The anterior cricoid split in puppies

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Summary

The acute and long-term effects of the anterior cricoid split on the subglottis of puppies intubated from 7 to 14 days are documented. The anterior cricoid split acutely increased the intralumenal cricoid surface area in puppies with intubation-induced airway injury. An intense inflammatory response with mucosal ulceration and granulation tissue is elicited by 14 days of intubation by using the canine model of induced subglottic stenosis developed by Supance et al. [19]. When animals intubated for 14 days underwent an anterior cricoid split on day 7, the airway appears essentially normal by day 14. The split cricoid cartilage maintained a 'U' configuration following the procedure. The region of deficient cartilage anteriorly is bridged by fibrous tissue with normal epithelium lining the lumenal surface. Splitting the cricoid cartilage anteriorly increased the intralumenal area and no long term complications resulted from the procedure. The mechanisms by which the anterior cricoid split expands the airway intralumenal area while maintaining airway support have been reviewed.

Introduction

The infant mortality rate (deaths of children under one year of age per 1000 live births) had declined steadily over the past 15 years. Advances in respiratory support of neonates have probably been the single most important factor for this decline. These advances have relied increasingly on mechanical respiratory support for

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immature infants with ventilatory failure. Prolonged intubation is generally well tolerated by neonates without significant morbidity or sequelae. However, potentially severe complications such as subglottic stenosis occasionally result from this form of treatment. Extubation is often difficult in these patients and a tracheostomy is often required for adequate ventilation.

In 1980, Cotton and Seid proposed the anterior cricoid split as an alternative to tracheotomy in the management of extubation problems in the premature child [3]. Several years later, Frankel et al. [7] described the use of the anterior cricoid split operation in infants with acquired subglottic stenosis. Holinger [11] recently reviewed his own experience as well as the reported cases utilizing the anterior cricoid split procedure. He concluded that a tracheotomy can be avoided in properly selected cases.

Several mechanisms have been proposed to explain the success of the anterior cricoid split. This study utilized a modification of a canine model of subglottic injury proposed by Supance [19]. The study elucidates the short- and long-term effects of the anterior cricoid split on an acutely injured canine larynx.

Methods

Twenty-four healthy, mongrel puppies 5–10 weeks of age were obtained from regional kennels and randomly assigned to 6 study groups. The average weight of the animals was 2.72 kg (range 1.6–4.5 kg). The average weight of the animals in each group was similar. The puppies were conditioned and housed at the University Medical Affiliates Animal Care Facility. Each animal was vaccinated against DHLP and PV (0.1 ml × 4 doses). The caloric intake and weight of each animal was closely monitored daily.

All of the animals were intubated under general anesthesia. Atropine (0.09 mg/kg) i.m. and Acepromazine (0.55 mg/kg) i.m. were used prior to the induction of general anesthesia with i.v. pentobarbital sodium (13.2 mg/kg). An uncuffed endotracheal tube (polyvinyl chloride) was placed after direct visualization of the endolarynx. The largest tube which permitted an air leak with a 20 cm positive pressure ventilation was chosen. The animals were then mechanically ventilated using oxygen and methoxyflurane.

Under sterile conditions, a vertical, midline neck incision was made exposing the upper 3–4 tracheal rings. The anesthesia was lightened to allow the animals to breathe spontaneously and the endotracheal tube was removed. This tube was replaced with a 4 cm segment of endotracheal tube placed endoscopically into the laryngotracheal complex with the superior aspect 5 mm above the vocal cords (Fig. 1). The tube was secured in place using a 3-0 prolene suture through the exposed trachea. The subcutaneous tissue and skin were closed. The animals were awakened with the endotracheal tube fixed in position in the laryngotracheal complex.

