

Duilio Della Libera

Core Messages

- The structural organisation of the larynx and some of its individual anatomical components determine the way in which the carcinoma spreads. This can be clearly seen from the studies of histological macrosections of the neoplastic larynx.

12.1 Laryngeal Structure and Neoplastic Spreading

The subdivision of the larynx into sites and subsites (supraglottis, glottis, and subglottis) is amply justified from the oncological viewpoint because it is useful for defining the prognosis and planning therapy for the laryngeal carcinoma. The means of neoplastic growth depend partly on the intrinsic properties of the tumour population but also on their site of origin and on the anatomical structures involved in their growth [1, 2]. In the process of neoplastic spread, assessed according to three-dimensional criteria [3], definite development vectors prevail which, at least in the initial phases, are conditioned by the anatomical permeability of the site of origin and by the presence and conformation of submucosal compartments and of ligamental and osteocartilaginous barriers [4]. The different means of growth also involve a different pattern of metastatic spread, for different access to the different laryngeal lymphatic networks.

For each laryngeal site, series of coronal and sagittal macrosections of pathological larynxes will be proposed, with the aim of identifying and discussing their tumoural growth patterns.

12.1.1 Infiltration of the Cartilages

Infiltration of the laryngeal cartilages is most frequent in their ossified portion. Bone cavities are occupied by adipose tissue and by well-vascularised hematopoietic tissue which do not offer any important mechanical resistance to neoplastic growth.

The hyaline cartilage has a compact organisation and is without blood vessels, so it offers greater resistance [1].

The ossified areas are present, from the third decade of life, at the point of insertion of ligaments, membranes, and muscles, mostly as a response to mechanical stress stimuli. In these areas, there is an interruption of the perichondral barrier and the development of a rich vascular network [5].

The most important sites of invasion of the cartilage are (1) the dihedral angle of the thyroid cartilage where the tendon of the anterior commissure is inserted, (2) the insertions of the cricothyroid membrane in the corresponding cartilages, (3) the anterior portion of the thyroid lamina near the origin of the vocal muscle, (4) the posterior margin of the thyroid lamina adjacent to the piriform sinus, and (5) the cricoarytenoid articular capsule [9].

12.2 Supraglottic Carcinoma

The definition of the site of origin of the supraglottic carcinoma is not easy because by the time of diagnosis, the tumour is frequently already evolved, involves a broad surface of mucosa, and may develop asymmetrically in both surface and depth. In less than a quarter of cases of supraglottic carcinoma, only one laryngeal subsite is involved at the moment of diagnosis, while in the remaining three quarters of cases, two or more subsites are involved. There are various classifications of supraglottic carcinoma, depending on the site of origin. We shall adopt a simple classification which subdivides supraglottic carcinomas into median (with symmetrical and asymmetrical development), lateral, and marginal.

Supraglottic carcinomas tend to grow for a long time within the submucosal spaces (preepiglottic space and superior paraglottic space), determining the late spread to the underlying glottic region as though there were a kind of barrier between the two laryngeal sites. Really, in the initial phases of their development, vestibular neoplasias are impeded medially by the vocal ligament and laterally by the ventricle. They tend to infiltrate the paraglottic spaces locally which, at this level, behave like real expansion vessels of neoplastic growth. The glottic extension is therefore a late event with the exception of median neoplasias which involve the region of anterior supracommissural region at the level of the pedicle of the epiglottis or which grow laterally to Morgagni's ventricle.

12.2.1 Median Supraglottic Carcinoma with Symmetrical Development

It originates from the central region of the laryngeal side of the epiglottis (Fig. 12.1) and extends symmetrically in all directions [6]. In the advanced phases, the ventricular bands and the aryepiglottic folds may be involved. In over two thirds of cases, the neoplasia prematurely invades the preepiglottic

Fig. 12.1 Medial symmetrical supraglottic carcinoma (axial macrosection). 1 Superficial carcinoma of laryngeal surface of epiglottis, 2 epiglottis, 3 preepiglottic space, 4 hyoid bone, 5 aryepiglottic fold

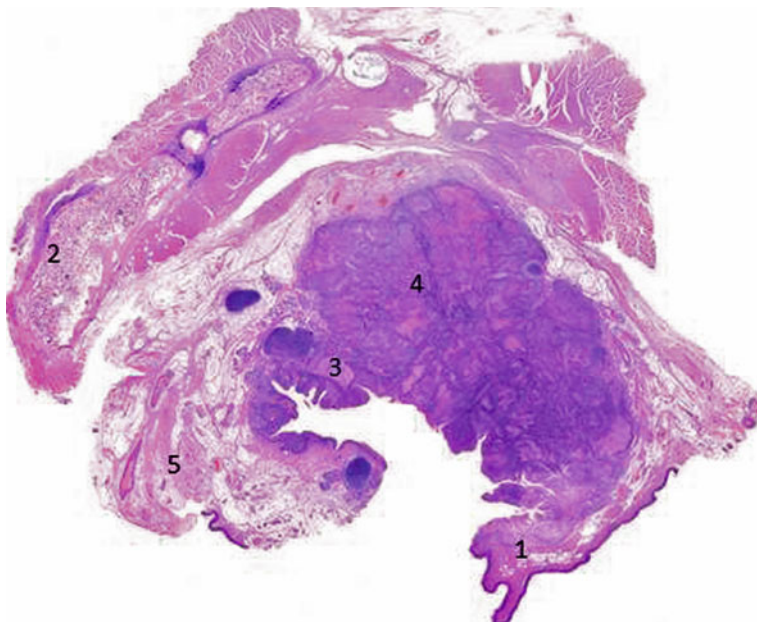


Fig. 12.2 Advanced medial supraglottic carcinoma (axial macrosection). 1 Aryepiglottic fold, 2 hyoid bone, 3 epiglottis, 4 neoplastic invasion of preepiglottic space, 5 superior paraglottic space

Fig. 12.3 Multiregional supraglottic carcinoma (coronal macrosection). *1* Advanced spread of multiregional supraglottic carcinoma with invasion of preepiglottic and paraglottic spaces and oropharyngeal region through hyoepiglottic membrane, *2* retrolingual region, *3* tongue, *4* hyoid bone, *5* thyroid cartilage, *6* cricoid cartilage, *7* epiglottis, *8* ventricular fold, *9* Morgagni's ventricle, *10* superior paraglottic space, *11* vocal cord, *12* cricothyroid space



space (Fig. 12.2) through the glandular foramina of the epiglottis, either encircling its lateral edges or destroying it. From the preepiglottic space [7, 8], the tumour may extend through the hyoepiglottic membrane to the adjacent oropharynx in the region of the glossoepiglottic valleculae and to the base of the tongue (Fig. 12.3). This fact is important because, as well as conditioning a less conservative surgical treatment, it leads to a new lymphatic drainage in the lateral lingual system of the oropharynx. Lymph nodal metastases are present in about 25 % of cases; they are late and often bilateral. In the advanced forms, the neoplasia often affects the superior paraglottic space [9], sometimes with marked asymmetries between surface and deep development. The thyroid cartilage is infiltrated in about 5 % of cases in those neoplasias whose development involves the anterior area of confluence of the two ventricular bands that

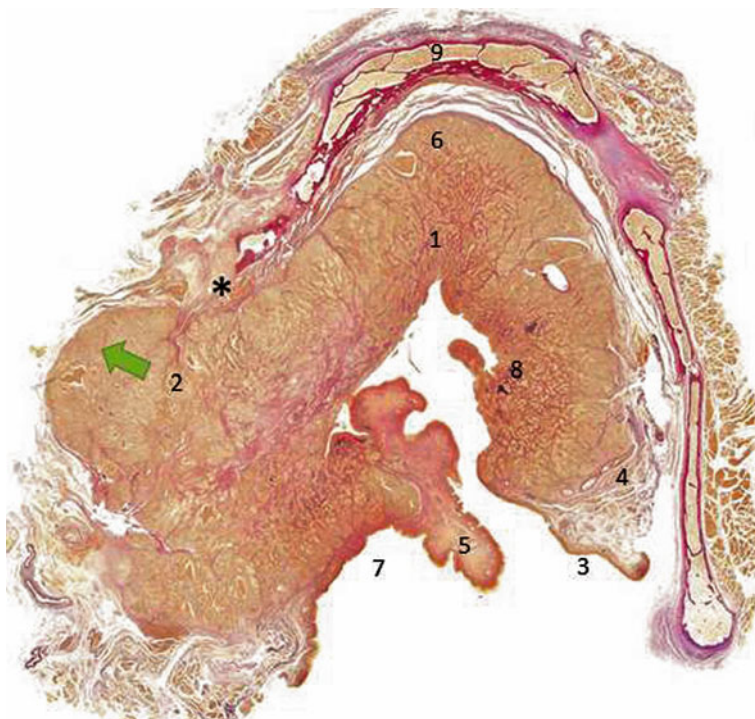


Fig. 12.4 Multiregional supraglottic carcinoma (axial macrosection). 1 Extensive vestibular spread of vestibular carcinoma with invasion of hyoid bone (*) and lateral lamina of thyroid cartilage, 2 lateral extralaryngeal diffusion, 3 ventricular fold, 4 superior paraglottic space, 5 aryepiglottic fold, 6 preepiglottic space, 7 piriform sinus, 8 epiglottis, 9 hyoid bone

correspond to the pedicle of the epiglottis and the underlying thyroepiglottic ligament (Fig. 12.4).

