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New Insights into the Pathomechanism of Postintubation Arytenoid Subluxation

Friedrich P. Paulsen, M.D., Heinrich H. Rudert, M.D.,† Bernhard N. Tillmann, M.D.‡*

Background: **Impaired movement of the cricoarytenoid joint with hoarseness and immobility of the vocal ligament can occur as a consequence of endotracheal intubation. The biomechanics and pathomechanism of cricoarytenoid subluxation have not been demonstrated to date.**

Methods: **The present study attempts to simulate the trauma that has been associated with arytenoid cartilage subluxation in intubation trials on 37 unfixed larynges in cadavers from persons aged 25 to 89 years. Larynges were intubated or extubated according to former conceptions of arytenoid subluxation, which assume that the arytenoid tip enters the lumen of the tracheal tube, or that a deflection of the arytenoid occurs during withdrawal of the endotracheal tube with the cuff of the tube only partially deflated. Also, manual attempts were carried out to subluxate the arytenoid cartilage. Subsequently after dissecting the left and right cricoarytenoid joint from each larynx, the morphologic changes induced experimentally were analyzed using gross microscopic and histologic methods.**

Results: **Within the scope of the experiment, it proved impossible to produce any subluxation of a cricoarytenoid joint. Histologic analysis revealed injuries of synovial folds, joint-surface impressions of the articular cartilage, and fractures in the area of the subchondral bone in some joints.**

Conclusions: **Based on the morphologic results, it was concluded that intubation trauma of the cricoarytenoid joint does not cause subluxation *per se*, but rather that formation of a hemarthros or fractures of the joint bodies lead to fixation of the joint surfaces in an abnormal position. Subsequent ankylosis may occur. (Key words: Arytenoid cartilage; cricoarytenoid joint dysfunction; intubation trauma; larynx.)**

This article is featured in "This Month in Anesthesiology." Please see this issue of ANESTHESIOLOGY, page 5A.

IN view of the frequency of endotracheal intubations, laryngoscopies, bronchoscopies, and other invasive methods currently applied to the respiratory tract, laryngeal complications are not rare events. They may include submucosal hemorrhage, granuloma formation, and subglottic edema or laryngitis with membrane formation and are often characterized by hoarseness or stridor.

Further complications include impaired movement of one or both vocal folds. Disturbances of mobility are difficult to classify by their genesis. One possibility may be damage of the laryngeal recurrent nerve.[1–10] A subluxation of arytenoid cartilage has also been held responsible in numerous case reports.[11–29]

In this latter case, one or both cricoarytenoid joints appear to be fixed in an abnormal position (fig. 1). Typical subluxation is then supposed to lead to an anterior shift of cartilage with vocal cord adduction.[12] or a posterior shift with vocal cord abduction.[16] Concepts based on the "subluxation mechanism" assume that the arytenoid tip is taken up into the lumen of the tracheal tube[13] (fig. 2) or that the arytenoid is deflected during withdrawal of the endotracheal tube with the cuff of the tube only partially deflated[18] (fig. 2). Kambic and Radsel[30] suggest an incidence of one arytenoid cartilage subluxation or less per 1,000 direct laryngoscopic intubations.

Anatomically, the human cricoarytenoid joint (CAJ) is a diathrodial joint supported by a wide joint capsule lined with synovia. The capsule is strengthened posteriorly by the cricoarytenoid ligament. Large synovial folds, which project extensively into the joint cavity, could be mistaken for a meniscus in a definite histologic section plane.[31] They are highly vascularized and lined with several layers of synovial cells.[31] The structural arrangement of the joint allows two principal types of motion: a

* Scientific Assistant, Department of Anatomy.

† Professor of Otorhinolaryngology, Head and Neck Surgery.

‡ Professor of Anatomy and Embryology.

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Address reprint requests to Dr. Paulsen: Department of Anatomy, Christian Albrecht University of Kiel, Olshausenstr. 40, 24098 Kiel, Germany. Address electronic mail to: fpaulsen@anat.uni-kiel.de