The puppies were randomized to one of 6 groups. Each group had 4 animals.
Group I - intubated 7 days
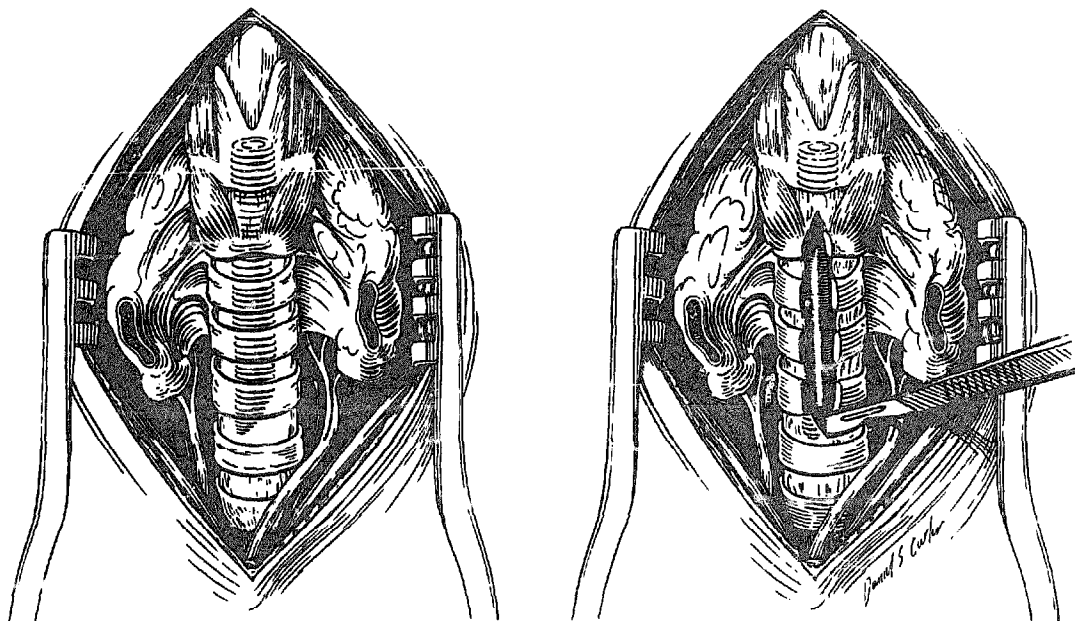Group II - intubated 14 days

Fig. 1. Artist sketch of the positioning of the endotracheal tube segment extending from 5 mm above the glottis into the trachea.

Fig. 2. Incision of the midline through the cricoid cartilage and upper tracheal rings.

Group III - intubated 7 days; anterior cricoid split on day 7

Group IV - intubated 14 days; anterior cricoid split on day 7; extubated and sacrificed day 14

Group V - intubated 14 days; anterior cricoid split on day 7; extubated day 14 and sacrificed day 31

Group VI - intubated 14 days; anterior cricoid split day 7; extubated day 14; sacrificed at 6 months of age.

Animals in Groups III, IV, V, VI had an anterior cricoid split performed. The anterior cricoid split was performed under local anesthesia, 2% lidocaine, after i.m. sedation with Innovar Vet (0.4 mg fentanyl/20.0 mg droperidol per ml), 1 ml per 6.8 kg. Under sterile conditions the cricoid cartilage and upper two tracheal rings were split in the midline (Fig. 2). The incision extended through mucosa exposing the indwelling segment of endotracheal tube. The neck skin was then loosely approximated for the closure. The animals in each group were extubated and sacrificed at a specific time. The laryngotracheal complexes were sectioned, photographed and submitted for histologic examination. The acute increase in intralumenal cricoid surface area resulting from performing the anterior cricoid split was measured in Group III animals. The dogs in Group III were sacrificed immediately after performing the anterior cricoid split. The laryngotracheal complex was harvested and the cricoid cartilage intralumenal area in the 'open' position was measured with a polar planimeter. The anterior extent of the airway lumen was defined by a line drawn between the posterior edges of the divided cricoid cartilage.

The cut ends of the cricoid cartilage were then re-approximated and the cricoid cartilage intralumenal area in its 'closed' position was measured. The area difference represented the change in intralumenal area created by the cricoid split.

Results

Group I–intubated 7 days

The animals in this group tolerated 7 days of intubation without difficulty. Coughing and occasional aspiration subsided in each of the animals after the initial 24–48 h of intubation.

Following 7 days of intubation, the subglottic mucosa grossly appeared erythematous and mildly edematous (Fig. 3). There were areas of superficial mucosal ulceration.

Microscopically, the specimens displayed submucosal inflammation and edema, acute mucositis with inflammatory changes in the minor salivary glands. Small areas of superficial epithelial ulceration were also present.