12.2.2 Median Supraglottic Carcinoma with Asymmetrical Development

Carcinoma of the larynx corner (Fig. 12.5) involves the join between the median portion of the epiglottis and a ventricular fold [10]. It is the most common supraglottic neoplasia (50 % of cases). In over 90 % of cases, the neoplasia makes an early infiltration of the submucosal membrane of the laryngeal vestibule, often with a marked asymmetry between the superficial mucosal component and the infiltrating component, which more frequently prevails over the former (Fig. 12.6). Due to their particular position on the border between the preepiglottic space and the superior paraglottic space, these neoplasias can grow both caudally and ventrally. In the latter



Fig. 12.5 Ventrolateral supraglottic carcinoma (larynx corner carcinoma) (axial macrosection). 1 Angular region, between ventricular fold and epiglottis, 2 epiglottis, 3 neoplastic invasion of preepiglottic space, 4 preepiglottic space, 5 aryepiglottic fold, 6 hyoid bone

case, anterosuperior growth leads to the invasion of the preepiglottic space (Fig. 12.7), often with destruction of the lateral margin of the epiglottis (Fig. 12.8). In some cases, through the preepiglottic space, they reach the adjoining oropharyngeal region of the glossoepiglottic valleculae and the base of the tongue. The second preferential direction of growth is posteroinferior, with infiltration of the superior paraglottic space and of the arytenoid region. The extension of the neoplasia beyond the lateral angle of Morgagni's ventricle is often accompanied by infiltration of the lateral lamina of the thyroid cartilage (Fig. 12.9), sometimes with extension to the medial wall of the piriform sinus. In this neoplasia, lymph node metastases are present in about half of the cases and are generally homolateral with the neoplasia.

12.2.3 Lateral Supraglottic Carcinoma

It arises in the mucosal surface of the ventricular folds in the area corresponding to the quadrangular membrane and in Morgagni's ventricle (Fig. 12.10). It often has a carpet-like surface growth pattern, and in half the cases, it infiltrates the superior paraglottic space [11]. The median vestibular regions of the larynx are involved by

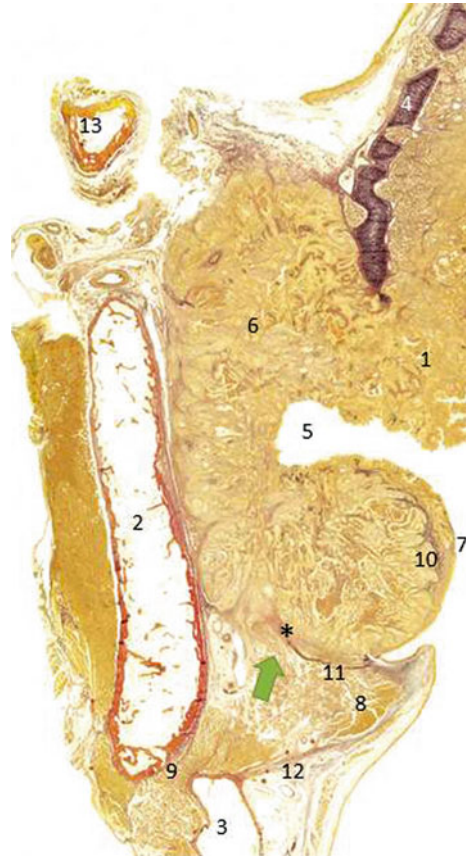


Fig. 12.6 Ventrolateral supraglottic carcinoma (larynx corner carcinoma) (coronal macrosection). 1 Advanced spread of lateral supraglottic carcinoma with invasion of paraglottic spaces through quadrangular membrane and preepiglottic space, 2 thyroid cartilage, 3 cricoid cartilage, 4 epiglottis, 5 ventricular fold, 6 Morgagni's ventricle, 7 superior paraglottic space, 8 vocal cord, 9 cricothyroid space, 10 glossoepiglottic valleculae

neoplastic growth at a late stage. Lymph nodal metastases are present in about one third of the cases and are generally homolateral.

As regards carcinoma of the ventricle [12], there is no general agreement on its definition. Some authors in fact use the term *ventriculosaccular* as a synonym for *transglottic*, while others consider it an independent neoplasia originating from the mucosa that covers the ventricle (Fig. 12.11). The primitive origin of the neoplasia of the ventricular mucosa is a rare event, considering the rarity of finding squamous metaplasia of the cylindrical ciliated epithelium of a respiratory type which habitually covers the ventricle. It is thought that the rare neoplasias confined only to the

Fig. 12.7 Ventrolateral supraglottic carcinoma (coronal macrosection). *1* Advanced spread of ventrolateral supraglottic carcinoma with invasion of superior paraglottic space and Morgagni's ventricle; glottic–supraglottic boundaries (*), *2* thyroid cartilage, *3* cricoid cartilage, *4* epiglottis, *5* Morgagni's ventricle, *6* superior paraglottic space, *7* ventricular fold, *8* vocal cord, *9* cricothyroid space, *10* quadrangular membrane, *11* ventricular ligament, *12* elastic cone, *13* hyoid bone



ventricular area, sometimes associated with the development of a secondary laryngocele due to ectasia of the ventricular saccule, may derive from metaplastic areas of the ventricular mucosa or from the join between the superior edge of the vocal cord and the ventricle floor.

12.2.4 Marginal Supraglottic Carcinoma

It arises in the marginal region of the laryngeal vestibule [13, 14], that is, at the level of the free margin of the epiglottis, the aryepiglottic folds, and the apex of the arytenoid. In the first case, it may spread to the lingual side of the epiglottis, to the oropharyngeal region of the glossoepiglottic valleculae, and to the laryngeal side of the epiglottis.

The neoplasia of the aryepiglottic fold (Figs. 12.12 and 12.13) generally presents a surface growth on the medial wall of the piriform sinus or in the region of the

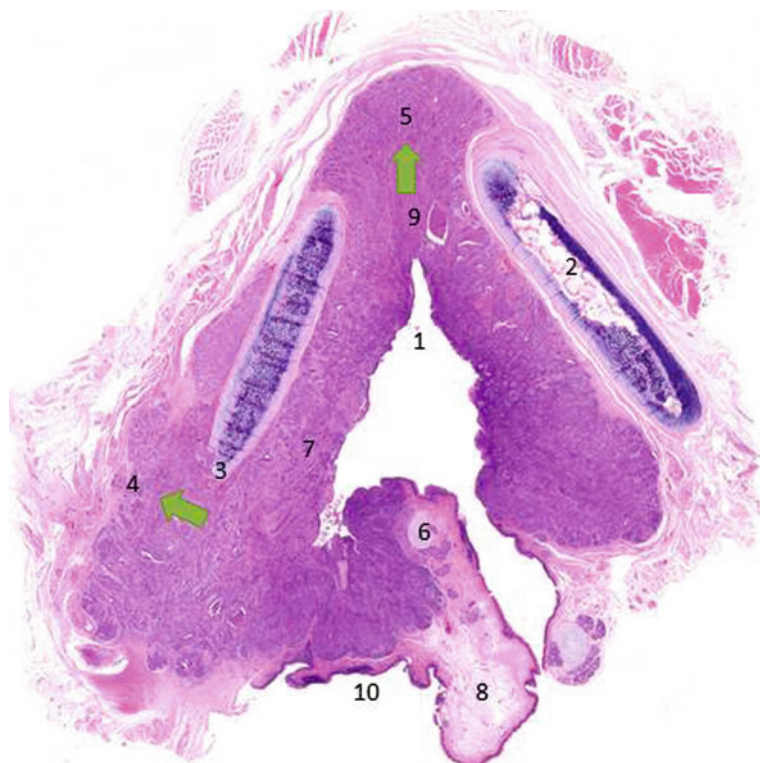


Fig. 12.8 Multiregional supraglottic carcinoma (axial macrosection). 1 Extensive vestibular spread of supraglottic carcinoma with invasion of paraglottic space, 2 thyroid cartilage, 3 infiltration of lateral lamina of thyroid cartilage, 4 lateral extralaryngeal diffusion, 5 medial extralaryngeal diffusion through anterior thyroid cartilage incisure, 6 ventricular fold, 7 superior paraglottic space, 8 aryepiglottic fold, 9 preepiglottic space, 10 piriform sinus

