetylcholine **muscarinic**

[2] the term "adrenergic" refers to adrenal and means that the receptor/transmitter pair uses norepinephrine or epinephrine or both with alpha- or beta-receptors.

muscarinic agonist
muscarine,
carbachol
muscarinic antagonist
atropine

Some effects of the autonomic ns:

Effector	Adrenergic Receptors	Adrenergic Response	Cholinergic Response
Heart			
Pacemaker	β_1	increase rate	decrease rate
Myocardium (I'm making lots of generalizations -- the myocardium is not the same everywhere in the heart)	β_1	increase contractility, increase automaticity (pacemaker activity), increase conduction velocity	slight decrease in contractility
Arterioles	α_1, β_2	usually vasoconstriction	vasodilatation
Systemic Veins	α_1, β_2	α_1 -- constriction; β_2 -- dilatation	none
Lungs -- bronchiole smooth muscles	β_2	relaxation	constriction
GI (very general)	α_2, β_2	decrease in motility, contraction of sphincters, decrease in secretions.	opposite of sympathetic effects
uterus motility and tone	α_1, β_2	if pregnant -- contraction; if not pregnant -- relaxation	variable -- depends on stage of cycle and amount of estrogen and progesterone in circulation
male sex organs	α	ejaculation	erection
skin pilomotor skin sweat glands	α	contraction localized secretion	none generalized secretion.
adipose cells	α_1, β_1	lipolysis	none