Group II—intubated 14 days

Whereas the initial 10–12 days of intubation were tolerated without difficulty, following the 12th day of intubation the oral intake dropped off considerably. One
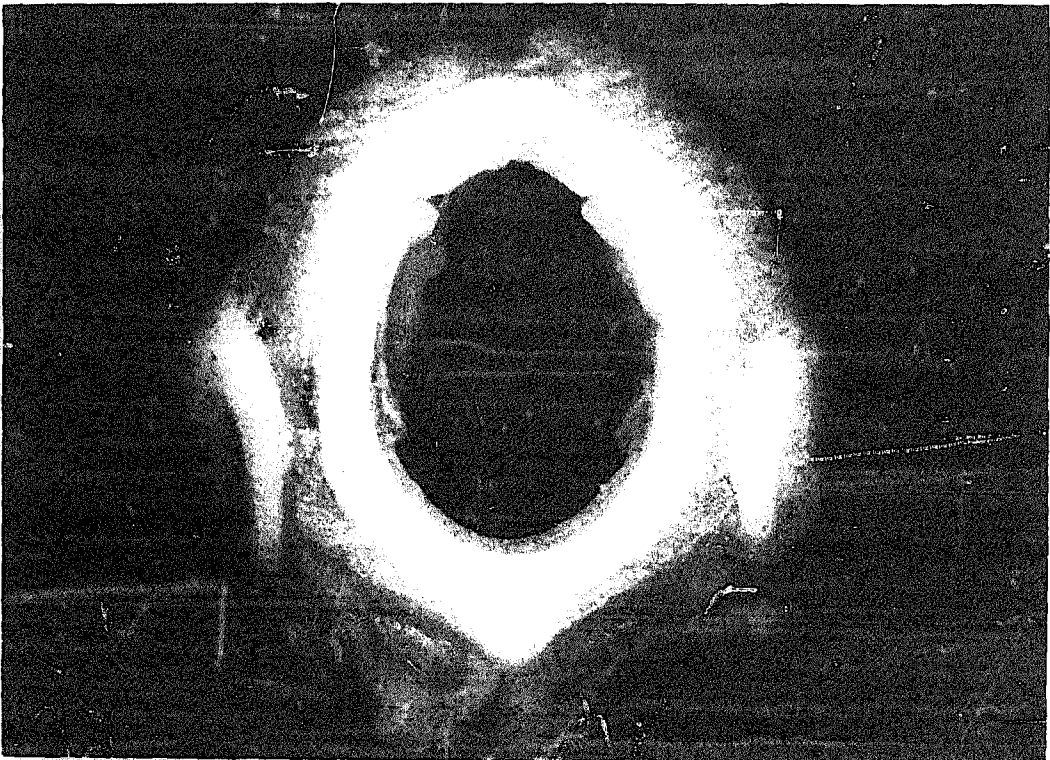

Fig. 3. Section through cricoid cartilage intubated for 7 days. Minimal mucosal edema (×3).

Fig. 4. Section through cricoid cartilage intubated for 14 days. Airway lumen narrowed by extensive granulation tissue (×3).

animal in this group was sacrificed on day 11 because of fulminant enteritis. The animals were extubated for approximately 1 h prior to being sacrificed. Marked inspiratory and expiratory stridor was present but the animals maintained adequate ventilation.

Grossly, the subglottic region in the animals intubated for 14 days was filled with pink, soft granulation tissue which narrowed the airway lumen (Fig. 4). Multiple areas of exposed cricoid cartilage were also observed.

Microscopically, a chronic inflammatory response characterized by a mono-nuclear cell infiltration was present. The granulation tissue displayed fibroblast proliferation and blood vessel ingrowth in a loose, edematous matrix. Perichondritis was evident in the areas of exposed cartilage.

