A COLOR ATLAS OF
OROFACIAL HEALTH AND DISEASE
IN CHILDREN AND ADOLESCENTS

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DIAGNOSIS AND MANAGEMENT

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Preface

Pediatricians, general medical practitioners, general dental practitioners, dental specialists, dermatologists, oncologists, otorhinolaryngologists and many others are called upon to diagnose and treat children who have oral problems. The first edition of the Atlas, aimed at these practitioners, was extremely well received. We have improved, updated and expanded this second edition of Color Atlas of Orofacial Health and Disease in Children and Adolescents and hope that it will continue to be a useful source of assistance to health care professionals.

This edition, like the successful first edition, covers the presentation of the common orofacial disorders and a wide range of less common and some rare disorders. However, the number of authors has been increased with the inclusion of Professors Catherine Flaitz and Oslei Almeida, to geographically broaden the expertise, and a wider range of examples as well as new conditions have been added, increasing the pictorial content by over 20%. The text has also been updated and strengthened, with sections highlighting the common complaints, and now with a synopsis of diagnosis and management given under each condition.

Additions include choristoma, cocaine use, Coffin–Lowry syndrome, dentine dysplasia type II, foreign bodies, fragile X syndrome, graft versus host disease (GVHD), granular cell tumor, hypophosphatasia, Kabuki make-up syndrome, leukedema, lymphoepithelial cyst, median rhomboid glossitis, Morquio’s syndrome, papillary hyperplasia, peripheral ossifying fibroma, plasminogen deficiency, Prader-Willi syndrome, retrocuspid papilla, severe combined immunodeficiency syndrome (SCID), superficial mucoceles, vitamin D resistant rickets and whistling face syndrome.

The layout has also been improved with separation of the congenital and hereditary disorders into those with sole or prominent orofacial involvement from those where orofacial lesions are a less marked feature. Pain and neurological conditions have been added, together with more background, and a new chapter highlights the orofacial diseases found in major medical conditions – in children these are mainly the hematological disorders including immunodeficiencies, and iatrogenic disease resulting from medical and surgical therapy.

We have arranged material by first considering the healthy mouth and normal variants and then discussing the obviously congenital and heritable disorders though, of course, few other conditions affecting the orofacial region do not have some genetic basis. These other conditions are covered in the remainder of the Atlas, which outlines diseases of teeth and their sequelae, gingival and periodontal disease, mucosal disorders, salivary gland disorders, musculoskeletal disorders, pain and neurological disorders, and oral lesions in medical conditions such as hematological disorders, immunodeficiencies and other patients with special needs.

With a few exceptions we have restricted the Atlas to intraoral photographs and radiographs though, of course, not only must the whole mouth, head and neck always be examined, but the patient treated as a whole person. Because there is no reliably logical classification available we elected to present common complaints first and then to arrange material alphabetically within each of these sections. The number of illustrations of any particular condition is not necessarily a reflection of the importance of the disorder. Occasional illustrations show the fingers of ungloved hands often of the patient or carer; clinicians must of course now always wear gloves during patient care.

This Atlas provides treatment recommendations for the most common oral diseases tailored to the pediatric age group (Flaitz CM, Baker KA. Dent Clin N. Amer 2000; 44: 671–696). When possible, more than one drug alternative is given for each of the different oral conditions for an improved success rate. It is essential for the clinician to understand that this should be used as a guide for managing oral and perioral lesions in children and adolescents. Specific dosages and formularies of drugs may require modification in the young child and always must be checked with a pharmacopeia. Consultation with a primary care physician and pharmacist is often needed to ensure the best possible outcome, especially when immunosuppressive drugs are indicated. Most importantly, patients with oral lesions that do not respond to therapeutic protocols should be referred to the appropriate specialist for definitive diagnosis and/or treatment. Management may be a challenge for a variety of reasons. Compliance issues are affected by dosing schedules, ease of administration, taste and texture of the agent, cooperation of the child and unpleasant side-effects. Other important issues concerning the child patient include a paucity of drug studies demonstrating the effectiveness of the therapeutic protocols and the appropriate dosing of medications based on the weight and age and health of the child.

