We became aware of the vascular anatomy of the postcricoid region by a cluster of 4 children seen from 1998 through 2002 who had “postcricoid hemangiomas” \(^1\) (Figure 1). Informal analysis of findings from subsequent office fiber-optic laryngoscopies suggested that a violaceous bulge, which at times appeared much like a postcricoid hemangioma, was more frequently seen in this region than would be expected, given the rarity of these vascular anomalies \(^2\)-\(^{11}\) (Video 1; http://www.archoto.com). In looking for postcricoid vascular coloration, we became conscious of a consistent phenomenon not, to our knowledge, previously appreciated: a cyclical engorgement of the postcricoid mucosa that was coincidental with the expiratory phase of the infant’s cry (Video 2). Interestingly, the prominence of these tissues, which occasionally has vascular coloration, seems to be most pronounced and most regularly observed in newborns and infants. We term this enlargement the “postcricoid cushion.”

Further insight into the morphologic significance of the postcricoid cushion occurred serendipitously during review of a French anatomy text from 1854 by Bourgery and Jacob\(^12\) in which there is a beautiful color illustration of the postcricoid region, demonstrating a rich vascular plexus at this site (Figure 2). Subsequent historical searches of the anatomical literature confirmed that this plexus of veins has been extensively studied.\(^{13\text{-}24}\) The plexus is relatively more prominent in fetal dissections than in adult ones.

Our hypothesis is that these observations are related and can be woven into a rational narrative that is anatomically, physiologically, and clinically consis-
To test this premise, we analyzed a random selection of findings from office fiber-optic laryngoscopies of infants and children to see if the presence of the cushion could be consistently confirmed, how often it had any vascular coloration, and if it is more frequently seen in younger compared with older children. We also performed a search of the literature on the assumption that there exists a body of knowledge in other subspecialty fields that may provide a unified understanding of the postcricoid cushion.

**LARYNGOSCOPIC ANALYSIS**

After institutional review board approval, we selected and reviewed 125 consecutive flexible fiber-optic laryngoscopic (FFL) examinations on patients from birth to age 17 years were selected from our archived collection. FFL is routinely performed in our pediatric otolaryngology outpatient clinic as part of the workup for a variety of disorders of the pharynx and larynx. Videos are recorded and stored using KayPentax software and then stored on labeled CD-ROM discs (KayPentax). Videos were viewed in real time and in slow motion for the presence or absence of engorgement of the postcricoid region. If seen, we separately rated the fullness “small,” “medium,” or “large.” Any vascular-appearing violaceous discoloration of the postcricoid region was also looked for and was categorized as “none,” “transitional,” or “obvious.” Each video was judged independently by each reviewer. On all videos for which the reviewer’s ratings were different, the video was replayed until a consensus was reached.

Guided by the consistent findings in the anatomical literature of a relatively larger plexus of vessels in fetuses and infants compared with those in older children and adults, we performed a logistic regression analysis on our data to determine the age at which the probability of seeing a cushion exceeds the probability of not seeing one. Based on this analysis, the patients were then divided into 2 groups according to the derived age. Statistical analysis was performed using IBM SPSS Statistics software (version 18; IBM Corp).

**RESULTS**

A total of 125 videos of office-based FFL examinations in pediatric patients were reviewed. Patient ages ranged...
from 3 weeks to 17 years, with an average age of 4.6 years and a median age of 2.3 years. The most common presenting symptom for laryngoscopy was stridor (Table 1), and the most common final diagnosis was laryngomalacia (Table 2). There was no association of the presenting symptoms or final diagnosis in clinic with the presence of a postcricoid cushion.

Of the 125 videos, 119 had technically adequate visualization of the postcricoid region, while 6 were considered unusable owing to inability to assess the postcricoid space. The postcricoid cushion was seen in 61.3% of videos and rated as large in 8.4% of all patients, medium in 27.7%, and small in 25.2%. It was not present in 38.7%.