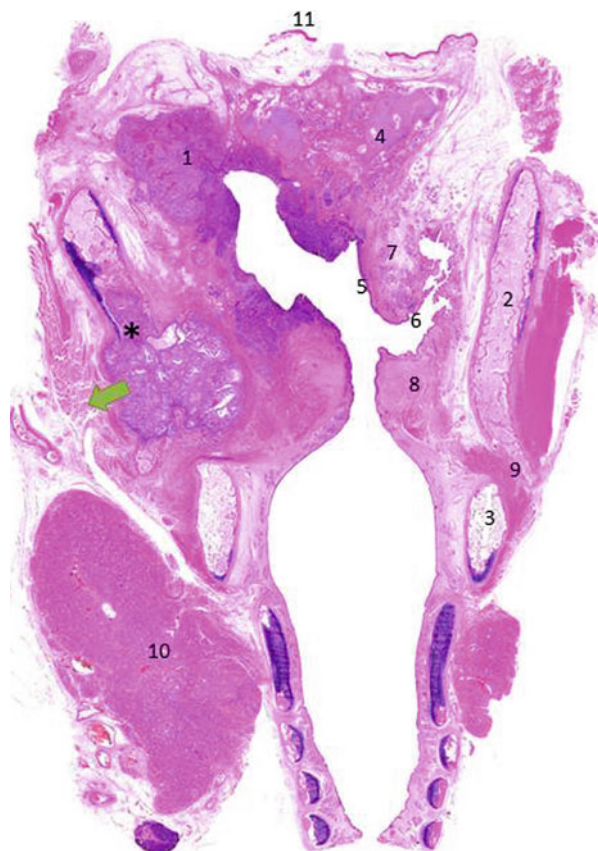
threefolds, the anatomical confluence of the aryepiglottic, glossoepiglottic, and pharyngoepiglottic folds.

Carcinoma of the apex of the arytenoid may extend to the medial wall of the piriform sinus, to the retroarytenoid area, and less commonly to the ventricular band and the aryepiglottic fold.

12.3 Glottic Carcinoma

Glottic carcinoma (Fig. 12.14) originates in most cases from the anterior portion of the vocal cord. In the initial phases, the neoplasia involves the free margin of the vocal cord which, due to the low supply of lymphatic vessels, is associated in this phase with a low risk of lymph nodal metastases. Afterwards, the tumour infiltrates

Fig. 12.9 Ventrolateral supraglottic carcinoma (coronal macrosection). *1* Advanced spread of ventrolateral supraglottic carcinoma with invasion of superior paraglottic space and lateral lamina of thyroid cartilage (*), *2* thyroid cartilage, *3* cricoid cartilage, *4* epiglottis, *5* ventricular fold, *6* Morgagni's ventricle, *7* superior paraglottic space, *8* vocal cord, *9* cricothyroid space, *10* thyroid, *11* glossoepiglottic valleculae



the vocal (thyroarytenoid) muscle and the inferior paraglottic space, and it may spread in a craniocaudal and lateral direction [15, 16].

12.3.1 Glottic Carcinomas Limited to the Glottic Site

In the initial phases, the carcinoma of the medio-anterior third of the vocal cord is characterised by an infiltration limited to the mucosa of the vocal ligament, to Reinke's space, and to the muscular fibres of the underlying thyroarytenoid muscle [17, 18]. Less frequently the glottic neoplasia arises in the posterior third of the cord (Fig. 12.15), with early involvement of the arytenoid cartilage, its vocal process, and sometimes the cricoarytenoid articulation. There are often peritumoural areas of dysplasia and carcinoma in situ. The initial glottic tumour of the anterior commissural area (Fig. 12.16) prematurely involves the vocal cords bilaterally and presents a high risk of infiltration of the dihedral angle of the thyroid cartilage (Fig. 12.17), even in the case of initial neoplasias with small dimensions (Fig. 12.18). Afterwards, the neoplasia infiltrates the thyroarytenoid muscle in depth and tends to involve the



Fig. 12.10 Lateral supraglottic carcinoma (axial macrosection). 1 Superficial spread of lateral supraglottic carcinoma with minimal invasion of superior paraglottic space through quadrangular membrane, 2 thyroid cartilage, 3 arytenoid cartilage, 4 interarytenoid muscle, 5 ventricular fold, 6 Morgagni's ventricle, 7 superior paraglottic space



Fig. 12.11 Ventricular supraglottic carcinoma (ventriculosaccular carcinoma) (axial macrosection). 1 Superficial spread of ventricular carcinoma with minimal invasion of paraglottic space, 2 thyroid cartilage, 3 petiole of epiglottis, 4 ventricular fold, 5 Morgagni's ventricle, 6 superior paraglottic space, 7 aryepiglottic fold, 8 preepiglottic space



Fig. 12.12 Marginal supraglottic carcinoma (axial macrosection). 1 Exophytic carcinoma of free margin of epiglottis and aryepiglottic fold, 2 epiglottis, 3 median glossoepiglottic fold, 4 glossoepiglottic vallecula, 5 retrolingual region, 6 laryngeal surface of epiglottis

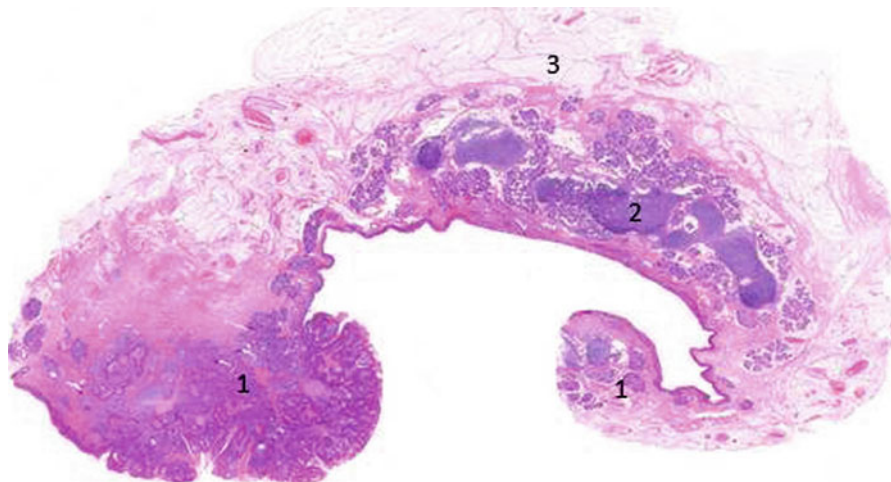
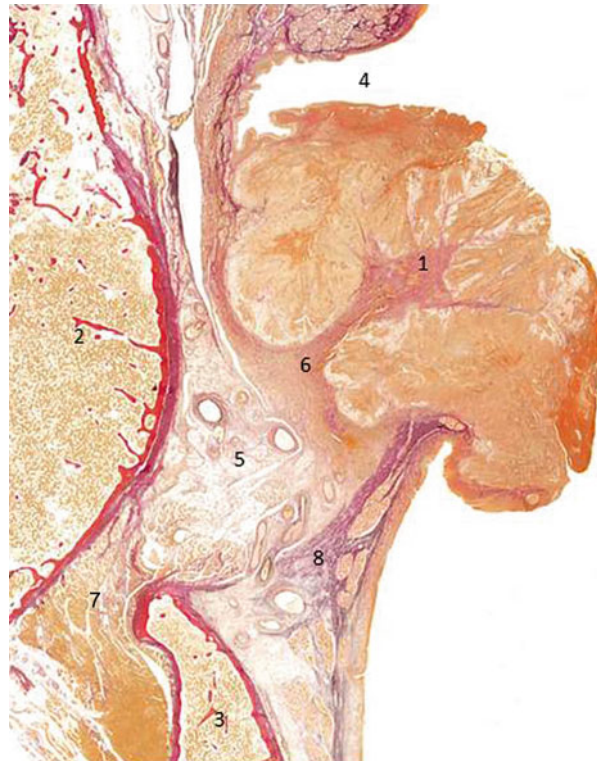


Fig. 12.13 Marginal supraglottic carcinoma (axial macrosection). 1 Exophytic carcinoma of aryepiglottic fold, 2 epiglottis, 3 preepiglottic space

inferior paraglottic space (Fig. 12.19), often with an ample exophytic intraluminal component. In over 85 % of cases, only one vocal cord is involved with extension to the anterior commissure [19] in about 10 % of cases and to the contralateral cord in less than 5 % of cases.

