

Disorders of the
Autonomic Nervous System



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Introduction and Acknowledgements

All human actions, voluntary and involuntary, are accompanied by activity of the autonomic nervous system. Nevertheless, clinical textbooks give little space to its dysfunction, and its disorders are often reported as medical curiosities. In recent years improvements in techniques have, however, made it possible to examine many aspects of autonomic function in man. It has become apparent that defects in autonomic function occur commonly in many diseases and are more dangerous and disabling than was appreciated. This book aspires to describe those aspects of the physiology of the autonomic nervous system which are important for clinical purposes, the tests which can usefully be done to demonstrate the nature of the defect, the diseases in which the autonomic nervous system is known to be involved, and their management.

William Hazlitt (1778-1830), the English essayist, wrote of a newspaper 'It is elaborate, but heavy; full, but not readable . . . and might be imagined to be composed as well as printed with a steam engine'. Writers of textbooks also risk this charge, but if the art of pleasing is to be pleased then we are hopeful: we have enjoyed writing this book.

This book has been made possible by the encouragement of many, both in its production and in the investigations from which it arose. We wish to thank all those who have helped in its preparation, and in particular Mrs Myra Cottingham. Some of the work described in this book has been carried out by ourselves, and we are grateful to all those who made this possible, particularly the patients, and also Professors A. Crampton Smith and K.E. Cooper who have collaborated in some of it, and Professors W. Ritchie Russell, Sir George Pickering and J.A. Simpson who have given encouragement.

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et al, *J. Appl. Physiol.* **23**, 347–352, fig. 1, c; Fig. 9.8 Bryce-Smith *et al*, *J. Physiol.* **145**, 77–84, fig. 6; Fig. 9.9 Johnson, *Ann. Roy. Coll. Surg. Eng.* **36**, 339–352, fig. 11; Fig. 10.1 Crockford *et al*, *J. Physiol.* **207**, 26–27P, fig. 1; Fig. 10.2 adapted from Murray & Thompson, *Brit. Med. Bull.* **13**, 213–219, fig. 6; Fig. 10.5 adapted from Boyd, *Brit. Med. Bull.* **13**, 207–212, fig. 1; Fig. 12.2 adapted from Nathan & Smith, *J. Neurol. Neurosurg. Psychiat.* **21**, 177–189, fig. 6; Fig. 12.5 adapted from Nathan & Smith, *J. Neurol. Neurosurg. Psychiat.* **14**, 262–280, fig. 15; Fig. 13.1 (adapted) & 13.2 Andrew & Nathan, *Brain*, **87**, 233–262, figs. 10B & C, & 10A; Fig. 13.3 Andrew & Nathan, *Proc. Roy. Soc. Med.* **58**, 553–555, fig. 2; Table 13.2 Tarabuley, *Paraplegia* **10**, 201–208, derived from data in the text; Fig. 14.1 Glaister *et al*, *Brit. Med. J.* **2**, 942–946, fig. 5; Fig. 14.2 adapted from Majid *et al*, *Brit. Med. J.* **4**, 328–334, fig. 1; Fig. 15.1 Wallace & Benson, *Scientific American* **226** No. 2, 85–90, lower fig. p. 87; Fig. 15.2 Blair *et al*, *J. Physiol.* **148**, 633–647, fig. 1; Fig. 15.3 Bevan *et al*, *Clin. Sci.* **36**, 329–344, fig. 4.

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CHAPTER 1

Anatomy of the Autonomic Nervous System

Sympathetic nervous system

Central
Peripheral

Parasympathetic nervous system

Central
Third cranial nerve
Seventh, ninth and tenth cranial nerves
Central pathway to sacral division
Peripheral
Third cranial nerve
Seventh cranial nerve
Ninth cranial nerve
Tenth cranial nerve
Sacral parasympathetic nerves

I propose the term *autonomic nervous system* for the sympathetic system and the allied nervous system of the cranial and sacral nerves and for the local nervous system of the gut.