We are grateful to our patients and to colleagues who have helped us with some material, particularly Mr Brian Avery (Middlesborough, UK), Professor Robert Berkowitz (Washington, USA), Mr NE Carter (Newcastle, UK), Professor A Craft (Newcastle, UK), Dr John Eveson (Bristol, UK), Dr PH Gordon (Newcastle, UK), Dr Mark Griffiths (Bristol, UK), Dr John Jandinski (New Jersey, USA), Dr Jane Luker (Bristol, UK), Dr RI Macleod (Edinburgh, UK), Professor John Murray (Newcastle, UK), Dr Anita Nolan (Newcastle, UK), Dr June Nunn (Newcastle, UK), Professor Stephen Porter (London, UK), Professor Stephen Prime (Bristol, UK) and Miss CA Reid (Newcastle, UK). A few of the illustrations have also appeared in A Colour Atlas of Stomatology (C Scully and S Flint) or Colour Atlas of Oral Disease (C Scully, S Flint, SR Porter) and in this respect we are particularly grateful to Martin Dunitz (London), Dr Stephen Flint (Dublin) and Professor Stephen Porter (London).

June 2001
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The healthy mouth and normal variants
**TEETH**

The teeth (Figs 1–9) develop from ectoderm. At about the sixth week of intrauterine life the oral epithelium proliferates over the maxillary and mandibular ridge areas to form primary epithelial bands, which project into the mesoderm, and produce a dental lamina in which discrete swellings appear – the enamel organs of developing teeth. Each enamel organ eventually produces tooth enamel, and the mesenchyme, which condenses beneath the enamel organ (actually neuroectoderm), forms a dental papilla, which produces the dentine and pulp of the tooth. Tooth development begins in the fetus at about 28 days in utero. Indeed, all the deciduous and some of the permanent teeth commence development in the fetus. The enamel organ together with the dental papilla constitute the tooth germ, and this becomes surrounded by a mesenchymal dental follicle, from

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**Figures 1–3.** The primary dentition. Primary teeth are whiter, smaller and more bulbous than permanent teeth. Except in severely crowded mouths there is normally some spacing between primary anterior teeth.

**Figures 4–6.** The mixed dentition. Permanent incisors have succeeded primary incisors. The permanent molar teeth (fissure-sealed) have erupted behind the primary molar teeth.
Figures 7–9. The permanent dentition. Permanent canine teeth have succeeded primary canines and permanent premolars have succeeded the primary molars.

which the periodontium forms – ultimately to anchor the tooth in its bony socket. Mineralization of the primary dentition commences at about 14 weeks in utero and all primary teeth are mineralizing by birth. Permanent incisor and first molar teeth begin to mineralize at, or close to, the time of birth, mineralization of other permanent teeth starting later. Tooth eruption occurs after crown formation and mineralization are largely complete but before the roots are fully formed (see Figs 10, 11 and Table 3).

There are ten deciduous (primary or milk) teeth in each jaw: all are fully erupted by the age of about 3 years (Figs 10, 11). The secondary or permanent teeth begin to erupt at about the age 6–7 years and the primary teeth begin to be slowly lost by normal root resorption. However, some primary teeth may still be present at the age of 12–13 years. The full permanent dentition consists of 16 teeth in each jaw: normally most have erupted by about 12–14 years of age, but the last molars (third molars or wisdom teeth), if present, often erupt later or may impact and/or never appear in the mouth.

Common sensation from the teeth is conveyed by the trigeminal nerve. The upper teeth are supplied by the superior alveolar nerves (branches of the maxillary division of the trigeminal nerve) while the lower teeth are supplied by the inferior alveolar branch of the mandibular division of the trigeminal nerve.
Variation in tooth eruption times
A delay in eruption of up to 12 months may be of little or no significance in an otherwise healthy child. Localized variations often result from local factors, such as impaction against a tooth in the path of eruption caused, for example, by insufficient space in the dental arch. Impaction and ectopic positioning most often occur in the permanent dentition, especially in the third molar, second premolar and canine regions, because these are the last teeth to erupt.

Teething
Eruption of primary teeth may be preceded by a bluish gingival swelling, usually caused by a transient hematoma, rarely an eruption cyst, which usually ruptures spontaneously (see p. 85). ‘Teething’ describes tooth eruption associated with localized swelling and erythema of the alveolar ridge. Less frequently, irritability, disturbed sleep, cheek flushing, drooling, mild pyrexia and/or a circumoral rash may develop. Teething does not cause mouth ulcers, diarrhea, otitis media or respiratory infections, such as bronchitis; however, these diseases may occur coincidentally.