Fig. 12.14 Glottic carcinoma (coronal macrosection). 1 Local spread of glottic carcinoma arising from the free edge of vocal cord, invasion of cricothyroid muscle, 2 thyroid cartilage, 3 cricoid cartilage, 4 Morgagni's ventricle, 5 inferior paraglottic space, 6 vocal cord, 7 cricothyroid space, 8 elastic cone



12.3.2 Glottic Carcinoma with Subglottic Extension

The neoplasia presents a caudal diffusion to the vocal ligament and to the inferior face of the vocal cord towards the anterolateral subglottic region (Fig. 12.20). In its initial phases, the neoplasia spreads within the inferior paraglottic space which at this level is bounded anterolaterally by the perichondrium of the thyroid cartilage and inferomedially by the conus elasticus. In this phase of its growth, the tumour is pushed downwards and tends to infiltrate the subglottic site [20]. Afterwards, the neoplasia reaches the cricothyroid space with possible infiltration of the cricothyroid membrane and extralaryngeal extension. In most cases, the extension to the cricothyroid space coincides with the infiltration of the inferior margin of the lateral lamina of the thyroid cartilage. In about half the cases, the neoplasia extends to the anterior commissure [21] and to the subglottic region under the anterior commissure [22], an event which is accompanied by a high percentage of infiltration of the cartilage and of the cricothyroid membrane with possible anterior extralaryngeal extension (Fig. 12.21).

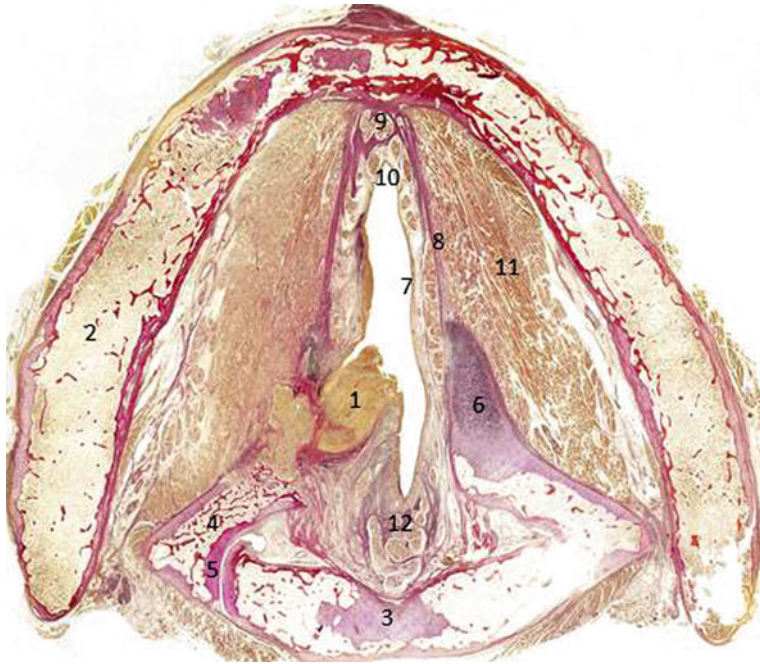


Fig. 12.15 Glottic carcinoma (axial macrosection). 1 Glottic carcinoma arising from the free edge of posterior third of vocal cord, 2 thyroid cartilage, 3 cricoid cartilage, 4 arytenoid cartilage, 5 cricoarytenoid joint, 6 vocal process of arytenoid cartilage, 7 vocal cord, 8 vocal ligament, 9 tendon of anterior commissure, 10 anterior commissure, 11 thyroarytenoid muscle, 12 posterior commissure



Fig. 12.16 Glottic carcinoma (axial macrosection). 1 Anterior commissure carcinoma, 2 cricoid cartilage, 3 arytenoid cartilage, 4 cricoarytenoid joint, 5 vocal process of arytenoid cartilage, 6 vocal cord, 7 cricothyroid space

Fig. 12.17 Primary anterior commissure carcinoma (coronal macrosection). *1* Advanced spread of anterior commissure glottic carcinoma, *2* thyroid cartilage, *3* anterior commissure tendon, *4* epiglottis, *5* superior paraglottic space, *6* ventricular fold, *7* quadrangular membrane, *8* conus elasticus, *9* preepiglottic space



12.3.3 Glottic Carcinoma with Supraglottic Extension

These are subdivided into neoplasias with limited or prevalent vertical extension, depending on the manner of growth.

Glottic carcinomas with limited vertical extension to the supraglottis are characterised by a prevalently mucosal spread along the tunica propria with minimum infiltration of the paraglottic space. They generally extend to the wall of Morgagni's ventricle and to its saccule and less frequently to the ventricular band along the arytenoid region and to the epiglottis along its pedicle. The anterior commissure [23] is involved in a low percentage of cases, sometimes with superficial spread to the contralateral vocal cord.

Glottic carcinomas with prevalent vertical extension to the supraglottis present a cranial diffusion along two preferential routes consisting of the anterior commissure and the paraglottic space. In the first case, the neoplasia spreads along the anterior commissural region [24] with infiltration of the thyroepiglottic ligament and of the caudal part of the preepiglottic and superior paraglottic



Fig. 12.18 Primary anterior commissure carcinoma (axial macrosection). 1 Anterior commissure carcinoma with thyroid cartilage infiltration and extralaryngeal tumour diffusion (*), 2 thyroid cartilage, 3 cricoid cartilage, 4 arytenoid cartilage, 5 cricoarytenoid joint, 6 vocal process of arytenoid, 7 vocal cord, 8 cricothyroid space

spaces [25]. In most cases, the neoplasias that originate from the medio-anterior third of the vocal cord rapidly reach the tunica propria of the ventricle and the inferior paraglottic space which at this level is largely occupied by the fibres of the vocal muscle. In these cases, the neoplastic growth (Fig. 12.22) is deviated upwards by the lateral lamina of the thyroid cartilage with infiltration of the superior paraglottic space (Fig. 12.23) [26], sometimes without involving the mucosal plane of the ventricular band. The role of Morgagni's ventricle in the cranial spread of the tumour may be decisive in some cases, especially in neoplasias originating from the superior edge of the vocal cord which at an early stage involve the floor of the ventricle and its aditus (Fig. 12.24). The endoventricular growth of the neoplasia generally involves the destruction of the ventricular walls and its spread into the superior paraglottic space. In some cases, the neoplasia enclosing the initial portion of the ventricle may produce a secondary laryngocele through a valve-type mechanism.

