UAB MEDICINE

April 2021 Education Newsletter

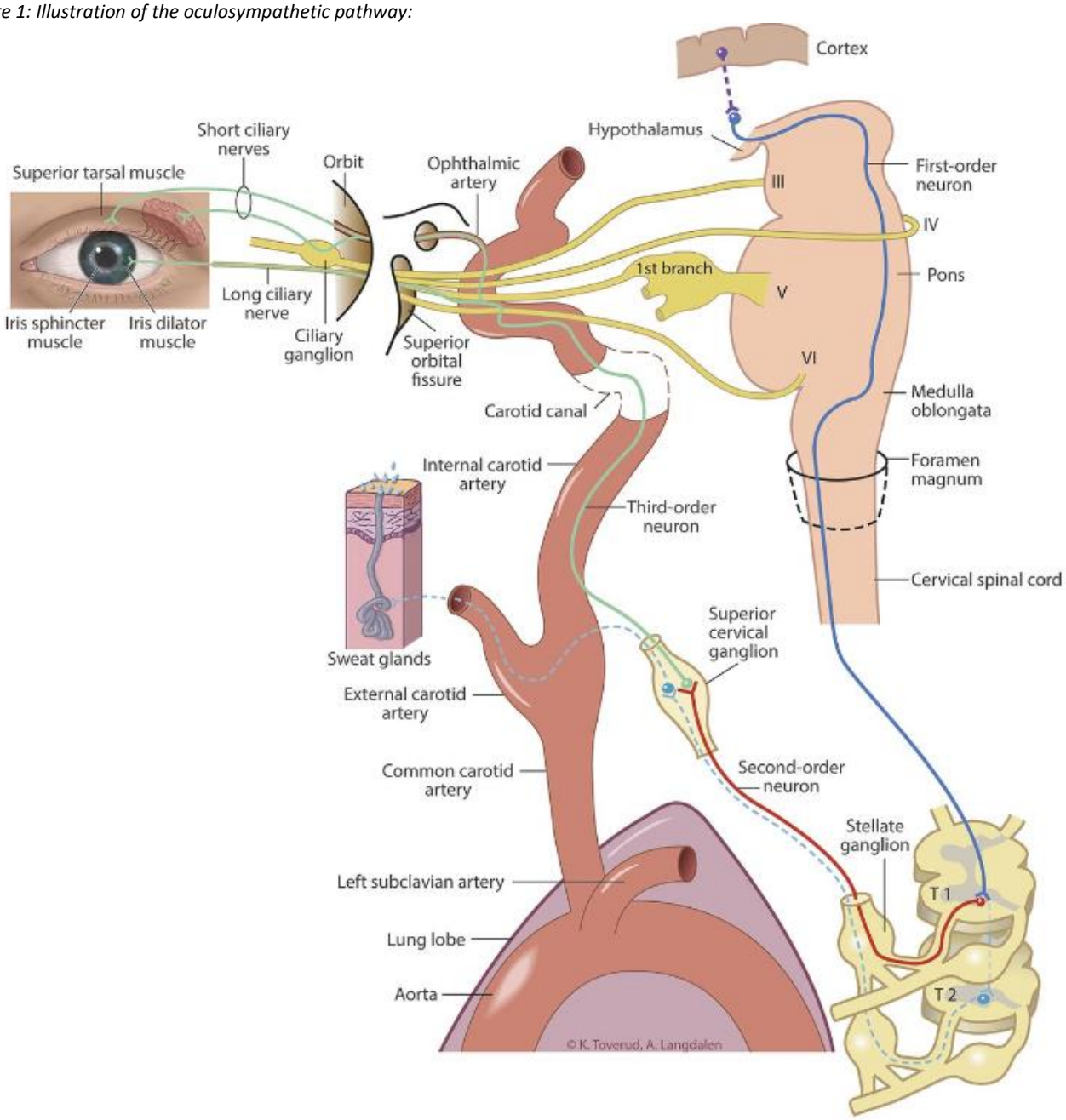
# The Neuroanatomy of Horner Syndrome

**Overview:**
Horner syndrome is a classic neurologic syndrome more technically known as *oculosympathetic paresis*. It occurs following damage to the sympathetic autonomic nervous pathway in the head, resulting in miosis (excessive constriction of the pupil), ptosis (drooping of the upper eyelid), and anhidrosis (inability to sweat). The differential for Horner is broad and includes stroke, tumors (that can occur in a variety of locations), demyelinating diseases, trauma, aneurysms, dissections, inflammation, thromboses, cluster headache, and more. The best approach to developing an accurate differential and, thus, implementing the appropriate work up, is to understand the neuroanatomy responsible.

**Classification and Neuroanatomy**:
*Refer to the figure below*. The sympathetic innervation of the eye is described as a 3-neuron-pathway, and, as such, Horner's is usually classified by which one of the three nerves are affected:

1) The first order neuron (***blue*** in the illustration below): this centrally-located sympathetic nerve runs from the hypothalamus through the brainstem, down into the cervicothoracic spinal cord where it then meets the second order neuron.
    a. The most common etiology for Horner syndrome involving the **first order neuron** is infarction (specifically a lateral medullary infarction), but any stroke, tumor, or demyelinating lesion along the sympathetic tracts within the brainstem or cervicothoracic spinal cord can also result in first order Horner syndrome.
    b. Since these are central nervous system lesions, most etiologies of Horner syndrome involving the first order neuron are associated with other symptoms which may overshadow the miosis, ptosis, and anhidrosis (such as severe vertigo, ataxia, sensory loss, or diplopia).
2) The second order neuron (***red*** in the illustration below): these sympathetic nerves run from the sympathetic trunk within the spinal cord, through the brachial plexus, over the lung apex, and finally to the superior cervical ganglion (located near the bifurcation of the carotid) where they then synapse with the third order neuron.
    a. The most common etiologies for Horner syndrome that involve the **second order neuron** (also called the pre-ganglionic neuron) include trauma, surgery, or malignancy within the spinal cord, thoracic outlet, or lung apex.
    b. When due to malignancy (such as a Pancoast tumor), second-order Horner's often presents occultly, though if sensory fibers are compressed within the brachial plexus, ipsilateral axillary or arm pain can occur.
3) The third order neuron (**green** in the illustration below): this nerve leaves the superior cervical ganglion and then ascends through multiple vascular structures (including the adventitia of the carotid artery as well as the cavernous sinus) and then to two cranial nerves (the sixth cranial nerve and the V1 of the fifth cranial nerve) and finally to oculosympathetic nerves in control of a variety of eye muscles (including the iris dilator muscle and muscles which are responsive for upper eye lid elevation).
    a. Common causes of Horner's that result from a **third order neuron** (or the post-ganglionic neuron) include carotid dissections, neck masses, and otitis media.
    b. Acute onset of Horner syndrome in the setting of neck or facial pain is the result of a carotid dissection until proven otherwise!

*Figure 1: Illustration of the oculosympathetic pathway:*




**Tim's Take Home Pearls:**

***Starting at the hypothalamus and ending at the pupil, there are three different nerves involved in the oculosympathetic pathway.**

***Disruption along this pathway, at any point, can result in Horner syndrome – which includes a triad of miosis, ptosis, and anhidrosis.**

***Horner's is traditionally categorized by which of the three nerves are affected along the pathway.**

***Acute Horner syndrome in the setting of neck or facial pain is the result of a carotid dissection until proven otherwise!**