Diagnosis
Diagnosis is clinical.

Management
Reassurance only is required. Systemic analgesics, such as acetaminophen/paracetamol, and teething gels may offer some relief. The child should be evaluated for other medical causes if marked or prolonged constitutional signs and symptoms are present.

Figure 12. Prominent palatal cusp on a first maxillary permanent molar (cusp of Carabelli).

Cusp of Carabelli
Cusp of Carabelli (Fig. 12) is an anatomical variant with an accessory palatal cusp on the upper deciduous or permanent molars.

Diagnosis
Diagnosis is clinical.

Management
Reassurance only is required.
MUCOSA

Racial pigmentation of mucosa
There is no direct correlation between skin color and oral pigmentation (Figs 13, 14), which may be seen in races with increased melanin pigmentation and others – such as those of southern European descent. Although any site may be affected, the attached gingiva and buccal mucosa are the most common sites of involvement. The intensity of the oral pigmentation often increases with age and local irritation.

Diagnosis
Diagnosis is clinical.

Management
Reassurance only is required.

Lining mucosa
Lining mucosa (buccal, labial and alveolar mucosa, floor of mouth, ventral surface of tongue, soft palate, lips) is nonkeratinized with broad rete ridges and connective tissue papillae and abundant elastic fibers in the lamina propria and is fairly mobile. Depending on the race/ethnicity of the child, the pink mucosal coloring may be interspersed with patches of tan, brown, gray or black.

Leukoedema
Leukoedema (Figs 15, 16) is a benign congenital condition resulting in a filmy white and wrinkled appearance of the mucosa. Considered to be a variant of normal, this common entity is more prominent in children of color. It is most obvious on the buccal and labial mucosa with a bilateral distribution and tends to fade or disappear when the mucosa is stretched.

Diagnosis
Diagnosis is clinical.

Management
Reassurance only is required.
Sebaceous glands (Fordyce granules or spots)

Fordyce granules or spots (Figs 17, 18) may be seen as creamy-yellow dots along the border between the lip vermilion and the oral mucosa or in the buccal mucosa, and are not associated with hair follicles. They are not usually clinically evident until after the age of 3 years, increasing during puberty and then again in later adult life. Probably 50–80% of the adult population have them, but they are often invisible in the young child. They are totally benign, although they may be confused with thrush in a child.

Diagnosis
Diagnosis is clinical.

Management
Reassurance only is required.

Figures 17, 18. Fordyce granules.
Masticatory mucosa
Masticatory mucosa (hard palate, gingivae; Figs 19–23), is adapted to pressure forces and friction and keratinized with numerous tall rete ridges and connective tissue papillae and little submucosa; it is tightly bound down. The alveolar bone, which bears the teeth, is covered by the gingivae, or gum which, in health are pink, stippled and tightly bound down, and form a close fitting cuff, with a small sulcus (gingival crevice) around the neck of each tooth. Depending on the race/ethnicity of the child, the pink mucosal coloring may be interspersed with patches of tan, brown, gray or black.
Retrocuspid papilla
A retrocuspid papilla is a sessile nodule (Fig. 24) with a smooth or stippled surface that occurs bilaterally on the lingual attached gingiva, adjacent to the mandibular canines. Occurring in 50% of children, this normal gingival variant typically regresses with age.

Diagnosis
Diagnosis is clinical.

Management
Reassurance only is required.

Specialized mucosa
Specialized mucosa on the dorsum of the tongue, adapted for taste and mastication, is keratinized, with numerous rete ridges and connective tissue papillae, abundant elastic and collagen fibers in the lamina propria and no submucosa (Fig. 25). The tongue is divided by a V-shaped groove, the sulcus terminalis, into an anterior two-thirds and a posterior one-third. Various papillae on the dorsum include the filiform papillae, which cover the entire anterior surface and form an abrasive surface to control the food bolus as it is pressed against the palate, and the fungiform papillae. The latter are mushroom-shaped, pink or red structures covered by nonkeratinized epithelium. In children of color, these papillae may be tan or brown (Fig. 14). They are scattered among the filiform papillae and have taste buds on their surface. Adjacent and anterior to the sulcus terminalis are eight to twelve large circumvallate papillae, each surrounded by a deep groove into which open the ducts of serous minor salivary glands. The lateral walls of these papillae contain taste buds.