Group III—-intubated 7 days. Anterior cricoid split day 7

The cricoid cartilage 'sprung open' 3.0–8.0 mm following the anterior cricoid split on the 7th day (Fig. 5). In one of the animals, the endotracheal tube was removed immediately before performing the anterior cricoid split but the cricoid opened in a similar fashion as those animals in which the endotracheal tube was present (Fig. 6). The animals were sacrificed and the intralumenal cricoid area was measured with a polar planimeter. A second measurement was taken after the cut ends of the cricoid were re-approximated (Fig. 7). The area with the cricoid cartilage
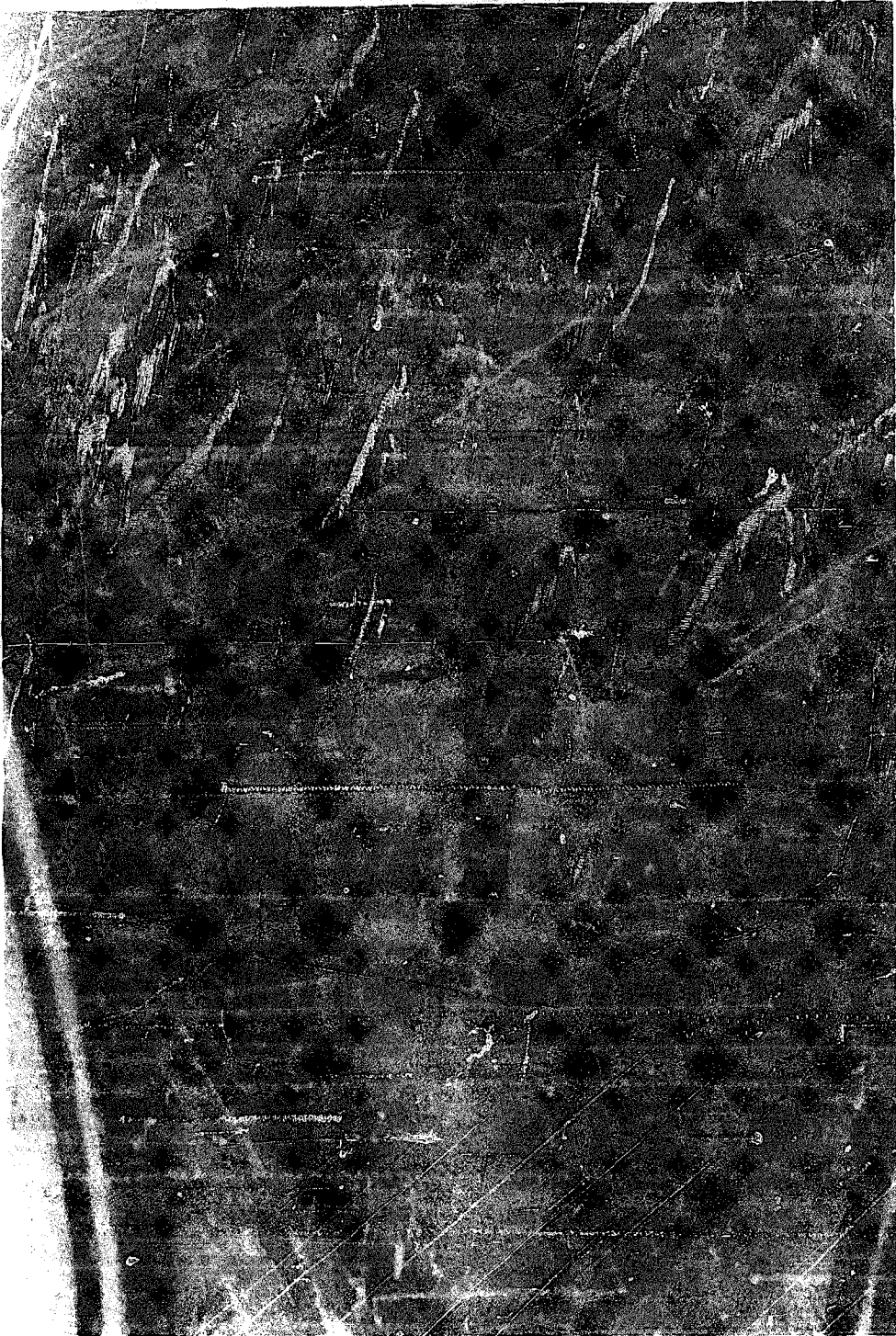
Fig. 5. Intra-operative photograph of a laryngotracheal complex intubated 7 days immediately after performing the anterior cricoid split. Endotracheal tube segment present in the airway lumen. Arrows point to the cricoid cartilage. Note how the cut ends of the cricoid and tracheal cartilages have 'sprung' open.

Fig. 6. Intra-operative photograph of a laryngotracheal complex intubated for 7 days. The endotracheal tube segment was removed prior to performing the anterior cricoid split.