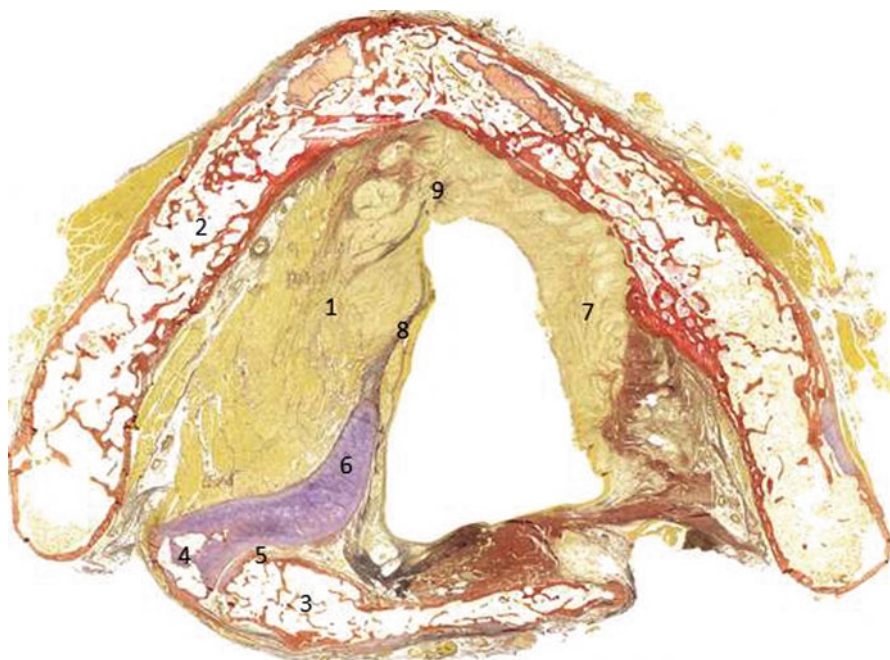


Fig. 12.19 Glottic carcinoma (axial macrosection). 1 Advanced glottic carcinoma, 2 thyroid cartilage, 3 cricoid cartilage, 4 arytenoid cartilage, 5 cricoarytenoid joint, 6 vocal process of arytenoid cartilage, 7 vocal cord, 8 vocal ligament, 9 anterior commissure

12.3.4 Transglottic Carcinoma

These are advanced forms of glottic neoplasias which extend in a craniocaudal direction along the axis of Morgagni's ventricle (Figs. 12.25 and 12.26). This term is often used to indicate voluminous tumours for which the seat of origin cannot be established with certainty or glottic neoplasias with supraglottic extension.

12.4 Subglottic Carcinoma

The primitive tumour of the subglottis [27] is a neoplasm in search of identity. In fact, if we consider neoplasias of the inferior face of the vocal cord as glottic tumours and exclude glottic neoplasias with a subglottic diffusion, primitive subglottic tumours [28] are extremely rare (Fig. 12.27) and account for less than 1 % of the most credible case histories [29]. In general, they present a growth and metastasisation pattern similar to tracheal tumours. They are characterised by early infiltration of the cricoid cartilage and of the cricothyroid membrane with possible extralaryngeal extension [30].

Fig. 12.20 Glottic carcinoma with subglottic spread (coronal macrosection). 1 Glottic carcinoma with spread to hypoglottis, 2 thyroid cartilage, 3 cricoid cartilage, 4 paraglottic space, 5 ventricular fold, 6 laryngocele, 7 vocal cord, 8 subglottis, 9 cricothyroid space, 10 epiglottis



12.5 External Laryngeal Carcinomas

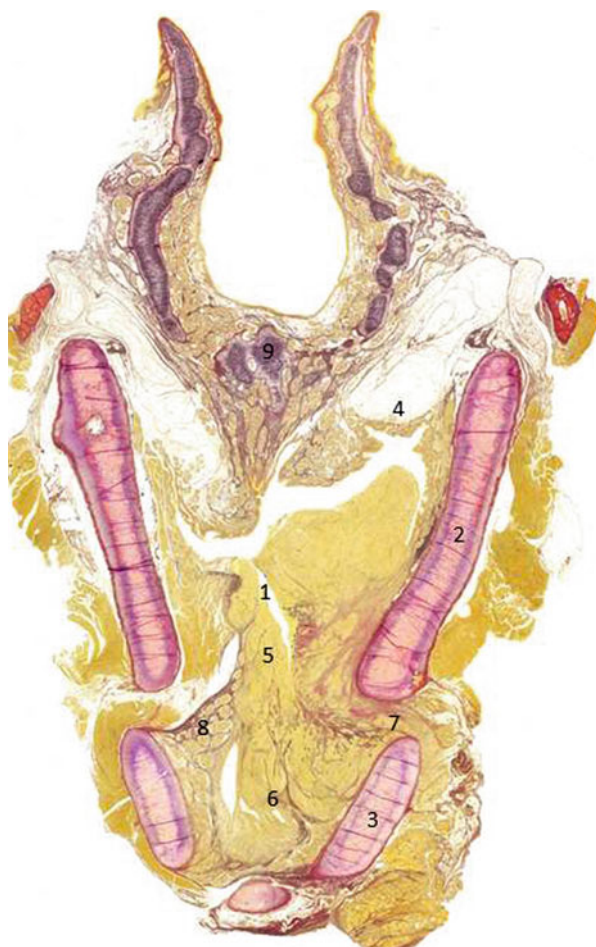
The definition of external laryngeal carcinomas includes neoplasias of the hypopharynx and of the inferior regions of the oropharynx (glossoepiglottic valleculae, retrolingual area, caudal lateral oropharyngeal walls including the region of the threefolds) which in their growth involve the different laryngeal regions and whose surgical treatment therefore includes laryngectomy.

12.5.1 Hypopharynx Carcinoma

The hypopharynx is divided into three subsites: piriform sinuses, posterior wall of the hypopharynx, and retrocricoid region.

Carcinoma of the piriform sinus (Figs. 12.28 and 12.29) is the most frequent hypopharyngeal neoplasia. In an early stage of its development, it involves the

Fig. 12.21 Glottic carcinoma with subglottic spread (coronal macrosection). 1 Glottic carcinoma with prevalent spread to anterior subglottis, 2 thyroid cartilage, 3 cricoid cartilage, 4 superior paraglottic space, 5 anterior commissure, 6 hypoglottis, 7 cricothyroid space, 8 conus elasticus, 9 epiglottis



laryngeal structures along three preferential routes: (a) anterolaterally first to the paraglottic space and then to the hyothyroepiglottic cavity; (b) spreading in a dorso-caudal direction towards the cricothyroid joint, the rear wall of the larynx, and the aditus of the oesophagus; and finally (c) spreading to the lateral vestibular walls. In the case of neoplasias of the piriform sinus, the presence of fixity of the vocal cord indicates infiltration of the cricoarytenoid complex, of the interarytenoid muscles, and, less frequently, of the recurrent nerve.

From the retrocricoid area (Fig. 12.30), the neoplasias spread craniocaudally towards the paraglottic spaces and the aditus of the oesophagus. From the rear wall of the hypopharynx, the neoplastic growth involves first the lateral wall of the piriform sinus, the lateral lamina of the thyroid cartilage, and the submucosal compartments of the larynx.



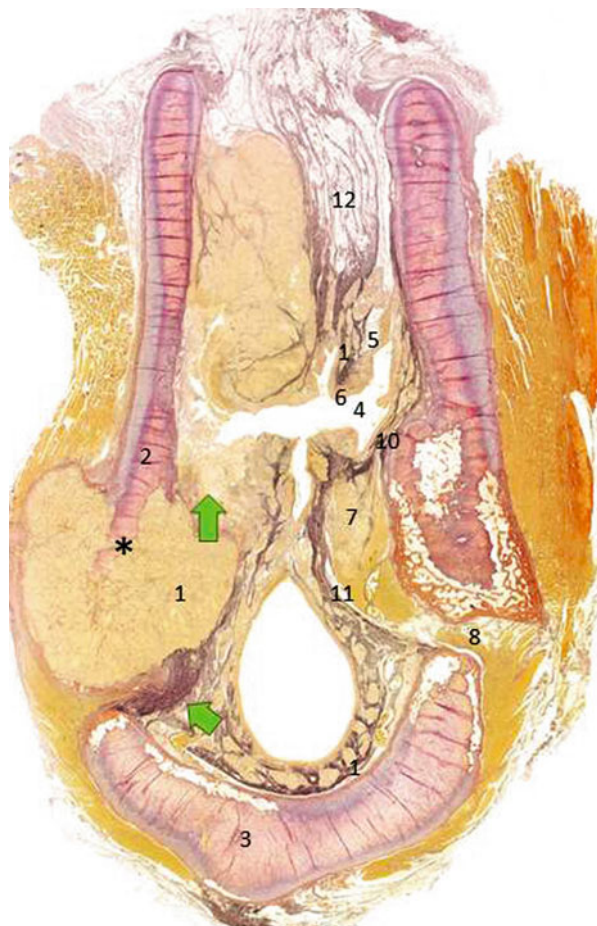
Fig. 12.22 Glottic carcinoma with supraglottic spread (coronal macrosection). 1 Advanced glottic carcinoma involving the superior paraglottic space, the ventricular fold (so-called transglottic carcinoma), and Morgagni's ventricle, 2 thyroid cartilage, 3 cricoid cartilage, 4 epiglottis, 5 Morgagni's ventricle, 6 superior paraglottic space, 7 ventricular fold, 8 vocal cord, 9 cricothyroid space, 10 quadrangular membrane, 11 ventricular ligament, 12 conus elasticus, 13 preepiglottic space, 14 hyoid bone, 15 hyoepiglottic ligament

12.5.2 Glossoepiglottic Region Carcinoma

The glossoepiglottic region includes (a) the glossoepiglottic valleculae, (b) the tongue base, and (c) the region of the threefolds.