Most children (74.8%) did not have any vascular coloration of the postcricoid region. An obvious bluish-purple color was seen in 4.2% of patients, and 21.0% were rated as transitional. Those patients with a large postcricoid cushion were much more likely to have an obvious bluish-purple discoloration (30.0%), compared with patients with a medium (3.0%), small (3.3%), or absent (0%) cushion (Figure 3). Logistic regression demonstrated that the age at which the probability of seeing a cushion exceeds the probability of not seeing one is 24 months (2 years). Therefore, patients were divided into 2 groups: those younger than 2 years and those 2 years or older. There were 56 patients younger than 2 years, 49 (88%) of whom had a visible postcricoid cushion; 14% had a large cushion, 45% had a medium cushion, and 29% had a small cushion. In this group, 13% did not have a visible cushion (Figure 4). There were 63 children in the group that was 2 years or older; 24 (38%) had a visible postcricoid cushion, 3% had a large cushion, 13% had a medium cushion, and 22% had a small cushion. In this group 62% did not have a visible cushion. The infants younger than 2 years had a significantly higher rate of postcricoid cushions than children 2 years and older (P < .001; 2-tailed Fisher exact test).

<table>
<thead>
<tr>
<th>Table 1. Primary Symptoms for Clinic Referral in 125 Patients</th>
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<tr>
<td><strong>Presenting Symptoms</strong></td>
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<tr>
<td>Stridor</td>
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<td>Hoarseness</td>
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<td>Snoring</td>
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<td>Noisy breathing</td>
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<td>Cough</td>
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<td>Weak voice</td>
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<td>Nasal obstruction</td>
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<td>Dysphagia</td>
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<td>Dyspnea</td>
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<tr>
<td>Cyanotic spells</td>
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<td>Velopharyngeal insufficiency</td>
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<td>Sore throat</td>
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<td>Recurrent pneumonia</td>
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<td>Globus sensation</td>
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<td>Failure to thrive</td>
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<th>Table 2. Final Diagnosis After Laryngoscopy in 125 Patients</th>
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<tr>
<td><strong>Diagnoses</strong></td>
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<tr>
<td>Laryngomalacia</td>
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<tr>
<td>Normal</td>
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<tr>
<td>Sleep apnea</td>
</tr>
<tr>
<td>Vocal cord paralysis</td>
</tr>
<tr>
<td>Gastroesophageal reflux disorder</td>
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<tr>
<td>Vocal cord nodule</td>
</tr>
<tr>
<td>Adenoid hypertrophy</td>
</tr>
<tr>
<td>Laryngeal web</td>
</tr>
<tr>
<td>Episodic croup</td>
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<tr>
<td>Hemangioma</td>
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<tr>
<td>Papillomatosis</td>
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<tr>
<td>Subglottic stenosis</td>
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<tr>
<td>Caustic ingestion</td>
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<tr>
<td>Epidermolysis bullosa</td>
</tr>
<tr>
<td>Laryngeal cleft, type II</td>
</tr>
<tr>
<td>Posterior glottic stenosis</td>
</tr>
<tr>
<td>Paradoxical vocal cord movement</td>
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<tr>
<td>Tracheal stenosis</td>
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<tr>
<td>Vocal cord cyst</td>
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</table>
The vascular anatomy of the postcricoid region has been well defined in the literature, however in a “frag-mented” fashion, with each specialty referring primarily to its own canon. A comprehensive review divided by specialty follows.

Anatomic Literature

The 1854 anatomy text of Bourgery and Jacob\textsuperscript{12} has already been cited, but historically, it was Jean Cruveilhier\textsuperscript{13,14} (1791-1874), the French anatomist, who first described a very considerable pharyngeal venous plexus in the postcricoid and posterior hypopharynx in 1834.

In 1871, the German anatomist, Hubert von Luschka\textsuperscript{15} (1820-1875), provided a more detailed description of this plexus, including the first illustration, and termed it the “pharyngolaryngeal plexus” (Figure 5). He noted that the plexus had a ventral portion on the dorsal aspect of the cricoid cartilage and a dorsal portion on the posterior pharyngeal wall. He observed that the plexus lies at the narrowest part of the pharynx. Unaware of a muscular upper esophageal sphincter, he suggested that the distension of these veins forms a barrier between the pharynx and cervical esophagus.