The foliate papillae consist of four to eleven parallel ridges, alternating with deep grooves in the mucosa, on the lateral margins on the posterior part of the tongue. There are taste buds on their lateral walls. These papillae may become inflamed (foliate papillitis; Fig. 26).

The lingual tonsils are found as oval prominences with intervening lingual crypts lined by nonkeratinized epithelium. They are part of Waldeyer’s oropharyngeal ring of lymphoid tissue. Frequently it is not possible to distinguish between the lingual tonsils and foliate papillae.
**BONE**

The jaw bones (mandible and maxilla) underlie alveolar bone, which is essential support for the teeth. The fibers of the periodontal ligament attach at one end to the alveolus and at the other through cementum to the dentine surface of the tooth root. In the absence of teeth, the alveolar bone fails to develop or atrophies.

**Torus mandibularis and torus palatinus**

Tori (Fig. 27) are common benign bony enlargements of developmental origin, especially seen in the Asian population. There may be some association with parafunction, such as bruxism.

Mandibular tori are uni- or bilateral bony enlargements lingual to the lower premolars. Palatal tori are common bony masses, typically in the midline vault of the hard palate.

**Diagnosis**

Diagnosis is clinical.

**Management**

Reassurance only is required.

**Temporomandibular joints**

The temporomandibular joints (TMJs) are diarthrodial joints between the condylar fossa in the temporal bone, and the mandibular condyle. Masticatory movements are controlled by the medial and lateral pterygoid muscles, the masseter, the temporalis and the mylohyoid and digastric muscles.

**MUSCLES**

The masticatory muscles include the masseters, temporalis, and pterygoid muscles, with a lesser contribution from digastric and mylohyoid muscles. They are controlled mainly by the trigeminal nerve. Muscles of facial expression are mainly the buccinator, platysma, frontalis and orbicularis oris and orbicularis oculi muscles. They are controlled by the facial nerve.

**SALIVARY GLANDS**

The major salivary glands are the parotid, submandibular and sublingual glands, but minor glands are scattered throughout the oral cavity, particularly in the lower lip and palate. The secretions from the different glands differ – for example the submandibular saliva contains far more mucus than parotid saliva, which is serous. Secretion is controlled via the glossopharyngeal (parotid) or chorda tympani (submandibular/sublingual) nerves. Normally clear saliva can be expressed from the major ducts or stimulated with citric acid and at rest there is a pool of clear saliva in the floor of the mouth. A ‘dental’ mirror slides easily over the oral mucosa when salivation is normal.
NERVE SUPPLY

Trigeminal nerve
Common sensation from the orofacial region is conveyed by the trigeminal nerve. This nerve supplies sensation to most of the scalp, face and mouth. The two roots of the trigeminal nerve emerge at the pons and enter Meckel’s cave at the tip of the petrous temporal bone and the foramen lacerum, where the ganglion (Gasserian ganglion) of the sensory root lies. Motor fibers run only with the mandibular division.

Maxillary division
The maxillary division of the trigeminal nerve runs for a short distance in the base of the cavernous sinus, giving off a meningeal branch to the dura mater of the middle cranial fossa. It leaves the middle cranial fossa through the foramen rotundum in the greater wing of the sphenoid bone to enter the pterygopalatine fossa, which it crosses to leave through the inferior ophthalmic fissure as the infraorbital nerve.

■ posterior superior alveolar nerve (to the upper molars and part of the maxillary antrum)
■ palatine nerves (to the palate)
■ nasal nerves (sphenopalatine nerves)
■ pharyngeal nerves to the mucous membrane of the upper pharynx
■ zygomatic nerve.

The zygomatic nerve enters the orbit by the inferior ophthalmic fissure and divides into a posterior (temporal) branch, which enters the frontal lobe of the temporal fossa behind the orbital cavity and then pierces the temporal fascia at the anterior margin of the temporal muscle to supply the skin between the eye and the ear. The other anterior (facial) branch appears through foramina on the facial surface of the zygomatic bone and supplies the overlying area of the skin. Parasympathetic fibers derived from the pterygopalatine ganglion run with the zygomatic nerve and join the lacrimal nerve as secretomotor fibers to the lacrimal gland.