The neoplasias of the vallecular region (Fig. 12.31) are limited in the first stages of their development by the hyoepiglottic membrane which favours their extension anteriorly towards the tongue base and posteriorly towards the lingual side of the epiglottis. Then, infiltrating the membrane, the neoplasia infiltrates the preepiglottic space, involving the laryngeal spaces and possibly destroying the epiglottis.

Fig. 12.23 Glottic carcinoma with supraglottic spread (coronal macrosection). 1 Advanced glottic carcinoma involving the superior paraglottic space, the ventricular fold (so-called transglottic carcinoma), and Morgagni's ventricle; invasion of cricothyroid space and thyroid cartilage, 2 thyroid cartilage (*), 3 cricoid cartilage, 4 Morgagni's ventricle, 5 superior paraglottic space, 6 ventricular fold, 7 vocal cord, 8 cricothyroid space, 9 quadrangular membrane, 10 ventricular ligament, 11 conus elasticus, 12 preepiglottic space



Carcinomas of the tongue base (Fig. 12.32) infiltrate the intrinsic and extrinsic muscles of the tongue at an early stage and then extend to the muscle of the buccal pelvis. Infiltration of the genioglossus muscle, inserted in fan formation between the mental spine of the mandible, the body of the hyoid bone, and the epiglottis, involves early neoplastic spreading to the body of the tongue and the floor of the mouth. Lastly, the absence of anatomical boundaries leads to constant involvement of the vallecular region with oropharyngeal–laryngeal extension. In both cases, the neoplasias of the glossoepiglottic region are characterised by early lymph nodal metastases in over 50 % of cases at the time of diagnosis, often bilateral.

Carcinomas of the region of the threefolds tend to extend to the adjoining regions of the larynx and the pharynx (Fig. 12.33).



Fig. 12.24 Glottic carcinoma with supraglottic spread (coronal macrosection). 1 Advanced glottic carcinoma involving the superior paraglottic space with mucosal integrity of ventricular fold; invasion of cricothyroid space, thyroid cartilage (*), and extralaryngeal extension, 2 thyroid cartilage, 3 cricoid cartilage, 4 Morgagni's ventricle, 5 superior paraglottic space, 6 ventricular fold, 7 vocal cord, 8 cricothyroid space, 9 quadrangular membrane, 10 ventricular ligament, 11 conus elasticus, 12 epiglottis, 13 preepiglottic space



Fig. 12.25 Transglottic carcinoma (coronal macrosection). *1* Advanced glottic carcinoma involving the superior paraglottic space, the ventricular fold, and Morgagni's ventricle; invasion of cricothyroid space and thyroid cartilage (*), *2* thyroid cartilage, *3* cricoid cartilage, *4* Morgagni's ventricle, *5* superior paraglottic space, *6* ventricular fold, *7* vocal cord, *8* cricothyroid space, *9* preepiglottic space, *10* epiglottis, *11* lateral extralaryngeal diffusion

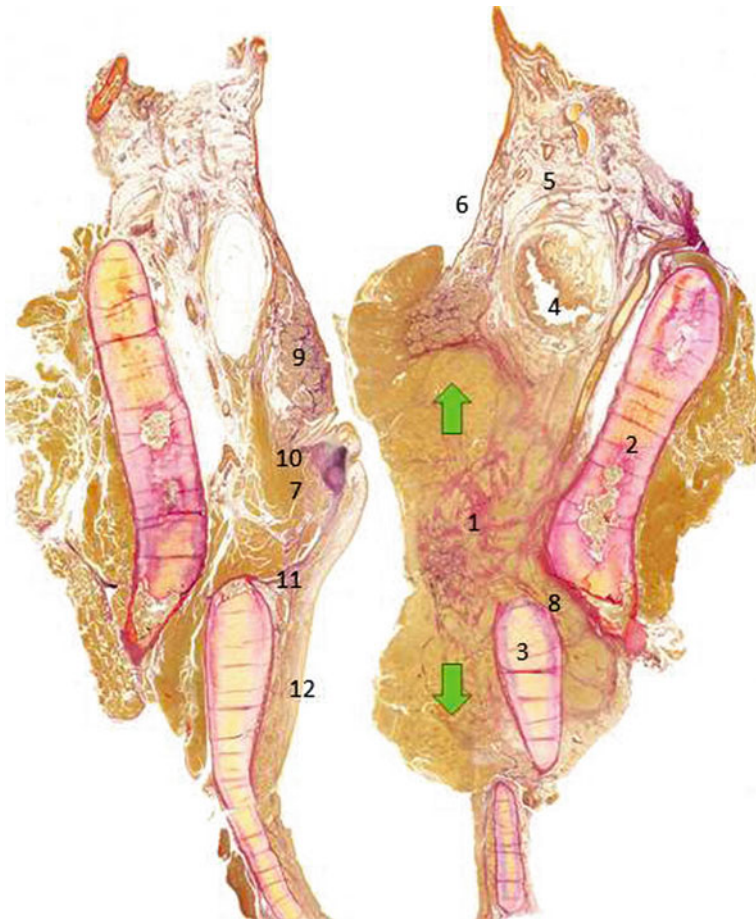


Fig. 12.26 Transglottic carcinoma (coronal macrosection). *1* Advanced glottic carcinoma involving glottis, superior paraglottic space, ventricular fold, Morgagni's ventricle, and hypoglottis, *2* thyroid cartilage, *3* cricoid cartilage, *4* Morgagni's ventricle, *5* superior paraglottic space, *6* ventricular fold, *7* vocal cord, *8* cricothyroid space, *9* quadrangular membrane, *10* ventricular ligament, *11* conus elasticus, *12* subglottis

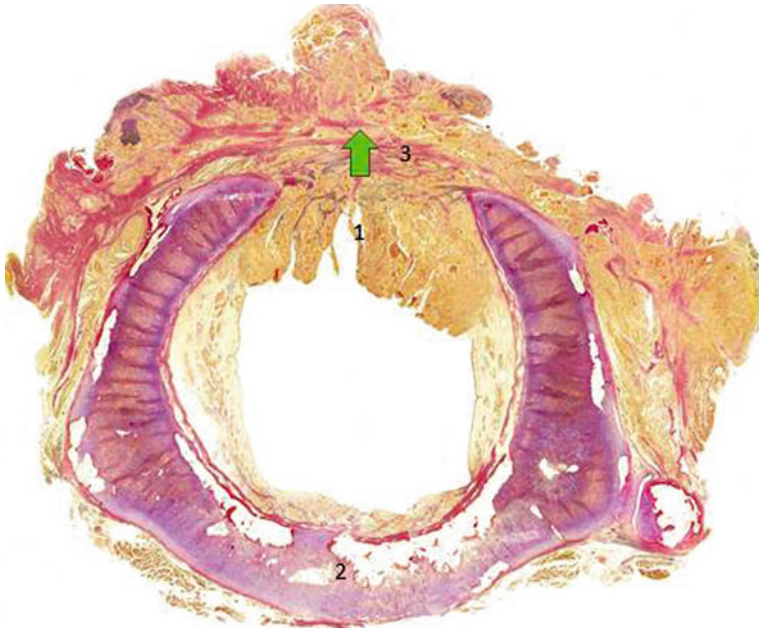


Fig. 12.27 Subglottic carcinoma (axial macrosection). 1 Anterior subglottic carcinoma, 2 cricoid cartilage, 3 cricothyroid membrane

