# Unilateral laryngeal palsy

## A histopathological study

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Many types of laryngeal palsy have been described. The ultimate position of a unilateral cord palsy may be still debatable but most of the controversy has been settled. A unilateral paralysis of one recurrent laryngeal nerve causes a recognizable clinical picture. During rest, on the healthy side the vocal cord is abducted; however, on the paralysed side the vocal cord maintains a fixed paramedian position a few millimetres from the midline. On phonation the normal vocal cord moves to the midline and in some instances beyond it to compensate by meeting the paralysed cord. The only muscle that can still influence the paralysed cord is the cricothyroid muscle which is supplied by the superior laryngeal nerve. Contraction of this muscle tenses the paralysed vocal cord bringing it more towards the midline. During breathing the paralysed cord is usually seen in a more inferior position than the normal cord. These observations and most of the documentation about vocal cord palsy have been established by laryngologists examining the larynx by indirect and direct laryngoscopy.

A new source of information became available when Ardran *et al.* (1954) used radiological methods to study normal laryngeal movements and laryngeal palsies. According to Ardran, during quiet breathing the lumen of the larynx is wide open and very little movement takes place. The vocal cord is turned upwards over the laryngeal ventricle so that its free edge is in close proximity to the lower surface of the vestibular fold. The cavity of the ventricle is thereby obliterated or reduced to a small slit. In the case of unilateral vocal cord palsy the vocal cord on the affected side projects into the lumen almost to the midline. The laryngeal ventricle is wide open and the vestibular fold is prominent and nearer to the midline than on the normal side.

Another source of information is the histological examination of the serially sectioned larynx. Kirchner (1966) studied by serial section the changes in the laryngeal muscles resulting from vagal paralysis. In our study a patient suffering with advanced carcinoma of the upper oesophagus was found to have a total left recurrent laryngeal nerve palsy. At post-mortem the larynx was taken for serial sectioning.

Figure 1 is a section taken at mid-cord level. The paralysed vocal cord protrudes into the laryngeal lumen almost to the midline. The false vocal cord also projects into the lumen. The paralysed vocal cord has



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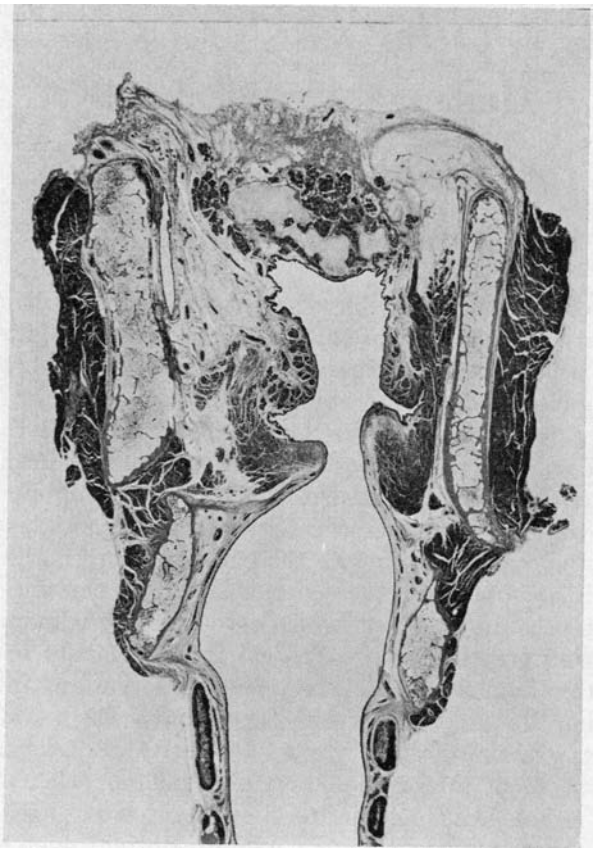


FIG. 1.