re-approximated was subtracted from the post-split area. The percent change was then calculated. The cricoid intralumenal area increased in every case with an average increase after the procedure of 26% (range 24–28). Except for the anterior
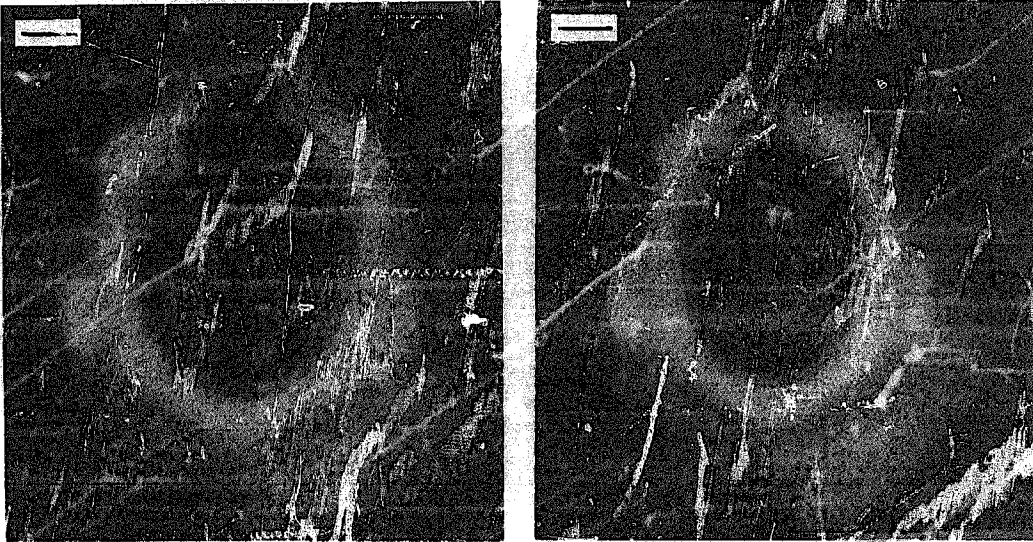
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Fig. 7. Sections through cricoid cartilage. Left, specimen harvested immediately after performing the anterior cricoid split. Right, cut edges of the cricoid catrilage re-approximated to calculate the pre-anterior cricoid split intraluminal surface area. (bar = 5 cm).
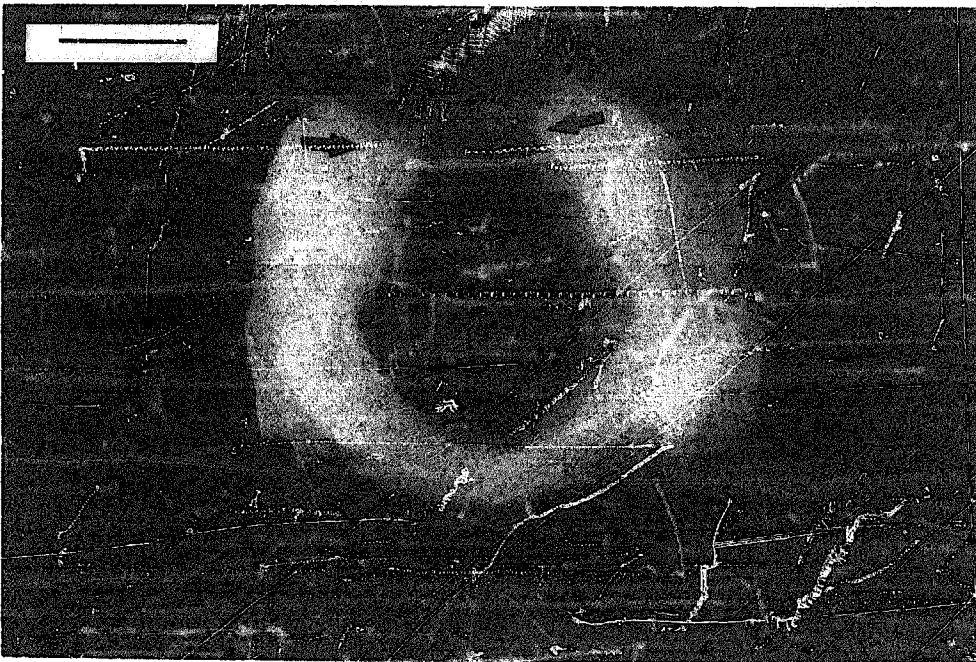