Langley* (1898)

The autonomic nervous system consists of two groups of pathways, the sympathetic or thoracolumbar group and the parasympathetic or craniosacral group. Both are efferent (effector) systems but the structures they innervate may also have afferent innervation. The afferent nerves then accompany the autonomic (efferent) nerves, and they are sometimes referred to as 'autonomic afferents', a phrase which has aroused much emotion. The activity of the autonomic nervous system is influenced not only by these nerves, but also by receptors within the brain and peripheral receptors which stimulate afferents in somatic nerves. In this book the autonomic nervous system is regarded as efferent. Visceral efferent pathways are given for convenience in Tables 1.1 and 1.2, and are also referred to in the text where it is necessary to do so for the understanding of the clinical physiology. The anatomical details in this chapter are those needed for the purposes of this book, and further details can be obtained from Kuntz (1953), Mitchell (1953) and Pick (1970).

* Langley, John Newport (1852-1925). Physiologist at Cambridge University, England. He introduced the terms *pre-* and *post-ganglionic* in 1895; and in 1905 restricted the use of *sympathetic* and introduced the term *parasympathetic*.

Sympathetic nervous system

CENTRAL

The hypothalamus controls many sympathetic functions including those concerned especially with cutaneous blood flow and body temperature regulation (Cooper 1966) (Chapter 8). The descending pathways are believed to pass close to and anterolateral to the aqueduct of Sylvius,* close to the red nucleus (Carmel 1968) and then more laterally in the medulla to synapse in the intermediolateral cell columns of the spinal cord between about T1 and L2 segments (Fig. 1.1).

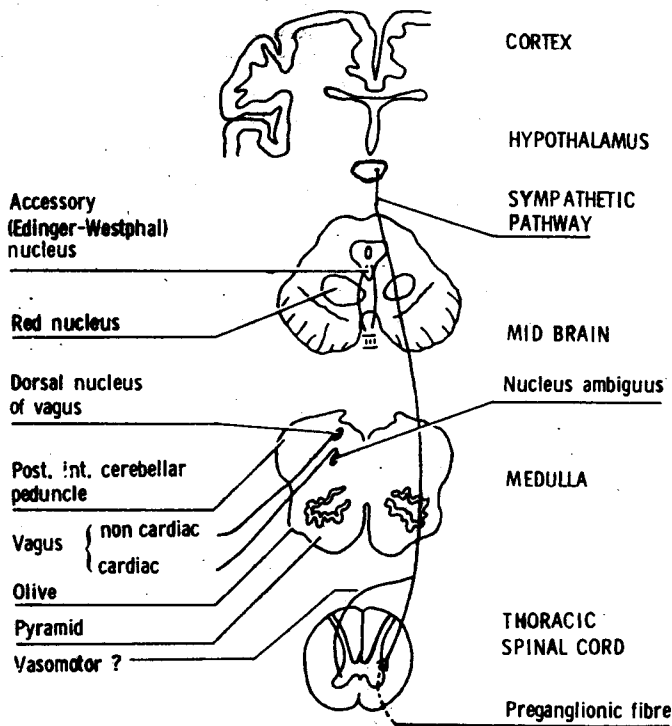


Fig. 1.1. On the right the course of sympathetic pathways from the hypothalamus to the cells in the intermediolateral columns of the thoracic spinal cord, from which arise the preganglionic sympathetic fibers. On the left the origin of parasympathetic fibers in the third and tenth cranial nerves.

* Sylvius. There is confusion between two Sylviuses, Jacobus (Jacques Dubois 1478–1555) and Franciscus (Francois de le Boë 1614–72). Jacobus is often credited with the description of the aqueduct of Sylvius, 'aqueductus cerebri', but recent investigations attribute it to Franciscus. Born of a French family in Hanau, Germany, Franciscus Sylvius received the doctor's degree from Basle, Switzerland, and became Professor of Practical Medicine at the University of Leyden, Holland.

PERIPHERAL

Cell bodies in the intermediolateral cell column have myelinated (preganglionic) axons which pass out in the anterior roots (Fig. 1.2) to reach the sympathetic

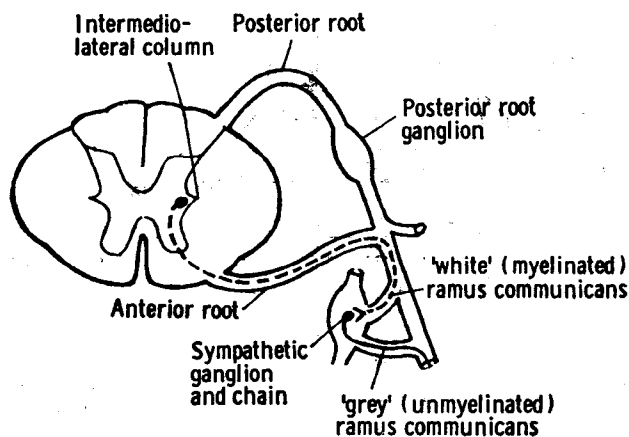


Fig. 1.2. Relationship of preganglionic (-----) and postganglionic (——) sympathetic fibers with nerve roots and sympathetic chain.