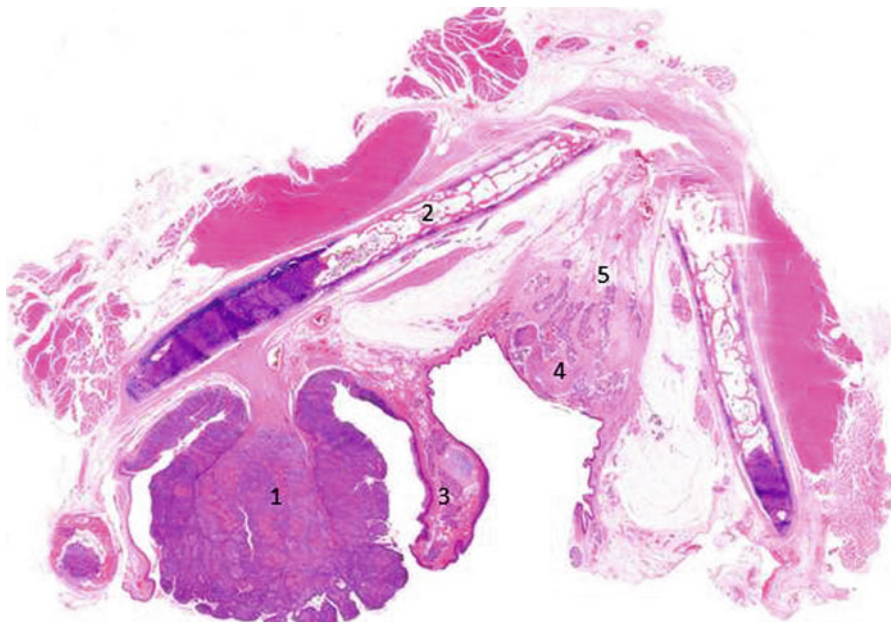


Fig. 12.28 External laryngeal carcinoma (axial macrosection). 1 Exophytic carcinoma of the piriform sinus, 2 thyroid cartilage, 3 aryepiglottic fold, 4 epiglottis, 5 preepiglottic space

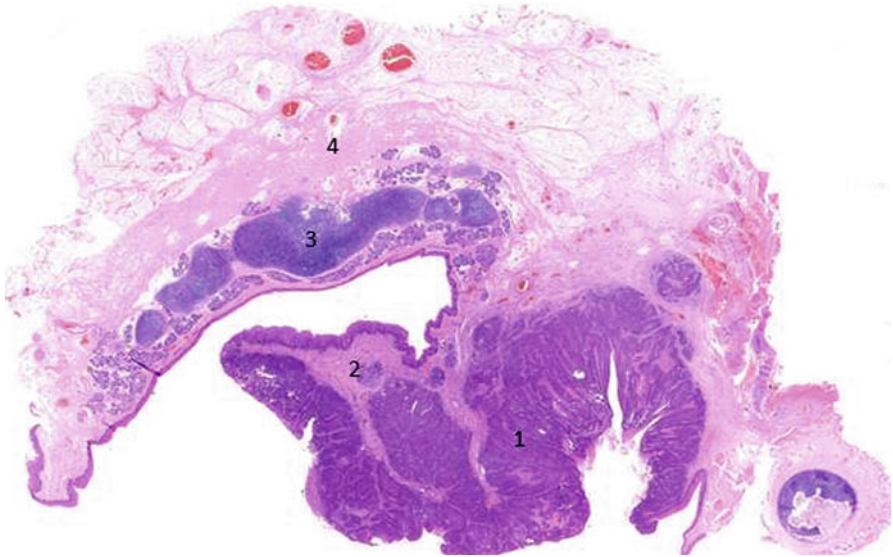


Fig. 12.29 External laryngeal carcinoma (axial macrosection). 1 Advanced carcinoma of the piriform sinus, 2 aryepiglottic fold, 3 epiglottis, 4 preepiglottic space

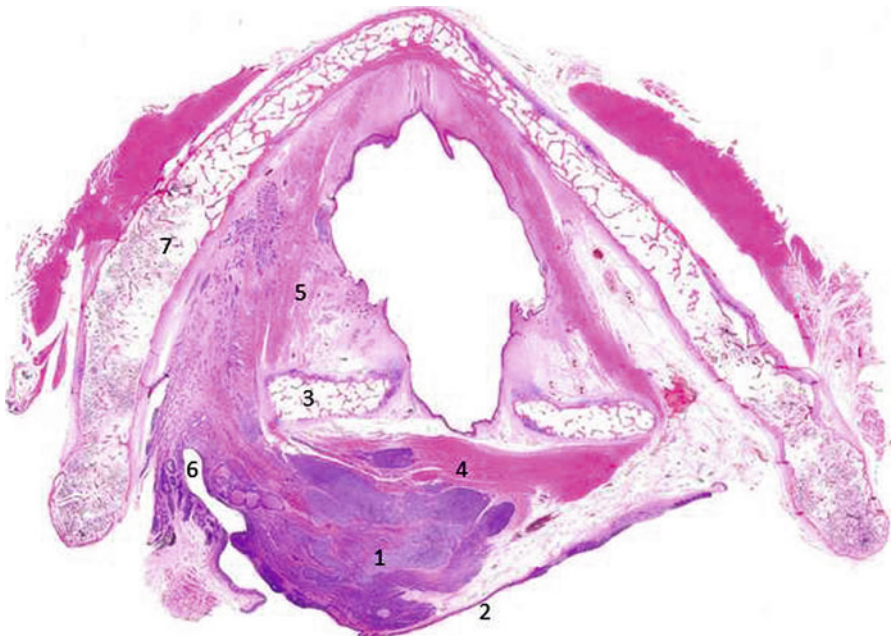


Fig. 12.30 External laryngeal carcinoma (axial macrosection). 1 Hypopharynx carcinoma (post-cricoid area), 2 postcricoid area, 3 arytenoid cartilage, 4 interarytenoid muscle, 5 inferior paraglottic space, 6 piriform sinus, 7 thyroid cartilage

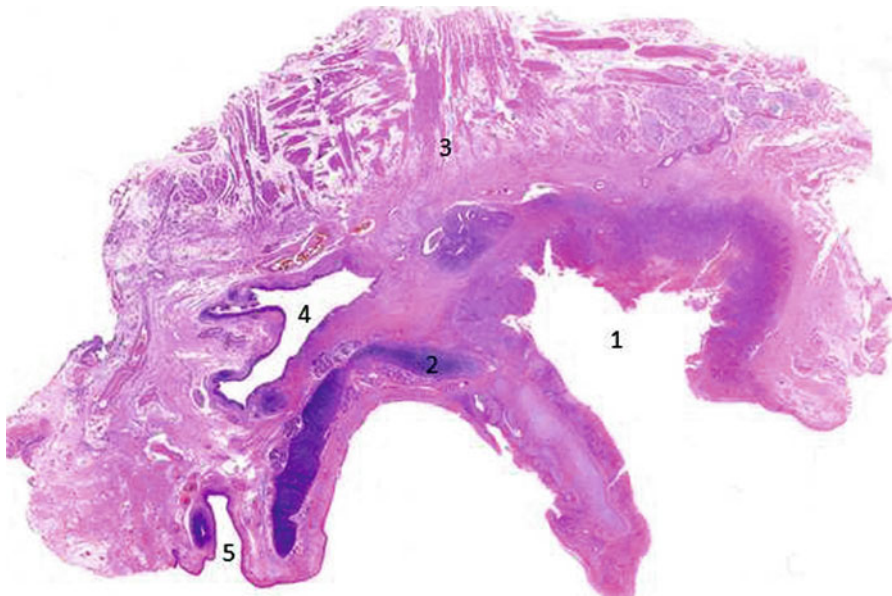


Fig. 12.31 External laryngeal carcinoma (axial macrosection). 1 Carcinoma of the glossoepiglottic vallecula (lateral), 2 epiglottis, 3 retrolingual region, 4 glossoepiglottic vallecula, 5 piriform sinus

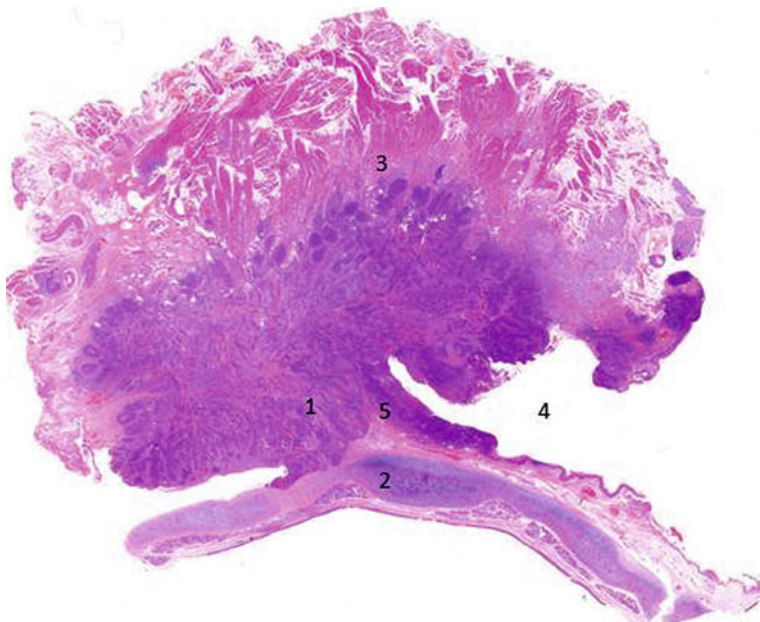


Fig. 12.32 External laryngeal carcinoma (axial macrosection). 1 Carcinoma of the glossoepiglottic vallecula (medial), 2 epiglottis, 3 retrolingual region, 4 glossoepiglottic vallecula, 5 glossoepiglottic medial fold