Section at mid-cord level. The palsied vocal cord projects towards the midline, opening out the ventricle. On the healthy side the vocal cord is turned up almost meeting the false vocal cord.

drooped downwards and inwards partially obstructing the laryngeal lumen and placing the free vocal cord margin at a more inferior level than the erect opposite uninvolved vocal cord. The conus elasticus which normally extends upwards and inwards from the cricoid as seen on the healthy side is inclined into an almost horizontal plane on the paralysed side. This collapse of the conus elasticus is due to atrophy and loss of tension in the muscle fibres of the thyroarytenoid muscle which is inserted into and helps suspend the vocal cord. As a result the free cord margin on the paralysed side has become thinner than on the healthy side.

On the paralysed side the floor of the ventricle has fallen downwards and inwards causing the ventricle to open and become wider than on the healthy side where the ventricle is demonstrated as a small slit.

Figure 2 shows distortion in the laryngeal vestibule and widening of



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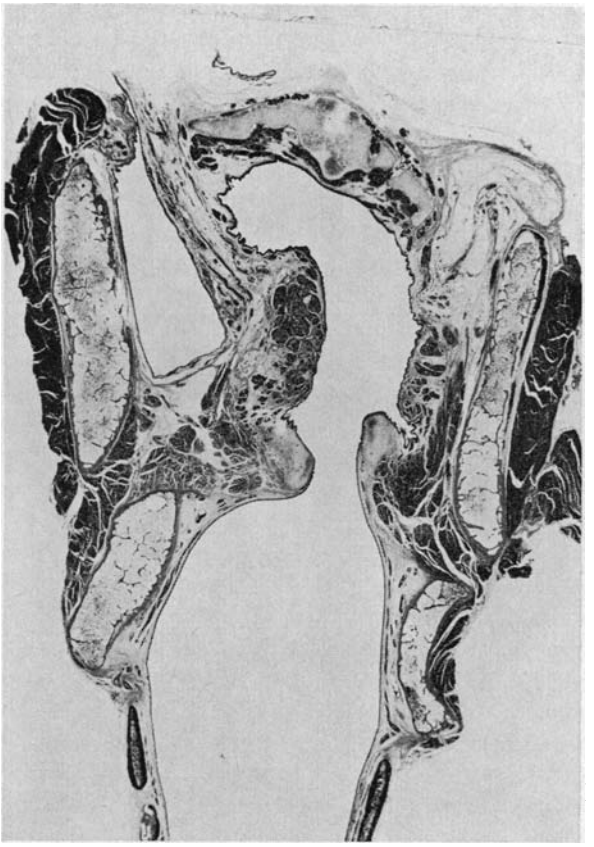


FIG. 2.

Section at level of vocal process. On the paralysed side the lateral laryngeal wall is seen to be dropping inwards; as a result, the laryngeal lumen is distorted and the pyriform sinus dilated.

the pyriform sinus. Superiorly the epiglottis is suspended by ligamentous attachments to the hyoid bone; however, inferiorly the aryepiglottic fold, lateral laryngeal wall, and false vocal cord rely mainly on muscle suspension. Paralysis of these muscles has resulted in the lateral laryngeal wall sagging inwards with the false and true vocal cords. The laryngeal lumen is thereby distorted; being constricted and displaced towards the normal side inferiorly, and then curving over towards the paralysed side more superiorly. There is compensatory widening of the lower portion of the pyriform sinus.

Figure 3 is a section through the cricoarytenoid joint. The paralysed arytenoid has become displaced towards the healthy side. This is due to the unopposed action of the healthy interarytenoid and posterior cricoarytenoid muscles. The slide shows the atrophy and loss of density in the