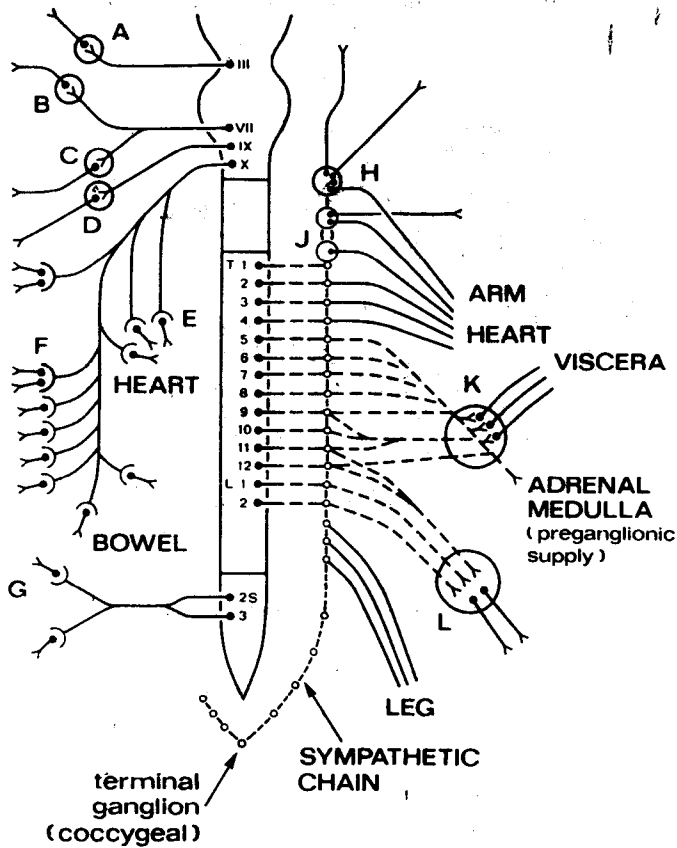
chain. The sympathetic chain consists of a series of ganglia and intervening nerves. It extends on each side from the base of the skull to the coccyx (Fig. 1.3). In the neck it lies behind the carotid sheath and in front of the transverse processes of the cervical vertebrae. In the thorax it lies on the heads of the ribs; in the abdomen on the anterolateral aspect of the bodies of the lumbar vertebrae; and in the pelvis on the front of the sacrum, medial to the anterior sacral foramina. In front of the coccyx the two chains meet in a terminal ganglion (coccygeal ganglion, ganglion impar).

In the neck there are three ganglia: the upper, which is about the level of the bifurcation of the carotid artery; the middle; and the lower or stellate ganglion, which is usually fused with the T₁ ganglion. In the thorax there are ten to twelve ganglia, usually four in the lumbar region and four or five in the sacral region. Often the ganglia are partly fused.

Some preganglionic fibers synapse in the nearest ganglion, some pass up or down the sympathetic chain to synapse in other ganglia of the chain and others pass on to synapse in more peripheral ganglia, notably those in the splanchnic area (Fig. 1.4). The ganglia contain cell bodies whose unmyelinated (postganglionic) fibers innervate the effector organ. Sympathetic ganglia, unlike the parasympathetic, are not usually immediately adjacent to their effector organ. In the limbs sympathetic fibers normally accompany the major peripheral nerve and then are distributed distally with the arterial supply.