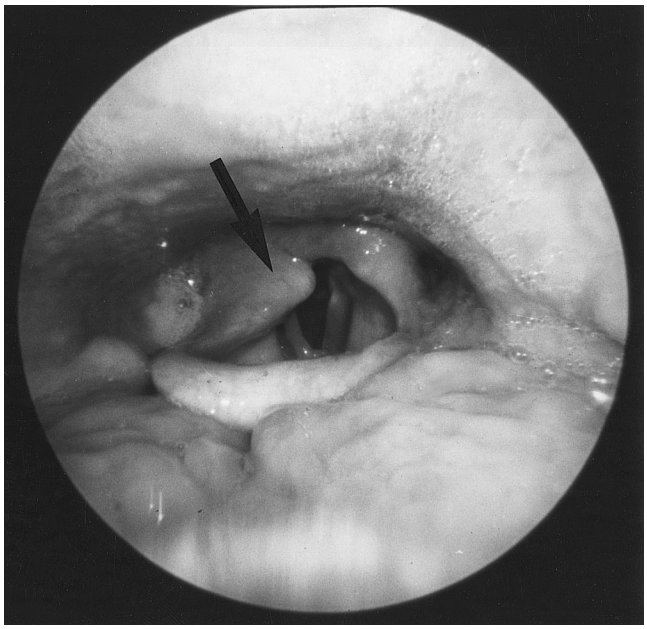

Fig. 1. Endoscopic view of larynx. Dislocation of the right arytenoid cartilage shown in anterior–medial position (*arrow*).

rocking or rotating movement around the axis of the joint, and a linear glide parallel to this axis.[32]

The present investigation attempts to elucidate the histologic consequences of the trauma that has been associated with arytenoid cartilage subluxation during endotracheal intubation. Attempts were made to simulate the forces induced by an endotracheal tube lumen displacing an arytenoid cartilage internally during intubation using unfixed human larynges, reproducing the clinical concept of intubation-induced subluxation. Extubation subluxation was emulated using the same model. Manual attempts were also made to sublux the arytenoid cartilage to further investigate the stability of the CAJ. Results and treatment options are discussed against the background of the current understanding of CAJ subluxation subsequent to endotracheal intubation.

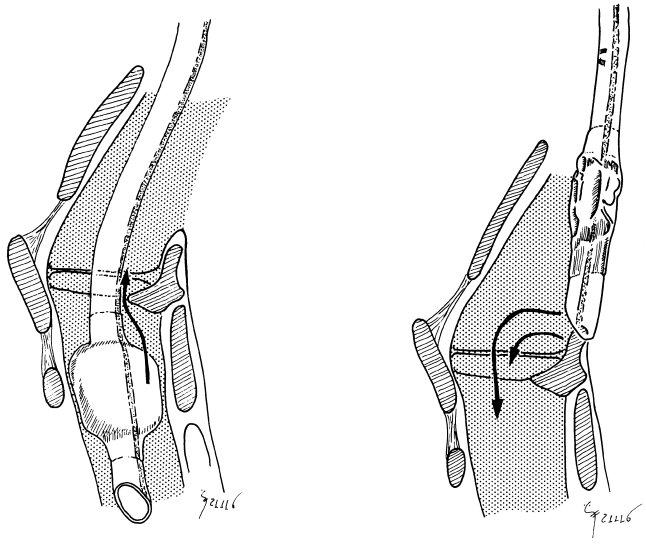

Fig. 2. Conceptions of the "subluxation mechanism." The tip of the arytenoid cartilage is loaded of with the lumen of the tracheal tube or the arytenoid cartilage is deflected during withdrawal of the endotracheal tube with the balloon of the tube only partially deflated.

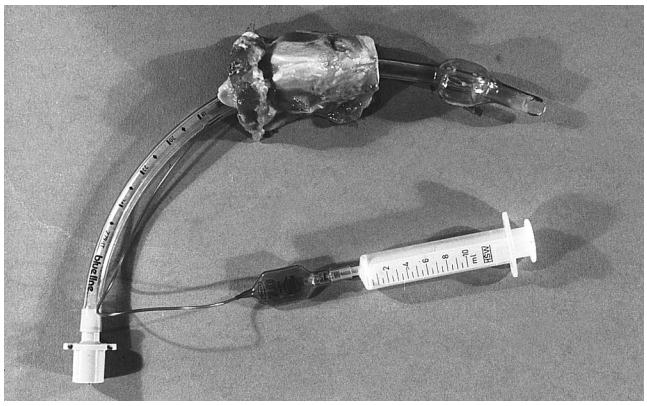

Fig. 3. Trial setup. The larynges were intubated with a tracheal tube and the subluxation mechanism of former concepts was imitated (see fig. 2).

Material and Methods

Fresh larynges were obtained from 37 body donors (19 men and 18 women; age range, 25–89 years) to the Department of Anatomy, Christian Albrecht University of Kiel, Germany, within 48 h of death. Limited information was available on the specimens, which were taken from individuals without recent trauma or infection of the larynx or diseases that might involve or affect laryngeal function. Also there was no information on whether any of the bodies had been intubated before death. For ethical reasons, the intubation attempts could not be performed with the larynges *in situ*. Experiments were carried out immediately after dissection of the larynges without previous fixation.