Fig. 8. Section through cricoid cartilage. Specimen 7 days after anterior cricoid split. Granulation tissue between cut edges of the cricoid cartilage which does not narrow the airway lumen. Arrows on the cricoid cartilage. (bar = 10 mm).
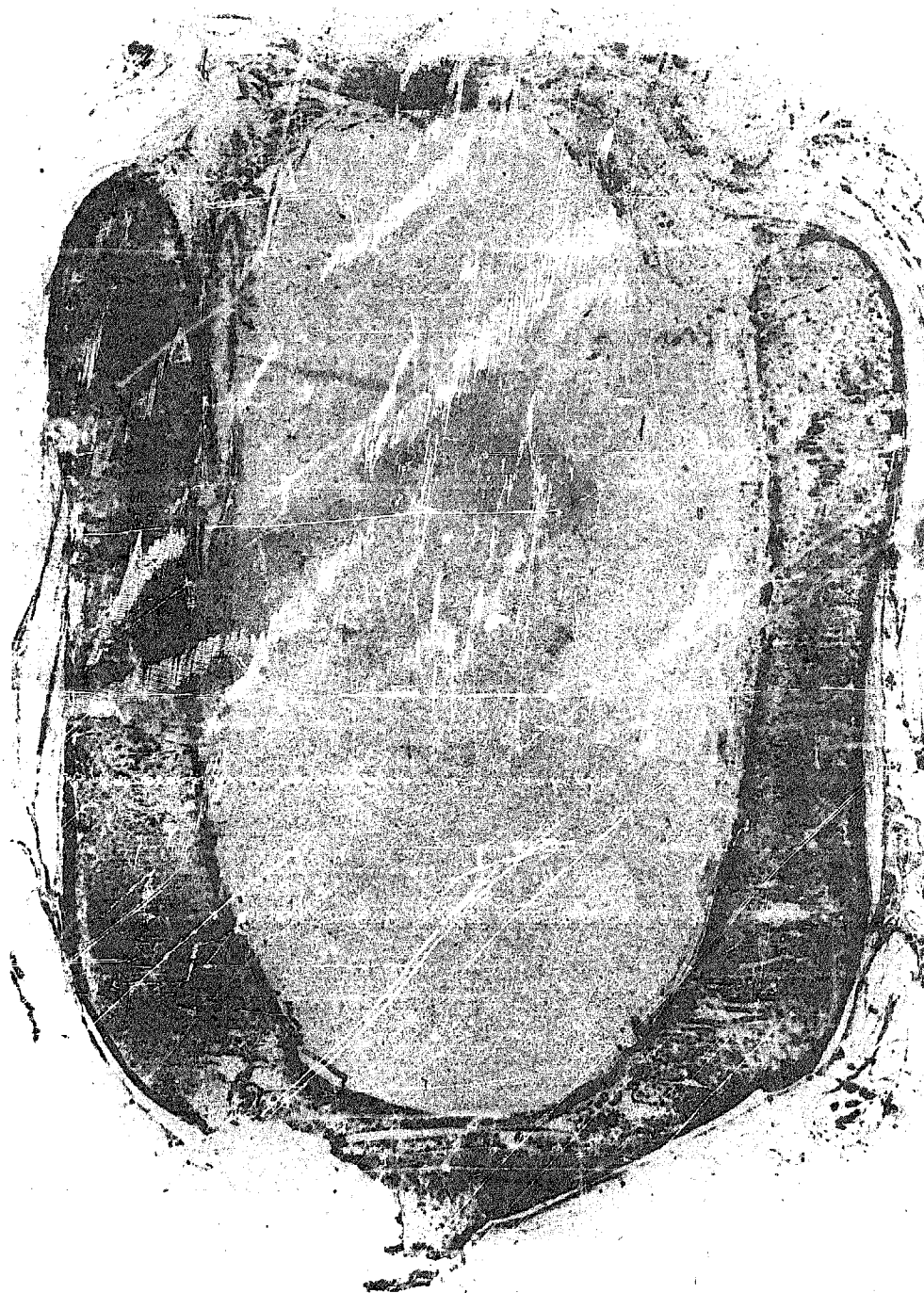
Fig. 9. Decalcified section through cricoid cartilage 6 months after anterior cricoid split. 'U' configuration demonstrated (×6).

cricoid cartilage cut, the gross and microscopic appearance of the specimens were similar to Group I.

Group IV—intubated 14 days. Anterior cricoid split day 7. Extubated and sacrificed day 14

This group of animals tolerated the endotracheal tube without any difficulty and gained weight quite rapidly during the second week of intubation.

Endoscopically on the 14th day, there was no evidence of laryngotracheal collapse at the site of the anterior cricoid split.

The subglottis in these animals at day 14 grossly appeared normal except for minimal mucosal edema. Granulation tissue was present anteriorly between the cut ends of the cricoid cartilage (Fig. 8). In contrast to the subglottis from the animals in Group II who were not split but intubated for 14 days, grossly the granulation tissue in these puppies was negligible and did not encroach into the airway lumen. Additionally, there were no areas of exposed cartilage in these animals.