PARASYMPATHETIC

SYMPATHETIC



Parasympathetic system
 from cranial nerves III, VII, IX, X
 and from sacral nerves 2 and 3

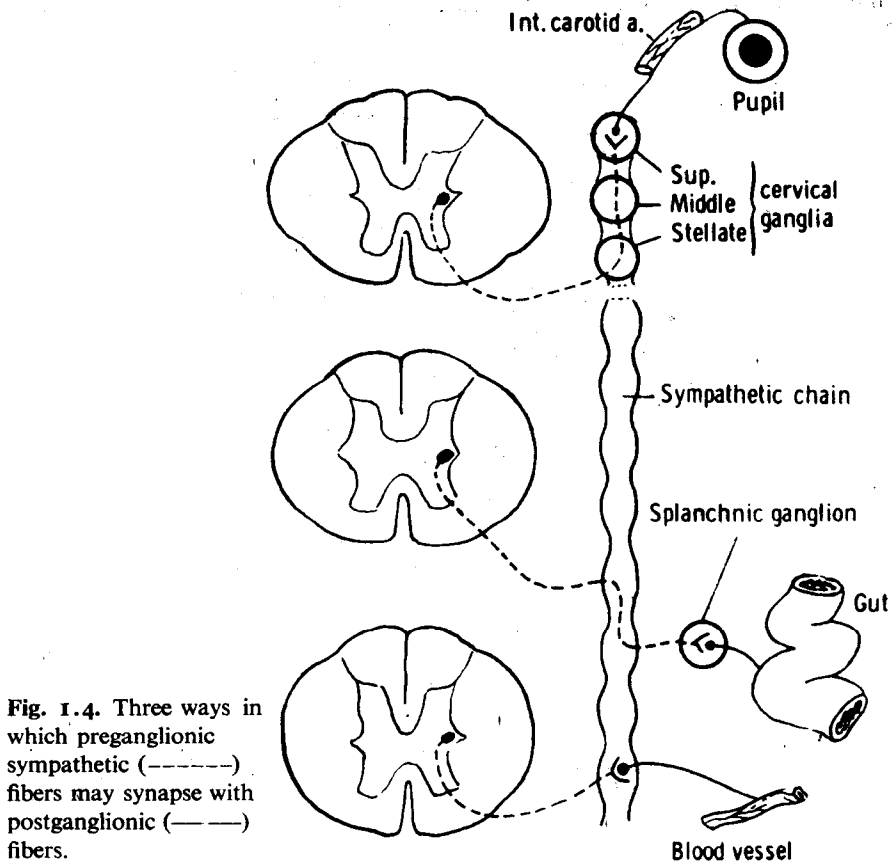
Sympathetic system
 from T1 to L2
 preganglionic fibers -----
 postganglionic fibers ———

- A ciliary ganglion
- B sphenopalatine (pterygopalatine) ganglion
- C submandibular ganglion
- D otic ganglion
- E vagal ganglion cells in heart wall
- F vagal ganglion cells in bowel wall
- G pelvic ganglia

- H superior cervical ganglion
- J middle cervical ganglion and inferior cervical (stellate) ganglion including T1 ganglion
- K coeliac and other abdominal ganglia
- L lower abdominal sympathetic ganglia

Fig. 1.3. The peripheral autonomic nervous system.

Although the sympathetic outflow of the spinal cord is usually T1 to L2, it is variable. If the brachial plexus moves up a segment in origin (i.e. is 'prefixed'), then the preganglionic outflow moves up one segment to C8. When the brachial plexus is 'postfixed', then the second thoracic ganglion may be the highest level of the outflow. Similarly the lumbar outflow may extend down to L3. There is also variation in the levels supplying the various structures of the body. Thus the origin of the sympathetic supply to the skin of the head and neck is usually T1 to T2, but may become C8 or T3 and T4. The oculopupillary fibers appear to be relatively constant from T1, and sudomotor fibers from below this level (Goetz 1948). The supply to the skin of the upper limb is commonly from T2 to T8 or T9, and the fibers from the lower segments run up the sympathetic chain. Therefore, in treating hyperhidrosis of the upper limb or face, removal of the T2 to T3 ganglia will usually effect a cure without producing ptosis and a small pupil. The fibers concerned with the pupil and upper lid and with sweating throughout the body do not cross (Carmel 1968), but it is possible that in man vasomotor



fibers cross in the upper cervical cord. The lower extremities depend on levels from T10 to L2 or L3 (Goetz 1948). The central area of the face may have accessory sudomotor innervation via the facial nerve, and the perineum via the sacral nerves (Monro 1959).