In 1887, Bimar and Lapeyre\textsuperscript{16} described a remarkable venous plexus consisting of veins measuring 1 to 3 mm and connected by many anastomoses belonging to the lower or laryngeal pharynx. They noted that in contrast with other venous networks that progressively enlarge with age, at this site the reverse was true: the pharyngeal venous plexus was proportionally larger and better developed in infants than in older children and adults.

In 1918, another German anatomist, Curt Elze,\textsuperscript{17} further refined the detail of the postcricoid venous supply. He noted valves in the plexus that direct blood flow cranially and described a “meandering” network receiving tributaries from the mucosa and draining into the superior laryngeal veins. He described this venous system as a “rete mirabile,” an anatomical term for a complex meshwork of similar vessels, and provided an illustration of the network. In a 1919 follow-up study, Elze and Beck\textsuperscript{18} demonstrated that the posterior cricoid rete mirabile is

**Figure 3.** Vascular coloration of the postcricoid cushion based on size.

**Figure 4.** Presence of a postcricoid cushion in relation to patient age.

**Figure 5.** Illustration of postcricoid venous plexus, reproduced with assistance from Stanford Medical History Center (http://lane.stanford.edu /med-history/index.html) from von Luschka.\textsuperscript{15}
present only on the posterior surface of the cricoid and not on the adjacent piriform sinuses. They also described a plexus on the posterior pharyngeal wall, which is more inferior than the posterior cricoid plexus, and suggested that the engorgement of the opposing venous networks formed a valve separating the pharynx and esophagus.

In 1942, Baston,19 while studying the vertebral veins via latex injections of the dorsal phallic vein of male cadavers, found that in several specimens the veins of the pharynx were also filled. To study this further, he injected yellow oil paint, thinned with turpentine, into the veins of the inferior constrictor muscle. He found a rich plexus of veins in the postcricoid region and posterior pharyngeal wall, confirming description by Elze17 and Elze and Beck20 of the postcricoid plexus being higher than the plexus in the posterior pharyngeal wall.

In 1951, Butler20 studied the pharyngeal venous anatomy of human fetuses and adult autopsy specimens with injected India ink or a cast producing neoprene. He provided a description of a pharyngolaryngeal venous plexus, with the ventral portion in the submucosa of the dorsal surface of the cricoid cartilage, lying on the medial part of the oblique and transverse arytenoid muscles and the tendon of origin of the longitudinal muscle of the esophagus. The plexus formed “two longitudinal masses”20(p281) on each side of the cricoid midline separated by a gap of 2 to 6 mm. Cranially, the 2 halves of the plexus were “united by numerous cross anastomoses”20(p281) embedded in a mass of mucous glands. The veins were confined to the postcricoid region and did not extend into the pyriform sinuses. Interestingly, Butler20 referenced an otolaryngologist, A. S. H. Walford, who described the bluish “bolster-like” swelling in the hypopharynx of a living person during mirror laryngoscopy.

From 1984 to 1985, using latex and then India ink injections, Tose et al23 visualized the anterior and posterior pharyngeal venous plexus and further delineated the area into venous networks and “pads.” They speculated that the pads may become turgid during inspiration and thereby obstruct the entrance of air into the esophagus.

More recently, Ramaekers et al,24 in a 1990 anatomical description of the pharyngoesophageal transition zone, suggested that the orientation of the venous plexus provides rigidity during swallowing for easier passage of the bolus into the esophagus. They also noted a striking difference between the plexus in the fetus and adult, with the fetal plexus being an extensive venous labyrinth with numerous fenestrations and in the adult a thin system of longitudinal parallel veins.

Radiographic Literature

Radiologists have consistently noted a convexity of tissue behind the cricoid on swallow studies. Pitman and Fraser,22 in a study of barium swallow examinations for dysphagia, identified a “postcricoid impression” in 86% of patients with dysphagia and 90% of control patients. Considering this to be a normal finding, they suggested that it is a prolapse of mucosa over a venous plexus. Additional injection studies demonstrated a distended postcricoid venous plexus, which they compared with the pseudostrangulation sign seen at autopsy in patients with congestive heart failure.

There is an odd report by Friedland and Filly26 of a postcricoid impression on barium esophagram, which led to the erroneous diagnosis of an esophageal tumor. Surgical exploration revealed no mass, and findings from a postoperative esophagram again demonstrated the impression, which was then determined to be a normal finding.

