Horner Syndrome

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Johann Freidrich Horner (Fig. 1), a student of Carl Ludwig and Albrecht von Graefe, wrote his most notable contribution to medicine entitled, “Ueber eine Form von Ptosis” (On a form of Ptosis) in 1869 (1). In this article, he described the case of a 40-year-old woman with ptosis, miosis, and enophthalmos. He also noted a distinct flushing and warmth of the ipsilateral hemiface and recorded the patient’s observation that she was not sweating on that side of her face. Horner documented temperature readings of each hemiface indicating a substantial discrepancy with higher temperatures on the involved side. He also reported the use of atropine and calabar (an extract containing physostigmine), showing a blunted effect of atropine and hypersensitivity to calabar on the involved pupil. Horner (1) comments, albeit briefly, on the potential mechanism:

“The vasomotor disturbance involves not only the trigeminal area, but also the fibres of the cervical sympathetic; it is not too much to assert that this experiment with belladonna and calabar speaks for the
dual control of the movements of the iris in man … we are dealing with a right dilator paralysis … Ptosis … remains gradual and incomplete … a paralysis of the musculus palpebrae superioris supplied by the sympathetic nerve.”

Despite the syndrome bearing his name today, Horner was not the first to describe the effects of sympathetic denervation to the eye and face. The first recorded observation in 1727 was by Pourfour du Petit (2). He severed the vago-sympathetic nerve trunk of dogs and recorded his observations, including a sunken globe, narrowing of the palpebral fissure, injection of the conjunctiva, relaxation of the nictitating membrane, and a decrease in the size of the pupil. The first report of a human case came from Edward Selleck Hare when Horner was only 7 years-of-age. Hare (3) described a patient with a tumor in the left side of the neck, noting ipsilateral miosis and ptosis. Hare was uncertain of the mechanism and wrote that these findings “must be regarded as an instance of that remote sympathy which is found to exist between distant parts of the same individual and is most frequently displayed in a person of nervous temperament.” Unfortunately, Hare died of typhoid fever in the same year of this publication. John Reid, in 1839, after seeing Hare’s case, commented that injuries of the cervical sympathetic in man could produce contraction of the iris and conjunctival inflammation (4).

Experimental studies of the sympathetic innervation of the eye, of which Horner was well aware, had been in progress in the years before Horner’s publication. In 1847, Ruete (1) proposed sympathetically innervated pupillary radial fibers and circular fibers being supplied by the third nerve. In 1851, Julius Ludwig Budge (1) experimented with sectioning sympathetic fibers, discovering that these fibers originated in the spinal cord. Both Ruete and Budge explained, in detail, the findings of pupillary alterations in their experiments.

Perhaps, the most notable experimental work in sympathetic innervation was done by Bernard (5). In 1852, he fully described the effects of sectioning the cervical sympathetic fibers in rabbits. He noted constriction of the pupil and injection of the conjunctiva, retraction of the globe and relaxation of the nictitating membrane, narrowing of the palpebral fissure, reduction in intraocular pressure and reduction in the size of the globe, reduction in the size of the nares, and increasing temperature over the involved hemiface with flushing. He also showed that with stimulation of the cut-end of the fibers, all these findings would completely reverse. In 1853, Budge (1) repeated Bernard’s experiment with the addition of severing the fibers at the spinal cord, again indicating that the sympathetic fibers arise from the spinal cord.

In 1861, 8 years before the Horner’s publication, Mitchell et al (6) described a 24-year-old soldier with a gunshot wound to the neck with ptosis, miosis, enophthalmos, and hemifacial flushing. Mitchell, being aware of the previous work by Budge and Bernard, called this patient “a case of injury of the sympathetic nerve.”

In the United States, the syndrome is referred to as Horner syndrome, whereas in the United Kingdom it is Bernard-Horner syndrome, and in France it is known as Claude Bernard syndrome. Although these eponyms are likely to continue, they do not give credit to the others who contributed to this discovery, including Mitchell, Reid, Hare and Budge.

REFERENCES