Microscopically, chronic inflammatory changes were present in these animals but to a lesser degree than in Group II. Ulceration was absent as was evidence of perichondritis or chondritis.

Group V—intubated 14 days. Anterior cricoid split day 7. Extubated day 14. Sacrificed day 31

The split cricoid cartilage maintained the open configuration without any evi-

Fig. 10. Photomicrograph of the split cricoid cartilage and intervening mature scar tissue 6 months after performing the anterior cricoid split (H&E, ×50).

dence of airway granulation tissue. Microscopically, the region between the cut ends of the cricoid cartilage demonstrated the formation of scar tissue with decreased inflammatory cells, decreased vascularity, and increased collagen formation. The cricoid lumen was entirely re-epithelialized by this time.

Group VI—intubated 14 days. Anterior cricoid split day 7. Extubated day 14. Sacrificed month 6

The laryngotracheal complex in dogs reaches full growth and maturity by 6 months. No long term complications were encountered in Group VI animals following the anterior cricoid split and extubation. The cricoid cartilage maintained the open configuration at 6 months after anterior cricoid split (Fig. 9). Mature scar bridged the gap between the ends of the cricoid cartilage (Fig. 10). Grossly, neither laryngomalacia or tracheomalacia occurred as a result of the procedure.

Discussion

Over the past 15 years there has been a steady decline in the infant mortality rate. Factors attributed to the declining rate of infant deaths include the introduction of the Medicaid program, increased emphasis on prenatal care, and improved technology in perinatology. Respiratory support has been one of the single most important factors contributing to the dramatic decrease in mortality.

Although mechanical ventilation was first used for hyaline membrane disease in 1953, it was not until the early sixties that it began to be used frequenly [10]. Increased use of mechanical ventilation necessitated a means to manage and maintain the airway on a long term basis. Tracheostomy has for many years been the safest method of securing the airway in these cases. In recent years, however, this philosophy has changed. McDonald and Stocks, in 1965, advocated prolonged nasotracheal intubation of babies and children with reversible respiratory inadequacy as an alternative to tracheostomy [14]. Their work provided the foundation for subsequent acceptance of prolonged endotracheal management of the upper airway in neonates.

As the indications and frequency of nasotracheal and endotracheal support of the airway increased, so did acute and late complications associated with their use. The spectrum of complications range from acute edema and hemorrhage to acquired subglottic stenosis. The incidence of acquired subglottic stenosis following prolonged intubation in neonates and infants has varied from 12 to 20% [5,8] in the late 1960's and early 1970's to less than 2% currently [4,10,18]. Estimates that up to 45% of admissions to neonatal intensive care units require intubation for periods longer than 7 days emphasize the need to develop ways to prevent acquired subglottic stenosis from occurring following prolonged intubation [16].

The cricoid cartilage is of central importance in the discussion of airway trauma induced by prolonged intubation. The subglottic space bounded by the cricoid is the narrowest portion of the airway [6]. The cricoid lumenal diameter in the full-term newborn measures 4.5–5.0 mm [13].

As the only complete cartilaginous ring in the upper respiratory tract, the cricoid cartilage is the most common site for airway narrowing following injury due to prolonged intubation. The subglottic area is the site of abundant, loose areolar tissue in a submucosal plane that underlies a layer of respiratory epithelium. This collagenous matrix of the submucosa is easily injured and is prone to ulceration and fibrosis. The cricoid cartilage and mucosa are fixed to the larynx above and to the trachea below rendering it susceptible to injury by the pulsion movement of an endotracheal tube. The posterior portion of the larynx is interposed between the cervical vertebrae and the endotracheal tube and acts as a fulcrum, thus significant pressure is exerted on the posterior lamina of the cricoid [12].

Airway injury resulting from prolonged intubation is associated with a specific sequence of events culminating in subglottic stenosis [3]. Initially, mucosal edema and hemorrhage are present. The next stage of ulceration can be quite superficial, or as time progresses or infection intervenes, quite extensive and deep. Exposed cartilage results in perichondritis and frank chondritis. Reparative efforts range from epithelial regrowth in cases of minimal damage to the formation of exuberant granulation tissue and subepithelial fibrosis producing dense scars.