Dodds et al27 reviewed findings from normal radiographic swallows and noted a posterior cricoid impression, distinct from cervical esophageal webs.

Allen et al28 reviewed fluoroscopic swallowing evaluations in adults with and without dysphagia. They identified a posterior cricoid “plication,” distinct from a web or cricoid arch impression, in a third of all patients, attributing it to prolapsing mucosal folds over the muscles of the postcricoid region. They noted that the plication often changed shape during the swallow.

Schmalfuss et al29 described the normal appearance of the postcricoid region on computed tomographic (CT) scans and magnetic resonance imaging (MRI) and noted marked enhancement of the mucosa in the postcricoid region in 87% with CT imaging and 78% of patients on MRI, with no significant differences based on age or sex.

Otolaryngologic and Endoscopic Literature

In the otolaryngologic literature, descriptions of the vasculature of the postcricoid region are mostly confined to case reports and small case series of postcricoid hemangioma. A retrospective review of the published cases, including the clinical photographs, reveals that in some cases, the findings are more comparable with an engorged postcricoid cushion than with a hemangioma, and often behaved as such (Table 3).

The first clinical report of a postcricoid hemangioma was by Goldsmith et al2(p851) who described a “dark, vascular appearing mass in the postcricoid area,” which “dramatically increased in size” with crying, and caused complete obstruction of the esophageal inlet leading to failure to thrive. After tracheotomy, the lesion was excised using carbon dioxide laser; the pathologic findings were consistent with a capillary hemangioma.

Tonsakulrungruang3 reported a dark blue mass on the posterior surface of the cricoid, just above the cricopharyngeal opening. Angiography showed that the mass was supplied by the left superior thyroid artery; the pathologic findings were consistent with hemangioma.

In a letter to the editor in 1997, Gyokeres et al4 described a hemangioma on the retrocricoidal area that extended to the anterior cervical esophagus, appearing during retching, moving in peduncular fashion.

Ludemann and Kozak5 described 4 infants who presented with stridor and dysphagia and who were noted to have swelling of the esophageal inlet mucosa, occasionally with dark reddish-purple staining, that was most prominent while the infants were crying. In at least 1 case, the lesion was difficult to visualize in the operating room.

Desutter et al6 reported the cases of 2 infants with postcricoid hemangioma presenting with dysphagia. One had
an MRI scan, which showed a hypervascular lesion at the approximate level of the dorsal pharyngeal plexus. Both patients underwent systemic steroids with methylprednisolone, with subsequent regression of the lesion and resolution of the dysphagia.

Discolo and Koltai reported the cases of 4 children, ages 8 to 15 months, with postcricoid hemangioma. These patients were referred to the otolaryngology department for different reasons, including stridor and aspiration, dysphagia, and laryngomalacia. The initial diagnosis was made on office flexible laryngoscopy, with which the vascular lesions in the postcricoid region were seen. All patients underwent operative airway evaluation, during which the mass was often difficult to identify. In 1 case, the lesion was difficult to visualize in the operating room, but a flexible laryngoscopy was performed in the recovery room with the patient crying again demonstrated a large, expansile lesion. All 4 children were treated conservatively with observation, with no further sequelae.

Zur et al reported 4 cases of postcricoid hemangioma, including a 3-year-old child with dysphagia who, while crying, had “ballooning of the postcricoid mass into a blush, grape-like vascular appearing mass.” A swallow study confirmed the ballooning effect during crying. Findings from an MRI scan were unrevealing, and Valsalva and Trendelenburg positioning was necessary to demonstrate the mass during operative endoscopy. The other 3 patients also had ballooning with Valsalva positioning or cry, and 3 of the 4 underwent excision with potassium-titanyl-phosphate laser or carbon dioxide laser. The pathologic findings of the resected tissue on 1 of their patients with extensive venous malformation (VM) and lymphatic malformations were consistent with a VM with an increased number of small stromal vessels and multiple, dilated vascular channels.