The branches of the infraorbital nerve are:
■ the middle and anterior superior dental nerve to the maxillary antrum, upper incisor, canine and premolar, teeth and buccal gingiva
■ three terminal branches, the labial, nasal and palpebral nerves, which supply the upper lip, cheek and lower eyelid.

Mandibular division
The mandibular division of the trigeminal nerve contains sensory and motor fibers. It leaves the middle cranial fossa through the foramen ovale in the greater wing of the sphenoid bone to give off branches to the tensor palati and tensor tympani muscles, the otic ganglion, the medial pterygoid muscle and a recurrent sensory branch (nervus spinosus), which passes through the foramen spinosum to supply dura mater in the middle cranial fossa. The mandibular division lies on the outer surface of the tensor palati muscle with the otic ganglion between the nerve trunk and the muscle. Behind it lies the middle meningeal artery and laterally is the upper head of the lateral pterygoid muscle. The auditory (eustachian) tube lies close to the nerve trunk as it emerges from the foramen ovale.

The mandibular nerve trunk then divides into:
■ a larger posterior division, giving off the auriculotemporal, inferior alveolar and lingual branches. The inferior alveolar nerves provides sensation to the lower teeth and associated structures.
■ a smaller anterior division, which provides motor supply to the temporal, lateral pterygoid and masseter muscles and continues on as the sensory buccal nerve (long buccal nerve).

Taste sensation
The special sense of taste is mediated by specialized cells related to various supporting or sustentacular cells. Taste buds are found in the mucous membrane of the tongue, soft palate, fauces and pharynx, and in the newborn, on the lips and cheeks. Taste buds are oval bodies made up of groups of neuroepithelial and supporting cells. The neuroepithelial cells are rod-shaped with a peripheral hairlike process projecting into the taste pores at the surface of the overlying mucous membrane. The terminal branches of the nerve fibers subserving taste end in close relationship to these special cells. Studies of taste thresholds in human subjects commonly use sucrose for the sweet taste, vinegar or citric acid to produce sour taste sensations, and sodium chloride for the taste of salt. Detection and recognition thresholds can be measured by applying the selected solution to precise regions of the oral mucosa. The tongue is most sensitive for salt and sweet tastes. Sour and bitter tastes can be recognized on the tongue, but not as well as by the palatal mucosa. Salt and sweet tastes can also be appreciated on the palate, but higher solution concentrations are required.

Taste buds on the tongue are on the fungiform, circumvallate and foliate, but not filiform, papillae. The four fundamental varieties of taste sensation (sweet, bitter, sour and salt) do not appear to be detected by structurally different taste buds. The cells of the taste buds undergo continual renewal, with a life span of about 10 days. Renewal is altered by nutrition, hormonal status, age, drugs, radiation and other factors.

Taste sensation from the anterior two-thirds of the tongue and the palate is mediated by the lingual and palatine nerves, respectively. Taste fibers from the anterior two-thirds of tongue and secretomotor parasympathetic fibers to the submandibular and sublingual salivary glands pass with the lingual nerve for part of its distal course. Taste from the anterior two-thirds of the tongue is mediated via the chorda tympani nerve and runs with the facial nerve.

Taste fibers from the posterior one-third of the tongue pass in the glossopharyngeal nerve and to the nucleus solitarius. The taste fibers pass into the brain stem along the chorda tympani and greater superficial petrosal branches of the facial nerve, respectively. The central processes of the chorda tympani nerve conveying taste impulses from the anterior two-thirds of the tongue pass, as the nervus intermedius, to the solitary tract, through which the taste impulses are carried to the nucleus solitarius.

The taste buds of the epiglottis are innervated by the vagal nerve fibers whose cell bodies are situated in the nodose ganglion and whose central processes terminate once again in the nucleus solitarius.

These various ‘taste’ fibers passing to the nucleus solitarius form the solitary tract (tractus solitarius). The secondary neurones of the pathway for taste (from the nucleus solitarius to the thalamus) cross to be included in the medial lemniscus of the opposite side and, on reaching the level of the thalamus, end along with other secondary fibers from the head region. Tertiary, or third order taste fibers, project to the inferior part of the postcentral gyrus and the adjacent cortex of the insula. Pontine neurones also project to the ‘feeding area’ in the hypothalamus.