Intubation Trials

In 27 larynges (15 men and 12 women, age range 25–89 years) simulation of intubation subluxation was carried out with a tracheal tube (Portex, Profile blue line 7.0; SIMS Portex Limited; Kent, United Kingdom). During the experiment, the handheld larynges lay on the

Table 1. Intubation Trials

Number	Sex, Age (yr)	Experiment	cuff (ml)	pc	r/sr	r/sd	r/f	r/i	r/pc	l/sr	l/sd	l/f	l/i	l/pc
1	m, 81	Intubation r	0	x	x					x				
2	f, 33	Intubation r	0											
3	f, 64	Intubation r	0											
4	f, 86	Intubation r	0	x	x	x		x						
5	m, 55	Intubation r	0											
6	m, 25	Intubation r	0											
7	m, 63	Intubation r	0	x	x			x		x				
8	m, 58	Intubation r	0											
9	m, 40	Intubation r	0	x	x			x		x				
10	f, 58	Intubation l	0											
11	f, 49	Intubation l	0											
12	m, 51	Intubation l	0							x	x		x	
13	f, 72	Intubation l	0								x			
14	f, 78	Intubation l	0											
15	m, 75	Intubation l	0	x						x			x	
16	f, 63	Intubation l	0											
17	m, 57	Intubation l	0	x						x		x		
18	f, 89	Intubation l	0											
19	m, 73	Extubation	10	x	x									
20	m, 68	Extubation	10											
21	f, 67	Extubation	10											
22	m, 59	Extubation	10	x	x					x				
23	f, 84	Extubation	10	x						x				
24	m, 66	Extubation	10											
25	m, 81	Extubation	4	x	x					x				
26	m, 75	Extubation	4	x	x					x				
27	f, 63	Extubation	4											
28	m, 79	Manual att		x						x			x	
29	f, 31	Manual att												
30	f, 59	Manual att										x		
31	f, 48	Manual att									x			
32	f, 65	Manual att												
33	f, 83	Manual att					x					x	x	
34	m, 81	Manual att		x	x		x	x		x				
35	m, 83	Manual att		x	x			x		x		x		
36	f, 63	Manual att		x	x			x		x	x			

Experiment = method applied to each larynx; r = applied to the right arytenoid; l = applied to the left arytenoid; cuff = filling (ml); pc = pathologic changes independent of experiment; r/sr = surface roughening in the right cricoarytenoid joint; l/sr = surface roughening in the left cricoarytenoid joint; r/sd = synovial damage in the right cricoarytenoid joint; l/sd = synovial damage in the left cricoarytenoid joint; r/f = fractures of the joint body in the right cricoarytenoid joint; l/f = fractures of the joint body in the left cricoarytenoid joint; r/i = impressions on the surface of the right cricoarytenoid joint; l/i = impressions on the surface of the left cricoarytenoid joint; r/pc = damage to the right posterior cricoarytenoid ligament; l/pc = damage to the left posterior cricoarytenoid ligament; att = attempts.

posterior side (fig. 3). The first mechanism (fig. 2)[13] was tested in nine right arytenoids (table 1, nos. 1–9) and nine left arytenoids (table 1, nos. 10–18). The tube was carefully introduced into the larynx, viewed with imaging equipment (Storz-endoscope 600 BA with 0-degree optic; Karl Storz GmbH & Co., Tuttlingen, Germany), until the tip of the left or right arytenoid was inside the lumen. Then the tubus was inserted with a jerky motion. The tube was held in the examiner's left hand for each intubation procedure. The second injury mechanism (fig. 2)[18] was tested in nine larynges (table 1, nos. 19–27). The tube was carefully introduced into the larynx under view until the cuff had passed the subglottic region. The cuff was then filled with air. In six larynges, the cuff was filled with 10 ml air, and in three larynges with 4 ml air. After this, the tube was pulled out with the filled cuff using a jerky motion. All intubation and extubation experiments were applied only once with each larynx (see table 1).

In nine larynges (from three men and 6 women, age range 31–83 years), manual attempts were made to subluxate the arytenoid cartilage of both sides. Each right CAJ was squeezed by the thumb of the examiner's left hand in the anterior–inferior direction. Each left CAJ was

squeezed by the right hand, applying the same pressure in a posterior–lateral direction.

No effort was made to control the force applied with the tube. In any case, the force applied in the experiments exceeded that which would be used by an anesthesiologist in a living patient.