Awwad and Mortelliti reported the cases of 4 patients who on flexible laryngoscopy had a bluish expansile lesion in the postcricoid region, with the “propensity to enlarge with crying or straining.” They described a patient with a significant postcricoid lesion while crying, although “when the child was calmed with a pacifier, the mucosal bulge diminished substantially.” Three patients had operative airway evaluation, during which the mass was often difficult to identify. In 1 case, the lesion was difficult to visualize in the operating room, but a flexible laryngoscopy was performed in the recovery room with the patient crying again demonstrated a large, expansile hemangioma. The MRI scans in 1 patient did not reveal any vascular abnormalities. One patient was followed for 6 years with serial laryngoscopy, with a substantial regression in the size of the lesion.

Folia et al described 4 cases of postcricoid hemangioma, 3 of which were treated with open excision after failing treatment systemic steroids. In 1 case, an MRI scan showed an encircling lesion in the cervical esophagus on T2 and T1 with gadolinium. Pathology results from the excisional cases are not provided.

Sternbach et al reported a 7-month-old with coughing spells while feeding. She had a round bluish mass in the postcricoid area during crying or straining, but it disappeared when she relaxed. She was treated with systemic steroids for 1 month, and the lesion was no longer seen at 18 months.

Finally, in an recent study by Parhizkar et al, patients with vascular anomalies of the airway were compared. Specifically, the characteristics of airway infantile hemangiomas (IHs), a vascular tumor, are contrasted...
with those of airway VMs, a vascular malformation. They found 11 patients with IHs and 6 with VMs. Those with IHs had a younger age at presentation, all presented with stridor, and typically had associated cutaneous IHs. The IHs were in the glottis or subglottis. The VMs were located in the postcricoid (in 6 of 6 patients) and epiglottic regions, and the patients presented at an older age and were less likely to require intervention. The distinction is confirmed with erythrocyte-type glucose transporter protein (GLUT-1) testing, which is a useful immunohistochemical marker for IHs. It has been shown that IHs have intense endothelial GLUT-1 immunoreactivity during all phases of these lesions (proliferation, plateau, involution), but other vascular malformations, including VMs, have no GLUT-1 immunoreactivity.31 In the study by Parhizkar et al,30 the IHs stained positive for GLUT-1, and the VMs were negative for GLUT-1, as expected.

**COMMENT**

Buttressed by our study and review of the literature, the analysis and interpretation of our findings are based on the following propositions:

1. There is a cyclical engorgement of the postcricoid region, most notably during the expiratory phase of an infant’s cry. We term this phenomenon “the postcricoid cushion.”
2. The postcricoid cushion is more prominent in infants and less noticeable or not observed in older children.
3. Vascular coloration of the postcricoid cushion is infrequently but regularly observed.
4. There is a well described venous plexus that corresponds to the site of the postcricoid cushion.
5. This plexus diminishes in relative size with initial growth and maturation.
6. Vascular anomalies of the postcricoid region are rare.
7. Stridor and dysphagia are the most common symptoms of postcricoid vascular anomalies.

In the videos, the recurring dilation of the postcricoid cushion, which was most prominent during the expiratory phase of crying, is well visualized in nearly every infant. The cycle of the cry begins with a large inspiratory effort, with hypopharyngeal and supraglottic dilation, a descent of the larynx, and maximal abduction of the vocal cords. The postcricoid cushion at this phase can generally be visualized as a rounded mound covered with loose mucosa. As the expiratory phase of the cry begins, the vocal cords adduct, and the larynx begins to rise as the hypopharynx constricts around it. As the cry emerges, the supraglottic folds constrict, and the postcricoid region becomes full and prominent. Expanding tightly against the posterior and posterolateral hypopharyngeal wall, which symmetrically appears to actively tighten toward it, the cushion presses both posteriorly as well as laterally at a level overlying the posterior lamina of the cricoid cartilage. The carotid arteries, which pulsate in the hypopharynx just lateral to the cushion, medialize during the cry and are tightly opposed by the cushion (Video 2). In most circumstances the shape of the cushion is ovoid, but at times it appears to have a midline raphe and is bilobed (Video 3). This is consistent with the dissections of Butler20 and Bassett and Gruber,21 which show the separate but connected bilateral plexus of veins at this site (Figure 6 and Figure 7). A modest 4.2% of the videos demonstrate a bluish to violaceous discoloration of the cushion that is more noticeable during expiration (Video 4). With extreme agitation, the entire hypopharynx constricts around the larynx, and the posterior pharyngeal wall overrides the cushion obliterating its view.