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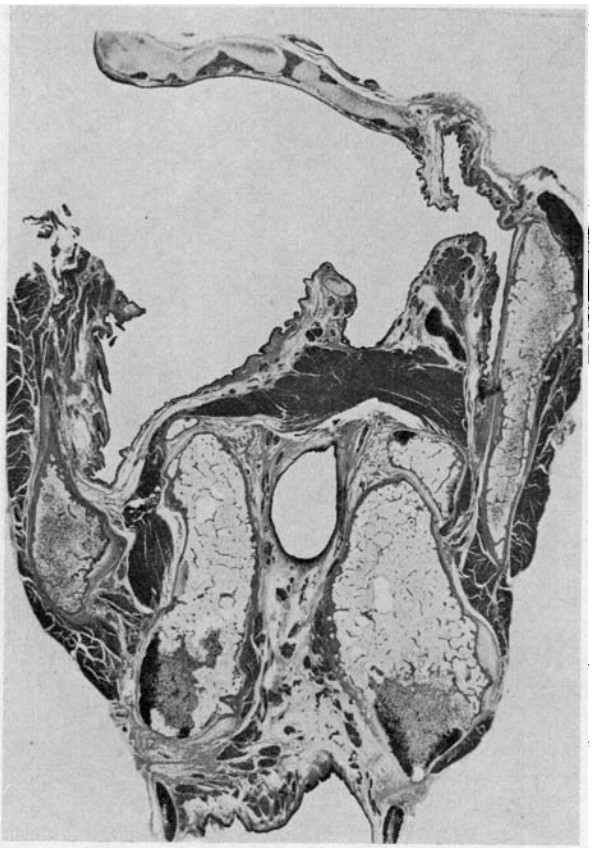


FIG. 3.
Section through the arytenoids. There is medial and forward displacement of the paralysed arytenoid. The dilatation of the pyriform sinus is also evident.

muscles surrounding the paralysed arytenoid as well as the forward and medial rotation of the arytenoid. The section passes through the middle of the cricoarytenoid joint on the normal side and completely behind the joint on the involved side. The displacement of the arytenoid has made the pyriform sinus wider and deeper.

## Discussion

The histopathological features of unilateral cord palsy are in keeping with the radiological findings. Ardran and Kemp (1966) in a radiological study of the normal larynx showed that during quiet breathing the laryngeal lumen is wide open. The vocal cords are turned upwards over the laryngeal ventricles so that the airway is not obstructed. In the histopathological study the healthy vocal cord was turned up; however, on the paralysed side the vocal cord was turned downwards and projected well



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into the lumen. This placed the vocal cord at a more inferior level and at the same time opened out the ventricle. The paramedian position of the vocal cord is due to the loss of the suspensory action of the muscles normally responsible for holding the vocal cord erect.

The radiological literature mentions the fullness which develops in the subglottic area on the paralysed side. Stam (1963) referred to this flattening of the subglottic angle as the paralytic curve. According to Landman (1970) the subglottic tissue may bulge to show a convex medial contour; however, in most instances the curve remains concave. This increase in tissue in the subglottic region is demonstrated in Figures 1 and 2 and is due to the downward and medial displacement of the conus elasticus and contiguous paraglottic tissues. The conus elasticus bulges inwards, bearing as it does the now inert weight of the paraglottic muscles and associated areolar tissue.

One of the most significant manoeuvres causing the paralysed vocal cord to move towards the midline is the downward, forward and medial rotation of the arytenoid on that side. Ardran and Kemp (1966) have described the normal arytenoid movements when the glottis is open and closed. When the glottis is opened the arytenoids are displaced upwards, backwards and outwards; the vocal processes are tilted upwards thus folding the posterior ends of the vocal cord into the laryngeal ventricle. When the glottis is closed the arytenoids move downwards, forwards and inwards; the bases of the arytenoids project over the border of the cricoid cartilage and the vocal processes are tilted downwards. The histopathological study shows that in the paralysed state the vocal cord and arytenoid tend to assume the position of the normal vocal cord when the glottis is closed. The paralysed vocal cord will also not be able to oppose other external forces acting upon it. Ardran and Kemp (1966) have mentioned the powerful action of the constrictor muscles in deglutition. The action of these muscles is to displace the arytenoid masses inwards and since the ability to reopen the glottis is palsied the vocal cord will adopt a downward and inward position.

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