The flavor of food results from chemical stimulation of both taste buds and olfactory neurones. Free nerve endings in the nose, mouth and throat also contribute to an appreciation of food and there is a strong element from higher centers.
Motor supply to the orofacial region

Motor supply to the orofacial region is complex. The masticatory muscles are innervated mainly by the trigeminal nerve, the palatal musculature mainly by the glossopharyngeal and vagal nerves and the tongue muscles mainly by the hypoglossal nerve. The muscles of facial expression receive motor innervation from the facial nerve.

Speaking

The act of speaking is a highly coordinated sequence of movements of the muscles of respiration, larynx, pharynx, palate, tongue and lips. Articulation and phonation are therefore under direct control of the vagus (and adjacent nerves), facial nerve and hypoglossal nerves. As with all muscle activity, phonation and articulation are under higher control from pyramidal and extrapyramidal influences.

Swallowing

In normal swallowing, the activities of the striated muscle of the pharynx and upper esophagus are integrated with those of the smooth muscle of the lower esophagus.

The initial phase of swallowing is voluntary, under the control particularly of the glossopharyngeal nerve, with the vagus controlling further phases of swallowing. Normal swallowing is dependent on adequate mastication of large food masses, lubrication with fluids and saliva, unobstructed lumens of the pharynx and esophagus, and normal coordinated neuromuscular mechanisms of swallowing.

A sphincter formed by the cricopharyngeus prevents air from filling the esophagus during respiration while a lower esophageal sphincter stops gastric reflux.

Glossopharyngeal (IX) cranial nerve

The glossopharyngeal cranial nerve is attached to the medulla and passes through the jugular foramen where it has the jugular and petrosal ganglia. It then runs in the carotid sheath between the internal carotid artery and internal jugular vein. It leaves the carotid sheath and runs on the stylopharyngeus muscle deep to the external carotid and ascending palatine arteries to the upper border of the middle constrictor muscle, where it enters the oropharynx.

The chief branches of the glossopharyngeal nerve are:

- The tympanic branch, which joins with a branch of the facial nerve to form the tympanic plexus from which arises sensory branches to the mucous membrane of the middle ear, the tympanic antrum and the eustachian tube, and the lesser superficial petrosal nerve, which contains secretomotor fibers derived from the glossopharyngeal nerve and destined for the otic ganglion and the parotid salivary gland.
- The carotid branch, which descends to the carotid sinus and carotid body, carrying autonomic fibers involved in blood pressure regulation.
- The motor branch to the stylopharyngeus muscle.
- Pharyngeal (sensory) branches to the pharyngeal mucous membrane.
- Tonsillar branches, which ascend to the upper part of the pharynx and eustachian tube.
- Lingual branches, which pass to the posterior one-third of the tongue.

Vagus (X) and accessory (XI) cranial nerves

The vagus cranial nerve arises at the medulla and passes through the jugular foramen where it has a superior ganglion and lower down a large inferior ganglion. The cranial part of the accessory nerve joins the vagus at the inferior ganglion and fibers are distributed with the pharyngeal and laryngeal branches of the vagus. The ganglia also communicate with the facial, glossopharyngeal, hypoglossal and sympathetic nerves. The vagus enters the carotid sheath between the internal jugular vein and the internal carotid artery to descend through the neck. On the right side the vagus enters the thoracic cavity after crossing the first part of the subclavian artery; on the left side after descending between the left common carotid and subclavian arteries it crosses in front of the aortic arch. On the right side the recurrent laryngeal branch loops around the subclavian artery; on the left side it passes around the arch of the aorta.

In the thorax, the vagus passes behind the hilum of the lung to form the esophageal plexus, from which trunks pass through the diaphragm in front of (left vagus) and behind (right vagus) the esophagus. The left vagus is distributed chiefly to the anterior surface and lesser curvature of the stomach, to the liver and to the gall bladder. The right vagus is distributed mainly to the stomach and midgut derivatives with branches to the celiac, splenic and renal plexuses.