Sympathetic nervous activity is largely reflex and it is therefore requires an afferent limb to the reflex arc. The afferent limb may be almost any afferent nerve. A startling sight (second cranial nerve), a sudden noise (eighth cranial nerve), a painful or warm cutaneous stimulus (superficial nerve), a thrombosed coronary artery or a distended viscus (deep nerve) can each set up reflexes whose motor limbs are in the sympathetic nervous system. Some afferent fibers ('sympathetic afferent') pass with motor fibers of the sympathetic nervous system (Cooper & Kerslake 1955). The sympathetic reflexes, like somatic reflexes may have only a short pathway in the spinal cord of one or a few segments (as with some bladder reflexes), may be over several segments, or may involve the brain with or without conscious appreciation.

The sympathetic innervation of the body is shown in Table 1.1 (Bonica 1968) and the cutaneous distribution in Fig. 1.5. Further details of sympathetic innervation to the eye are given in Chapter 11, to the genito-urinary systems in Chapter 12 and to the alimentary tract in Chapter 14. Sweating is described in Chapter 10.

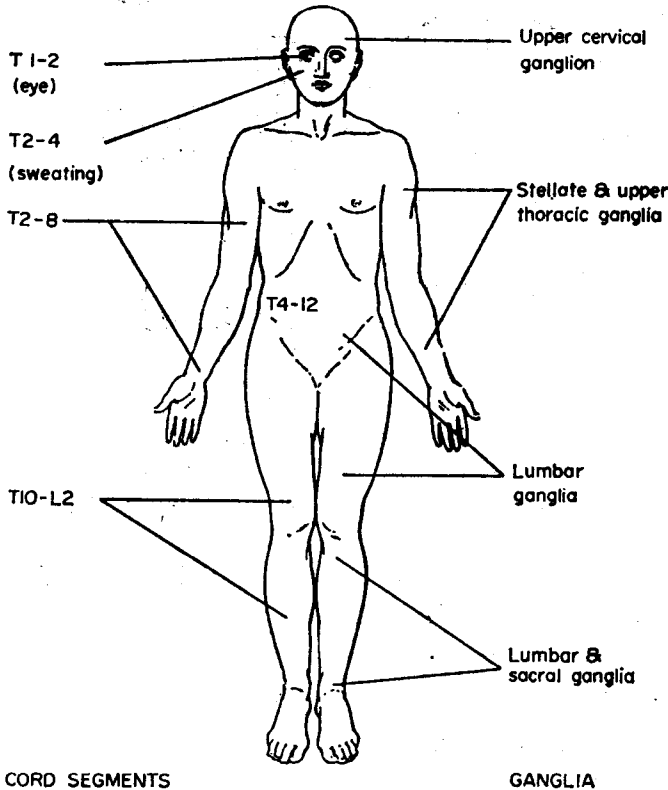


Fig. 1.5. Sympathetic innervation of the skin by cord segments (left) and ganglia (right).

Parasympathetic nervous system

CENTRAL

The hypothalamus plays a part in the control of the parasympathetic nervous system, but little is known about the central connections in man.

THIRD CRANIAL NERVE

The parasympathetic preganglionic cells of the third nerve lie in the accessory (Edinger-Westphal*) (Edinger 1885; Westphal 1885) nucleus in the postero-superior part of the oculomotor nuclear complex in the floor of the aqueduct in the mid-brain (Fig. 1.1).

SEVENTH, NINTH AND TENTH CRANIAL NERVES

The nuclei of the parasympathetic parts of these nerves are closely related in the medulla. The parasympathetic preganglionic fibers of the tenth cranial nerve which supply the heart arise in cells close to the nucleus ambiguus (Fig. 1.1). The remaining preganglionic fibers in this nerve arise in the dorsal nucleus of the vagus in the floor of the fourth ventricle (Mitchell 1953). The superior end of the dorsal nucleus forms the inferior salivary nucleus which contains cell bodies of preganglionic fibers concerned with the innervation of the parotid gland through the ninth cranial nerve (Fig. 1.6) (Chapter 14). Superior again to this is the superior salivary nucleus which supplies parasympathetic fibers to the seventh cranial nerve. These are secretomotor and vasodilator for the sub-mandibular and sublingual salivary glands. Fibers which are secretomotor for the lacrimal gland arise slightly superior, just within the pons.