Light Microscopy

After dissecting the left and right CAJ from each larynx, the CAJs were fixed in 4% formalin for 1 week, decalcified in 20% EDTA, dehydrated in graded concentrations of ethanol, and embedded in paraffin. Sections (7-μm) in a horizontal or frontal plane were stained with toluidine blue (*p*H 8.5) and resorcine–fuchsine–thiazine–picric acid using the methods of Gomori and Goldner.[33] The sections were examined with a Zeiss–Axiphot microscope. CAJs not in contact with the tube in the intubation experiments served as controls (table 1, left CAJs of nos. 1–9 and right CAJs of nos. 10–18).

Results

Intubation Trials

Our attempts to simulate the proposed subluxation mechanism (fig. 2) with a tracheal tube demonstrated the impossibility of subluxating the arytenoid cartilage. Nor did manual attempts show any effect. The arytenoid cartilage moved back to its starting position following each manipulation. In some larynges, a cracking noise was heard during the intubation procedure or manual attempts.

Light Microscopy

Histologic analysis revealed injuries in the synovial folds of some CAJs as a result of the intubation attempts (fig. 4). Such synovial damage was seen in three CAJs of the intubated larynges and in two CAJs of the larynges subjected to manual attempts (table 1). Impressions on the articular surface were also observed (fig. 4), visible in five CAJs of the intubation group and in five CAJs of the manual group (table 1). In one CAJ of the intubation group and in four CAJs of the manual group (table 1), fractures were visible in the area of the joint bodies (fig. 5). The cartilaginous joint surface had been ruptured and the subchondral bone was broken (fig. 5) in these joints. No synovial damage, cartilage impressions, or fractures of the joint body were observed in the extubation group. Histologic examination of the posterior cricoarytenoid ligament did not reveal signs of damage in any CAJ.

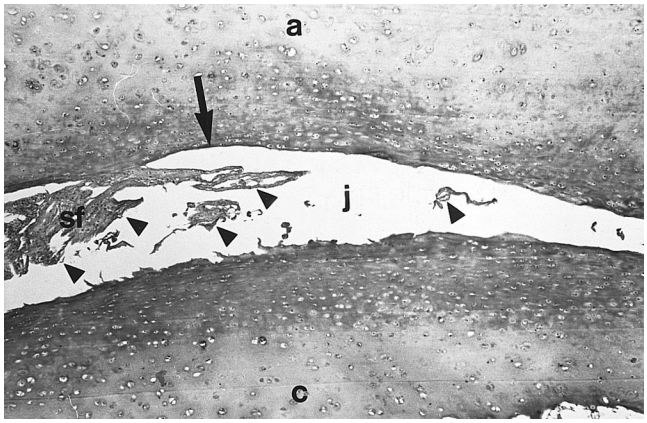

Fig. 4. Frontal section of a left cricoarytenoid joint (51-year-old man) showing synovial damage (*arrowheads*) with ragged parts of a synovial fold (sf) inside the joint cavity (j). A surface impression (*arrow*) is visible at the articular surface of the arytenoid cartilage (a). The articular surface of the cricoid cartilage (c) is fibrillated in some places. The underlying cartilage layers appear normal, showing no pathologic changes. (Original magnification, 45×.)

Several CAJs showed chondrocyte clusters near the joint surface (fig. 6), or a roughening of the entire cartilaginous joint surface (fig. 6). Fissures in the joint surface were also visible in some of these joints. Because these changes also were reflected in some of the joints in the control group (table 1, left CAJs of nos. 1–9 and right CAJs of nos. 10–18) those changes were classified as pathologic ones, which were present previous to the performed experiments (table 1). However, it is also possible that the pathologic changes were exacerbated by the experiments performed.

Discussion

Contradictions in the literature suggested the procedure of imitating endotracheal intubation in unfixed larynges with an endotracheal tube and by manual manipulation. Our experiments demonstrated that a trauma occurring during endotracheal intubation does not cause subluxation of the arytenoid cartilage *per se* and that arytenoid subluxation in the sense of a "typical subluxation mechanism" seems rather unlikely.