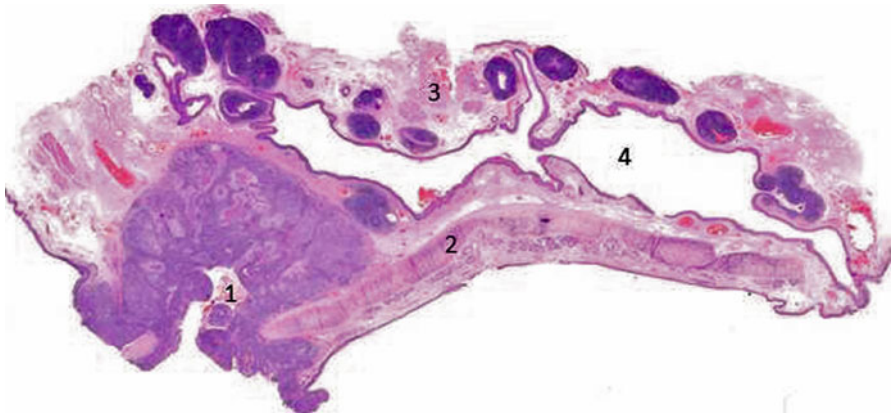


Fig. 12.33 External laryngeal carcinoma (axial macrosection). 1 Carcinoma of the “threefolds” (aryepiglottic, glossoepiglottic, and pharyngoepiglottic folds), 2 epiglottis, 3 retrolingual region, 4 glossoepiglottic vallecula

Take-Home Messages

- In laryngeal carcinoma, the proliferating cells grow in number forming complex neoplastic agglomerates but still with a certain structural organisation. The development of this tissue is apparently chaotic only because of its interaction with healthy laryngeal structures, which condition and direct its growth with “lines of strength” and “lines of weakness”.

References

1. Carlon G (1990) Il carcinoma della laringe. Dalla patologia alla clinica. Piccin, Padova
2. Carlon G, Della Libera D, Dei Tos AP (1990) A whole organ sectioning method for histologic examination of laryngeal and hypopharyngeal specimens. In: Sacristan T (ed) Otolaryngology, head and neck surgery. Kugler & Ghedini, Amsterdam, pp 2367–2371
3. Beitler JJ, Mahadevia PS, Silver CE, Wadler S, Rubin JS, Bello JA, Mitnik RJ, Vikram B (1994) New barriers to ventricular invasion in paraglottic laryngeal cancer. *Cancer* 15(73):2648–2652
4. Bryce DP, van Nostrand AW, Brodarec I (1983) Growth and spread of laryngeal cancer. *Adv Otorhinolaryngol* 29:9–23
5. Lam KH (1983) Extralaryngeal spread of cancer of the larynx: a study with whole-organ sections. *Head Neck Surg* 5:410–424
6. Lam KH, Wong J (1983) The preepiglottic and paraglottic spaces in relation to spread of carcinoma of the larynx. *Am J Otolaryngol* 4:81–91
7. Cleri LH (1944) The preepiglottic space – its relation to carcinoma of the epiglottis. *Trans Amer Laryng Rhinol Otol Soc* 11:127–131
8. Dayal VS, Bahri H, Stone PC (1972) Pre-epiglottic space. An anatomic study. *Arch Otolaryngol* 95:130–133
9. Kirchner JA, Som ML (1971) Clinical and histological observations on supraglottic cancer. *Ann Otol (St Louis)* 80:638–646

10. McDonald TJ, Desanto LW, Weiland LH (1976) Supraglottic larynx and its pathology as studied by whole laryngeal sections. *Laryngoscope (St Louis)* 86:635–648
11. Meyer-Breiting E (1984) Squamous cell carcinomas of the anterior wall of the larynx. In: Wigand ME, Steiner W, Steli PM (eds) *Functional partial laryngectomy conservation surgery for carcinoma of the larynx*. Springer, Berlin, pp 140–143
12. Micheau C, Luboinski B, Lanchi P, Cachin Y (1978) Relationship between laryngocele and laryngeal carcinomas. *Laryngoscope (St Louis)* 88:680–688
13. Micheau C, Leonardelli GB, Gérard-Marchant R, Cachin Y (1973) Modalités d’envahissement des tumeurs du vestibule larynge: aspects histopathologiques et statistiques. *Nuovo Arch Ital Otol* 1:279–291
14. Sessions DG, Ogura JH (1976) Classification of laryngeal cancer. In: Alberti PW, Bryce DP (eds) *Workshops from the centennial conference on laryngeal cancer*. Appleton-Century-Crofts, New York, pp 83–89
15. Olofsson J (1976) Growth and spread of laryngeal carcinoma. In: Alberti WP, Bryce DP (eds) *Workshops from the centennial conference on laryngeal cancer*. Appleton-Century-Crofts, New York, pp 40–53
16. Kirchner JA (1977) Two hundred laryngeal cancers: patterns of growth and spread as seen in serial sections. *Laryngoscope (St Louis)* 87:474–482
17. Broyles EN (1943) The anterior commissure tendon. *Ann Otol Rhinol Laryngol* 52:342–345
18. Kleinsasser O, Glanz H (1982) Microcarcinoma and microinvasive carcinoma of the vocal cords. *Clin Oncol* 1:479–487
19. Andrea M (1981) Vasculature of the anterior commissure. *Ann Otol Rhinol Laryngol* 90:18–20
20. Freelançi AP, Van Nostrand P (1976) The applied anatomy of the anterior commissure and subglottis. In: Alberti PW, Bryce DP (eds) *Workshops from the centennial conference on laryngeal cancer*. Appleton-Century-Crofts, New York
21. Bagatella F, Bignardi L (1981) Morphological study of the laryngeal anterior commissure with regard to the spread of cancer. *Acta Otolaryngol* 92:167–171
22. Kirchner JA, Fischer JJ (1976) Anterior commissure cancer – a clinical and laboratory study of 39 cases. In: Alberti PW, Bryce DP (eds) *Workshops from the centennial conference on laryngeal cancer*. Appleton-Century-Crofts, New York, pp 645–651
23. Barbosa MM, Araujo VJ, Boasquevisque E, Carvalho R, Romano S, Lima RA, Dias FL, Salviano SK (2005) Anterior vocal commissure invasion in laryngeal carcinoma diagnosis. *Laryngoscope* 115(4):724–730
24. Andrea M, Guerrier Y (1981) The anterior commissure of the larynx. *Clin Otolaryngol* 6:259–264
25. Bagatella F, Bignardi L (1983) Behavior of cancer at the anterior commissure of the larynx. *Laryngoscope (St Louis)* 93:353–356
26. Della Libera D, Bittesini L, Falconieri G (1997) Anterior commissure involvement in glottic laryngeal carcinoma. In: *Advances in laryngology in Europe. Proceedings of the first scientific conference of the European Laryngological Society*. Elsevier Science B.V, pp 355–357
27. Harrison DF (1971) The pathology and management of subglottic cancer. *Ann Otol* 80:6–12
28. Steli PM, Gregory I, Watt J (1980) Morphology of the human larynx. II. The subglottis. *Clin Otolaryngol Allied Sci* 5:389–395
29. Olofsson J (1976) Specific features of laryngeal carcinoma involving the anterior commissure and subglottic region. In: Alberti PW, Bryce DP (eds) *Workshops from the centennial conference on laryngeal cancer*. Appleton-Century-Crofts, New York, pp 626–644
30. Lund WS (1976) Classification of subglottic tumors and discussion of their growth and spread. In: Alberti WP, Bryce DP (eds) *Workshops from the centennial conference on laryngeal cancer*. Appleton-Century-Crofts, New York