The branches of the vagus nerve in the neck are:

- A recurrent meningeal branch to the dura mater of the posterior cranial fossa.
- The auricular nerve (of Arnold), which supplies the mucous membrane of the external auditory canal and the outer surface of the tympanic membrane.
- The pharyngeal branch, which contains motor fibers, mainly from the cranial part of the accessory nerve, to the constrictor muscles of the pharynx, and the palatopharyngeus, levator palati, palatoglossus and uvular muscles.
- The superior laryngeal nerve, which consists chiefly of fibers derived from the cranial part of the accessory nerve and gives an internal laryngeal (sensory) branch, and a motor branch (the external laryngeal) to the cricothyroid and inferior constrictor muscles.
- Upper and lower cardiac branches supplying autonomic (parasympathetic) control to the heart.
- Recurrent laryngeal nerves, which are motor to the muscles of the larynx (except for the cricothyroid) and sensory to the laryngeal mucous membrane below the vocal folds and the mucous membrane of the trachea. Some motor fibers to the lower part of the inferior pharyngeal constrictor may run in the recurrent laryngeal nerves.

The cranial part of the accessory nerve arises from the medulla and the spinal part from the spinal cord as far down as the attachment of the fifth cervical nerve. The spinal portion enters the foramen magnum and unites with the cranial part to form a common trunk, which passes through the jugular foramen between the internal jugular vein and the internal carotid artery. The cranial part then separates to join the vagus and is distributed through the pharyngeal and laryngeal branches of the vagus to pharyngeal, soft palatal and laryngeal muscles. The spinal part passes backwards to supply sternomastoid and trapezius muscles.

Hypoglossal (XII) cranial nerve

The hypoglossal nerve arises from the medulla and leaves the cranial cavity through the anterior condylar canal to enter the carotid sheath where it is joined by branches from the first and second cervical nerves and from the superior cervical sympathetic ganglion. The nerve turns forwards, hooking around the origin of the occipital artery and lying superficial to the external carotid, facial and lingual arteries in the carotid triangle. It leaves the carotid triangle deep to the digastric and stylohyoid muscles and enters the floor of the mouth between the mylohyoid and
hyoglossus muscles, communicating with the lingual nerve and penetrating the genioglossus muscle to enter the tongue below the sublingual gland. Within the tongue it supplies all muscles, except the palatoglossus.

**Facial (VII) cranial nerve**

The nerve controlling movements of the facial musculature is the facial cranial nerve. Lesions of this nerve (lower motor neurone lesions) or its central connections (upper motor neurone lesions) or muscle disease, can lead to facial weakness. The facial nerve is attached to the brain stem at the upper end of the medulla by a motor and sensory root, which cross the subarachnoid space above the vestibulocochlear (VIII) cranial nerve and enter the internal auditory meatus where they unite and enter the facial canal, winding through the petrous temporal bone between the semicircular canals of the inner ear behind and the cochlear in front, to reach the medial wall of the middle ear. Here it takes a sharp turn backward (the genu) and runs along the wall of the middle ear cavity.

At the genu is the sensory nucleus (geniculate ganglion) from where a branch containing secretomotor sympathetic fibers, the greater superficial petrosal nerve (sympathetic fibers from the superior cervical ganglion) to form the nerve of the pterygoid canal (vidian nerve). In the pterygopalatine fossa this nerve joins the pterygopalatine (sphenopalatine) parasympathetic ganglion. In its course through the tympanic plexus, to which the glossopharyngeal nerve also contributes, the facial nerve gives off the chorda tympani branch, which enters the middle ear cavity, crosses the tympanic membrane and leaves through the squamotympanic fissure to join the lingual nerve. The chorda tympani nerve contains taste fibers (sensory) from the anterior two-thirds of the tongue and secretomotor parasympathetic fibers destined for the submandibular ganglion – which supplies submandibular and sublingual salivary glands.

The facial nerve then pierces the fascial sheath of the parotid gland and divides into:

- an upper branch giving rise to temporal, zygomatic and upper buccal branches;
- a lower branch giving rise to lower buccal, mandibular and cervical branches.

The temporal branch supplies the anterior and superior auricular, frontalis, upper part of orbicularis oculi and corrugator supercilii muscles. The zygomatic branch supplies the orbicularis oculi. The buccal branches supply buccinator, muscles of the upper lip, risorius and muscles of the nose. The mandibular branch supplies the lower lip and mentalis muscles. The cervical branch supplies the platysma and may send a branch to join the mandibular branch.