CENTRAL PATHWAY TO SACRAL DIVISION

Descending pathways from the brain pass in the lateral columns of the spinal cord to reach the intermediolateral nucleus of the sacral spinal cord where the preganglionic cell bodies are situated. This nucleus is in the most lateral part of the grey matter, lateral to the central canal, but does not form an intermediolateral column as the preganglionic cells of the sympathetic system do in the thoracic spinal cord. See also Chapter 12.

PERIPHERAL

THIRD CRANIAL NERVE

The parasympathetic fibers of the third nerve, supplying the pupil and ciliary muscles, join the fibers supplying the external ocular muscles to emerge from the brain in the medial sulcus of the cerebral crus. They form part of the third

* Edinger, Ludwig (1855-1918). Anatomist and neurologist at Frankfurt-am-Main. Westphal, Karl Friedrich Otto (1833-90). Professor of Psychiatry at Berlin.

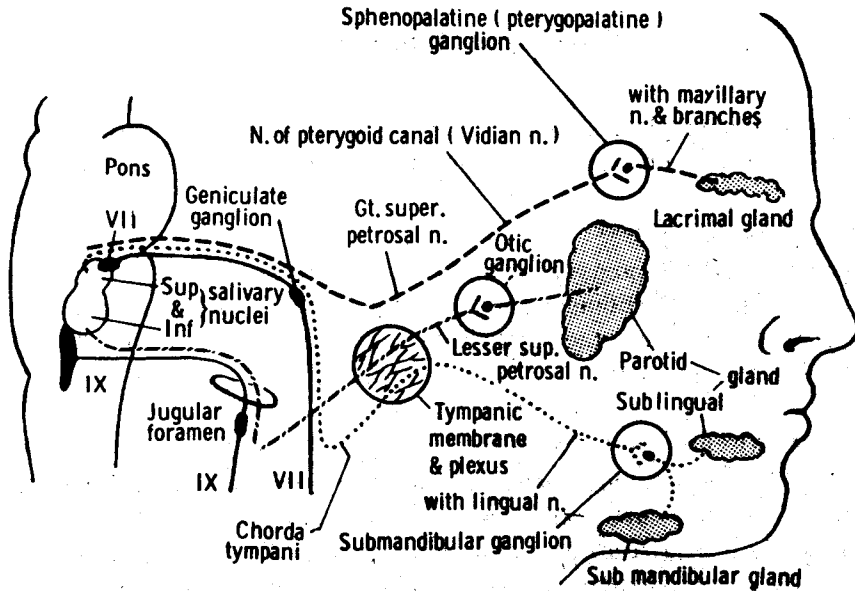


Fig. 1.6. Course of parasympathetic fibers leaving the brain in the seventh, ninth and tenth cranial nerves. ----- Parasympathetic fibers in the facial nerve supplying lacrimal gland. Parasympathetic fibers in the facial nerve supplying submandibular and sublingual glands. -.-.-.- Parasympathetic fibers in the glossopharyngeal nerve supplying the parotid gland. ——— Somatic nerve fibers.

nerve and accompany the fibers to the inferior oblique muscle, leaving it to relay in the ciliary ganglion which lies loose in the fat in the posterior part of the orbit. The postganglionic fibers reach the eye in the short ciliary nerves which tend to run in groups above and below the optic nerve. They penetrate the sclera, run forward in the perichoroidal space and innervate the sphincter pupillae and the ciliary muscle. Autonomic abnormalities from lesions of the third nerve are described in Chapter II.

SEVENTH CRANIAL NERVE

The parasympathetic fibers of the seventh cranial nerve accompany the skeletal motor nerve fibers and emerge at the lower border of the pons (Fig. 1.6). They pass with the small sensory component of the facial nerve (nervus intermedius) which lies between the seventh and eighth cranial nerves. At the geniculate ganglion (which is a sensory ganglion of the facial nerve, not a parasympathetic relay station), those parasympathetic fibers which are secretomotor for the lacrimal gland leave the main nerve in the greater superficial petrosal nerve. They continue as the nerve of the pterygoid canal (Vidian* nerve) to enter the

* Vidius, Vidus (Guido Guidi) (1500–69). Physician to Francis I of France and Professor of Medicine at the University of Pisa from 1548. His original work *De anatomic corporis humani* was published posthumously in Venice in 1611.

pterygopalatine fossa and relay in the sphenopalatine (pterygopalatine) ganglion. The postganglionic fibers then join the maxillary nerve and reach the lacrimal gland with branches of that nerve. Vasodilator and secretomotor fibers for the middle meningeal arteries and for vessels and glands in the mucosa of the nose and its related sinuses also pass in the greater superficial petrosal nerve (Mitchell 1953).