The postcricoid cushion can also be demonstrated on direct microlaryngoscopy by lifting the larynx forward with a horizontally held right angle probe (Figure 8 and Video 5). What becomes apparent is the posterior aspect of the circumferential vascular plexus mentioned by von Luschka, Elze, and others.15-23 The posterior component is less prominent than the cushion and somewhat caudal to it. This region is harder to see on our fiberoptic examinations because the cushion, being more superior, overlaps it during engorgement.
We propose that the plexus of veins overlying the posterior cricoid cartilage, so well documented in the anatomical literature, is the same mucosally covered entity that forms the postcricoid cushion we observe during flexible laryngoscopy in our patients and that is seen radiographically during swallowing. The locations correspond. The variability in morphologic characteristics, with prominence of the cushion’s midline raphe, is in agreement with the dissections. The intermittent violaceous discoloration infers a vascular relationship. The prominence of the cushion in our youngest patients parallels the relative robustness of the plexus in fetal dissections. There is also a dynamic physiologic correspondence between the cushion and the plexus.

During the expiratory phase of an infant’s cry, there is a sharp increase in intrathoracic pressure with a concurrent decrease in venous return into the right atrium. The resulting venous congestion of the head and neck, most recognizable in the plethoric facial discoloration of a wailing infant, is coincidental with the balloononing prominence of the cushion during the cry. Indeed, the case reports repeatedly described a cyclical engorgement and discoloration associated with the patient cry, which resolved on relaxation.²,⁵,⁷,⁹,¹¹

The correspondence of location between the postcricoid plexus, cushion, and published descriptions of postcricoid vascular anomalies strongly implies the plexus as the source of those lesions. However, the review of the literature on postcricoid hemangiomas suggests 2 separate entities:

1. A vascular stained postcricoid cushion, visually engaging but asymptomatic.
2. A true vascular anomaly causing dysphagia and/or airway obstruction.

Our study suggests that the former are a fairly common physiologic phenomenon, whereas the latter are both rare and pathologic. The question that then remains is whether the true vascular anomalies are indeed hemangiomas or are instead VMs. While it is reasonable to assume that a hemangioma can occur at this site, the pathologic evidence is not convincing. Goldsmith et al² described pathologic findings consistent with capillary hemangioma, and Tonsakulrungruang³ described pathologic findings consistent with hemangioma. The problem is that in the late 1980s there was much uncertainty regarding the classification of vascular malformations and what we classify today as VMs were often referred to as capillary hemangiomas. Several reports describe involution with systemic steroid treatment, but the same reports also site loss of visualization with maturation.⁶,⁹,¹¹

Zur et al² describe the treatment dilemma of infants seen for dysphagia who had postcricoid hemangiomas or vascular malformations diagnosed on office fiberoptic laryngoscopy, yet whose lesions were difficult to visualize in the operating room unless Valsalva and Trendelenburg measures were performed. They questioned why a postcricoid hemangioma is pressure dependent, whereas a subglottic hemangioma is not, and suspected that these lesions represent vascular malformations instead. It has been our observation that VMs, in general, become more prominent during anesthesia. This suggests that malformations of the postcricoid region may be expected to behave this way. In 1 child of our original 4 the lesion became more distended under anesthesia compared with what was seen on office fiber-optic endoscopy (Figure 9). In retrospect, today we would call this a postcricoid VM and not a hemangioma. We would also suggest that several of the previous case reports of postcricoid hemangioma may actually have been postcricoid VMs, or a large postcricoid cushion with vascular coloration.

The pathologic uncertainty has begun to be resolved by Parhizkar et al,³⁰ using the IH-specific immunohistochemical marker GLUT-1. They confirm that there are distinct differences in the examination and presentation of hemangiomas and VMs, and this is borne out with GLUT-1 staining. Similarly, propranolol will also help resolve this question.³² Until then, we propose the term “postcricoid vascular anomaly” (PCVA) to bridge the uncertainty.