Cotton and Seid in 1980 were able to relieve the airway compromise resulting from subglottic narrowing in a group of neonates who were intubated for prolonged periods by vertically incising the cricoid cartilage and upper tracheal rings with mucosa to allow the damaged mucosa and cartilage to 'decompress' [3]. Review of the cases reported in the literature demonstrates that 72% of the children have been successfully extubated and a tracheostomy avoided by utilizing the anterior cricoid split [1,2,3,7,9,11,15,17].

The indications for performing the anterior cricoid split have recently been expanded to include older children with a history of acquired subglottic stenosis resulting from prolonged intubation who developed progressive airway difficulties several months after extubation. Holinger used the term 'secondary' anterior cricoid split to describe this distinctly different group of children [11]. These children are successfully extubated in 85% of the cases [1–3,7,9,11,15,17]. Cotton states the success in this 'secondary' group is influenced by the improvement in pulmonary function that occurs with increasing age [2].

Cotton and Seid have gone on to outline specific criteria for the use of the anterior cricoid split [3]. Neonates ready for extubation with subglottic pathology in whom the alternative would be a tracheotomy are candidates. However, extensive glottic pathology was a contraindication. The criteria have been broadened to include young infants. The oldest child in Cotton's series was 28 months old [2]. Infants with glottic pathology have also been extubated. This may be the result of a change in the actual procedure, extending the incision through the anterior commissure superiorly to within 2 mm of the thyroid notch instead of splitting just the cricoid and upper two tracheal rings.

Holinger has outlined several mechanisms which may be responsible for the success of the anterior cricoid split [11]. Interruption of the cricoid cartilage decreases the pressure exerted by the endotracheal tube on the cricoid mucosa, restoring circulation and decreasing mucosal inflammation. Secondly, Holinger

states that incision of the mucosa and other soft tissue within the lumen opens this tissue to drainage, including submucosal cysts. Thirdly, the inherent elasticity of the cricoid cartilage allows it to 'spring' open after being split resulting in an increased intralumenal surface area.

Hawkins [10] discusses the resiliency of the neonate's cartilage. Embryonic cartilage is hypercellular with little intervening matrix. With growth, the matrix increases in amount and the composition changes. There is a decrease in water content and more fibrous tissue is present, thus the cartilage is more rigid.

We postulate that the cricothyroid muscle may play a role in the 'springing' open of the divided cricoid cartilage and the persistent open configuration. The orientation of the cricothyroid muscle predicts that the cut ends of the cricoid cartilage would be pulled laterally, superiorly and posteriorly. This action bilaterally tends to separate the cut ends of the cricoid cartilage and therefore maintain its open position.

An excellent canine model to study the mechanisms of acquired subglottic stenosis was developed by Supance et al. [19]. A short segment of endotracheal tube was placed endoscopically into the laryngotracheal complex. After 7 days of intubation, minimal and reversible subglottic disease is present whereas after 14 days, the subglottic injury produced ultimately results in a mature cicatricial subglottic stenosis.

We were able to confirm the findings of Supance et al. [19]. Seven days of intubation in our animals produced a mildly injured laryngotracheal complex, whereas 14 days elicited a much more intense reaction. Performing the anterior cricoid split after 7 days of intubation not only prevented the progression of subglottic injury from intubation, but essentially allowed the subglottis to return to a nearly normal status.

The cricoid cartilage 'sprung' open in each of the animals in which the anterior cricoid split was performed. The elastic nature of the puppy cartilage along with the action of the cricothyroid muscle contributes to this action. The cricoid cartilage opened even if the endotracheal tube was removed just prior to splitting the cartilage. The hypercellular nature of this immature cartilage accounts for its plasticity whereas older cartilage is less cellular, more fibrous and more rigid. We postulate that the cricothyroid muscle may play a role in distracting the cut ends of the cricoid cartilage.

The animals in Group III gave us the opportunity to accurately measure the acute increase in intralumenal surface area seen by performing the anterior cricoid split. The surface area within the cricoid cartilage was increased by an average of 26%. This increase does not take into account the added increase in surface area that results from the expected resolution of mucosal edema and granulation tissue.

Within 3 weeks of performing the anterior cricoid split, the lumen of the cricoid was re-epithelialized. At no point post-operatively was there evidence of collapse at the site of the split and no long term complications from the anterior cricoid split were appreciated.

At full maturity, the cricoid cartilage maintained the 'U' configuration. Thick, fibrous tissue bridged the gap between the cut ends of the cricoid. Histologically,

functionally and grossly, the cricoid cartilage matured in a normal fashion in the animals in which the cricoid split had been performed.

Acknowledgements

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