Parasympathetic fibers supplying the submandibular and sublingual salivary glands leave the facial nerve a few millimeters above its exit from the facial canal at the stylomastoid foramen, and form part of the chorda tympani of which the greater part consists of sensory fibers subserving taste. They cross the tympanum and emerge from the skull to run on the medial aspect of the lateral pterygoid muscle. With the rest of the chorda tympani they join the lingual nerve, and they relay in the submandibular ganglion. The postganglionic fibers innervate the submandibular and sublingual glands, and are secretomotor and vasodilatory. In lesions of the facial nerve autonomic disturbances are usually inconspicuous, but occasionally faulty regeneration causes gustatory sweating (Chapter 10), 'crocodile tears' (Chapter 11) or thermal salivation (Chapter 14).

NINTH CRANIAL NERVE

The parasympathetic fibers of the ninth cranial nerve, supplying the parotid salivary gland, pass laterally from the inferior salivary nucleus and emerge from the medulla with the rest of the nerve in the groove between the olive and the inferior cerebellar peduncle. The nerve leaves the skull through the jugular foramen (Fig. 1.6) and in or immediately below this foramen the fibers from the inferior salivary nucleus leave the main nerve. They ascend to the tympanic cavity through a canal in the bone separating the jugular foramen from the carotid canal. In the tympanum they take part in the tympanic plexus to which fibers are also contributed from the facial nerve and from the sympathetic plexus around the internal carotid artery. All or most of the fibers from the ninth cranial nerve, however, pass on as the lesser superficial petrosal nerve through a canal which emerges from the anterior surface of the petrous temporal bone into the middle cranial fossa. They leave the skull through the foramen ovale with the third division of the fifth cranial nerve. On leaving the skull, the preganglionic fibers relay in the otic ganglion, and the postganglionic fibers reach the parotid gland with the auriculotemporal nerve. They are secretomotor and vasodilator. As they enter the parotid gland, they pass close to the postganglionic sympathetic fibers from branches of the external carotid artery which are distributed with the auriculotemporal nerve. In lesions of this nerve, autonomic disturbances are usually slight unless there is abnormal gustatory sweating due to faulty regeneration along sympathetic pathways in the auriculotemporal nerve (Chapter 10, Fig. 10.3).

As has been mentioned, almost any afferent nerve can convey impulses which will affect an autonomic reflex. The afferent impulses from the baroreceptors

and the carotid sinus are, however, so important an afferent pathway for control of the circulation and therefore for autonomic activity, that it is justifiable to mention here that their afferent supply is in the ninth cranial nerve.

TENTH CRANIAL NERVE

The cardiac parasympathetic fibers pass from the neighborhood of the nucleus ambiguus backwards towards the floor of the fourth ventricle. They turn laterally to join the fibers from the dorsal nucleus with which they pass laterally to emerge from the medulla below the ninth cranial nerve in the groove between the olive and the posterior cerebellar peduncle (Fig. 1. 1). The nerve emerges as rootlets which unite to form a single trunk which leaves the skull through the jugular foramen with the eleventh cranial nerve, posterolateral to the ninth nerve. It then passes down within the carotid sheath to the thoracic inlet. On the right side the nerve descends through the superior mediastinum behind the right innominate vein and then behind the superior vena cava to lie on the right side of the trachea. It reaches the posterior aspect of the root of the right lung, and splits into a posterior and a smaller anterior part which together form the main components of the pulmonary plexus. At the lower margin of the lung root, the nerve largely reforms and passes down close to the lower esophagus, reaching its lower end almost on its posterior aspect. On the left side, the nerve on entering the chest descends through the superior mediastinum, crosses the arch of the aorta on its left and passes beside the trachea to form the pulmonary plexus around the root of the left lung, as on the right side. Below the lung root it reforms on the left side, passes down at first on the left and then on the anterior aspect of the esophagus. While accompanying the esophagus, many branches are interchanged with the vagus nerve on the opposite side.