Ludemann and Kozak⁸ are correct when they suggest that the red or purple mass seen at the esophageal inlet which engorges during cry is likely due to increased intrathoracic pressure, and that postcricoid prolapse is not a surgical disease. However, in the literature there are sev-
eral case reports of infants who presented primarily with dysphagia and have an identifiable, usually large, postcricoid vascular lesion, not what we are describing as a normal postcricoid cushion. The cause of dysphagia has to be inferred, but a mechanism based on an excess of vascular tissue above the esophageal inlet causing a “ball-valve” type obstruction seems plausible.

Evidence for this effect is found in the radiologic literature, where there are consistent descriptions of a ridge of tissue, distinct from the arch of the cricoid cartilage, which forms an impression or plication in the postcricoid region on esophagram. This is considered a normal anatomic finding corresponding to the postcricoid venous plexus seen on adult anatomical dissections and presumably to the postcricoid cushion as well. The normal plication is considered to be dynamically passive, being drawn into the esophageal inlet during the negative pressure phase of the hypopharyngeal swallow. The cause of dysphagia with PCVA is a consequence of this same effect; however, it is magnified by their greater scale compared with normal plications, resulting in a ball-valve barrier to passage of the bolus.

The ball-valve mechanism suggests that reducing the size of the PCVA should diminish symptoms. Based on the case reports in the literature, time seems to be effective in most cases. A few authors have reported success with systemic steroids and surgical excision. Because we have had no experience actively treating PCVAs we have no recommendations in this regard. Attempt at medical management seems reasonable, but without a histologic diagnosis, knowing what to treat becomes problematic.

Three theories about the function of the posterior cricoid venous plexus have been previously proposed:

1. Protection from aspiration. The cushion facilitates the bifurcation of the bolus during swallowing to flow lateral to the larynx, particularly at the level of the interarytenoid notch, with the 2 streams then coming back together at a level inferior to the larynx. This would be particularly important in the neonate and explains why the pad is proportionally larger in the younger population.

2. Protection from regurgitation. The venous plexus adds a cephalic myoangiogenic component to the upper esophageal sphincter.


All of these theories derive from anatomical dissections. Yet from our clinical perspective, the physiologic phenomenon of the postcricoid cushion is most dynamically expressed during an infant’s cry. With the expiratory phase of the cry, nearly every muscle of the child is tensed, and there is a sharp increase in both intra-abdominal and intragastric pressure. The upper and lower esophageal sphincters restrict gastric content within the stomach during these dynamic pressure changes. On endoscopy, there is a tight compression of the cushion against the posterior hypopharynx at the peak of the cry. We propose that the postcricoid cushion adds a third layer of more proximal protection against emesis during the cyclical periods of maximal intra-abdominal pressure that occur in the expiratory phase of a cry. It is interesting to note that there were no episodes of gastric regurgitation on any of our videos.

The weakness of our study is that it is neither prospective nor randomized. The observers who viewed and scored the videos were not blinded or disinterested in the topic. We acknowledge these limitations and look forward to our results being tested with further studies.

CONCLUSIONS

We have analyzed the fiber-optic laryngoscopy videos of 119 children independently selected from our clinic archive and found a consistent cyclical engorgement of the posterior cricoid region, occasionally stained violaceous, that is most prominent during crying and most conspicuous in the first 2 years of life. This is the clinical counterpart of the well described venous plexus overlying the postcricoid region and is relatively larger in the infant. We term this structure the postcricoid cushion.

The cushion is clinically related to, yet distinct from, the true vascular anomalies that occur at this site. A better appreciation of the normal physiologic phenomenon of crying and the anatomic basis for postcricoid pathologic characteristics may provide direction for effective therapy when symptoms warrant. We propose that the function of the cushion is to augment the upper and lower esophageal sphincter in preventing emesis and gastric regurgitation from elevated intra-abdominal pressure during the expiratory phase of crying. Our interpretation weaves together multiple threads of evidence from a variety of sources into a coherent and unifying narrative of the vascular anatomy, pathologic characteristics, and physiologic mechanisms, of the postcricoid region.
REFERENCES