The cardiac branches are variable but are usually given off from the main nerve in the upper and lower parts of the neck, and at or shortly below the thoracic inlet. They tend to run with and may unite with the cervical sympathetic cardiac nerves which are derived from the superior, middle and stellate ganglia. These parasympathetic and sympathetic fibers, from both sides with some smaller contributions from the second to fourth thoracic sympathetic ganglia, together form the cardiac plexus on the anterior aspect of the bifurcation of the trachea. From there, parasympathetic and sympathetic fibers are distributed to the heart.

The pulmonary branches are mainly given off when the vagus nerve lies behind the root of the lung. They pass out to be distributed in the lung with sympathetic fibers from a loose plexus around the lower part of the esophagus. Parasympathetic fibers also sink into the wall of the esophagus and synapse with ganglion cells of the esophageal enteric plexus. This applies only to the lower part of the esophagus, the upper part having striated muscle in its wall. In the abdomen the tenth cranial nerve supplies the stomach, liver, gall bladder.

bile ducts, small intestine, pancreas and the large intestine almost to the splenic flexure. In the intestines the preganglionic fibers synapse in the enteric plexus. Further details are given in Chapter 14.

In spite of the extensive distribution of this nerve, autonomic abnormalities due to lesions of the tenth cranial nerve are not often easy to demonstrate except in complete lesions due to surgery (Chapter 14, p. 250). Abnormal regeneration can however cause troublesome disturbances of sweating (Chapter 10, p. 184), and loss of intramural ganglion cells can cause dilatation of the organ affected (Chapter 4, p. 63, and Chapter 14, pp. 256, 260-261).

SACRAL PARASYMPATHETIC NERVES

The parasympathetic supply to the urino-genital tract is described in Chapter 12. Sacral parasympathetic nerves also supply the large gut from just above the splenic flexure to the proximal part of the anus. The nerves reach the gut from the inferior hypogastric plexus and hypogastric nerves, and preganglionic fibers synapse in the enteric plexus. Megacolon may occur if the ganglion cells in this plexus are deficient (Chapter 14, p. 260-261).

Tables 1.1 and 1.2 follow overleaf

References

- BONICA J.J. (1968) Autonomic innervation of the viscera in relation to nerve block. *Anesthesiology* **29**, 793-813.
- CARMEL P.W. (1968) Sympathetic deficits following thalamotomy. *Archs Neurol.* **18**, 378-387.
- COOPER K.E. (1966) Temperature regulation and the hypothalamus. *Br. med. Bull.* **22**, 238-242.
- COOPER K.E. & KERSLAKE D.MCK. (1955) Vasoconstriction in the hand during electrical stimulation of the lumbar sympathetic chain in man. *J. Physiol.* **127**, 134-142.
- EDINGER L. (1885) Über den Verlauf der central Hirnnervenbahnen mit Demonstration von Präparaten. *Arch. Psychiat. Nervenkr.* **16**, 858-859.
- GOETZ R.H. (1948) The surgical physiology of the sympathetic nervous system with special reference to cardiovascular disorders. *Int. Abstr. Surg.* **87**, 417-439.
- KUNTZ A. (1953) *The Autonomic Nervous System*. Philadelphia: Lea and Febiger.
- LANGLEY J.N. (1898) On the union of cranial autonomic (visceral) fibres with the nerve cells of the superior cervical ganglion. *J. Physiol.* **23**, 240-270.
- MITCHELL G.A.G. (1953) *Anatomy of the Autonomic Nervous System*. London: Livingstone.
- MONRO P.A.G. (1959) *Sympathectomy*. London: Oxford University Press.
- PICK J. (1970) *The Autonomic Nervous System*. Philadelphia: Lippincott.
- WESTPHAL K.F.O. (1885) Über einen merkwürdigen Fall von periodischer Lähmung aller vier Extremitäten mit gleichzeitigen Erlöschen der elektrischen Erregbarkeit während der Lähmung. *Berl. klin. Wschr.* **22**, 489-491; 509-511.