Based on the light-microscopic results presented, it is supposed that *in vivo* injuries—caused by unphysiologic stress and, in case of incomplete neuromuscular blockade, motor reactions during endotracheal intubation—to the large synovial folds and the outermost layers of the articular cartilage lead to joint cavity hemorrhage or serosynovitis. After the hemarthros or the serosyno-

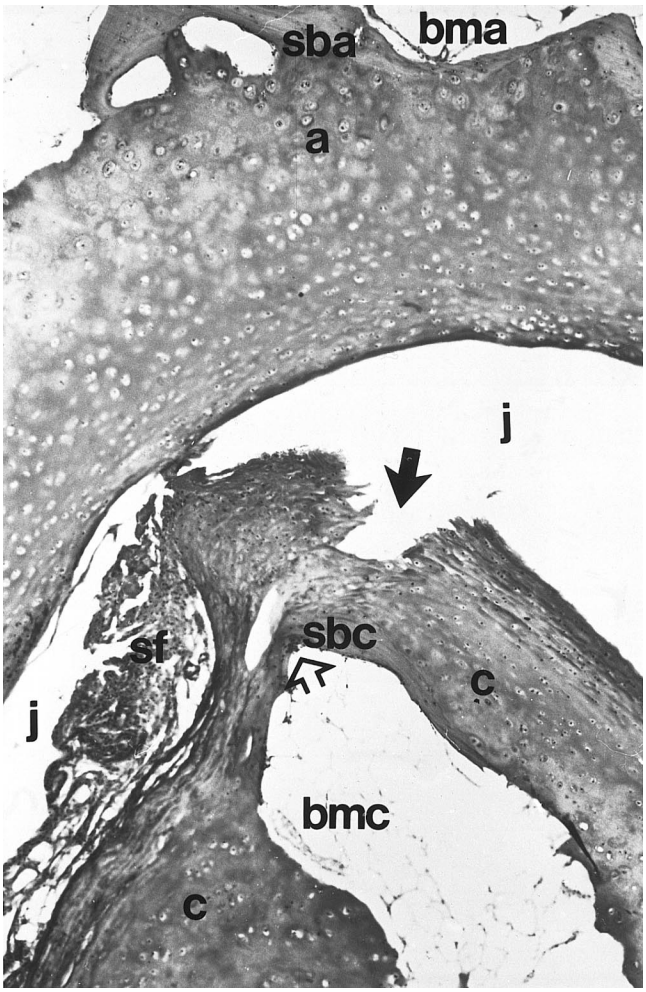

Fig. 5. Frontal section of a left cricoarytenoid joint (57-year-old man) showing a fracture of the cricoid joint body (*arrows*). The cartilaginous joint surface has been ruptured (*closed arrow*) and the subchondral bone of the cricoid (sbc) is broken (*open arrow*). j = joint cavity; a = arytenoid cartilage; sba = subchondral bone of the arytenoid cartilage; c = cricoid cartilage; bmc = fat-containing bone marrow of the cricoid; bma = fat-containing bone marrow of the arytenoid. (Original magnification, 45×.)

vitis is absorbed, this can be followed by adhesion of articular surfaces or periarticular structures that fix the arytenoid in an abnormal position. Depending on the localization of crushed synovial folds in the joint cavity, the arytenoid cartilage shifts in an anterior–medial or posterior–lateral direction. Muscle contractures conditioned by subluxation then sustain the false position of the arytenoid cartilage. Progress without treatment is characterized by fibrosis and consequent vocal fold immobility.

Reported occurrences of arytenoid cartilage subluxation are rare. Rudert[16] attributes this to earlier reports of intubation traumas in which injuries of the nervus recurrens are held responsible. Subluxation is followed by vocal cord fixation and thereby leads to a false diagnosis of recurrent laryngeal nerve damage. However, several authors have pointed out in the past that damage to the CAJ joint subsequent to endotracheal intubation is more plausible than damage to the recurrent nerve or one of its branches.[16,34] Direct trauma of the CAJ was first reported in the 1970s.[11–14,35] On the basis of the genesis of arytenoid cartilage trauma, restriction of movement can lead to transitory or permanent vocal fold fixation.[36] Analyzing the CAJs of dogs, Stanley and Colman[37] demonstrated that even slight dysfunctions of the joint capsule can result in disturbed joint function. Studying the literature of recent years on arytenoid cartilage subluxation, it is generally noticed that endotracheal intubation was in most cases carried out for a short time interval and managed without difficulty (table 2). Interestingly enough, in many cases symptoms of arytenoid cartilage subluxation were not noticed directly after extubation, but rather within 1–7 days of extubation.[12,14,20,22,25] These results are unusual for a "typical mechanically caused subluxation" of a joint. Normally, subluxation is evident immediately during mechanical manipulation and hardly any delay is noticed.

Several theories have been formulated to explain the subluxation mechanism.[13–15,18,24] "Typical subluxation" leads to arytenoid cartilage shifting in an anterior–medial[12] (fig. 1) or a posterior–lateral[16] direction. It has

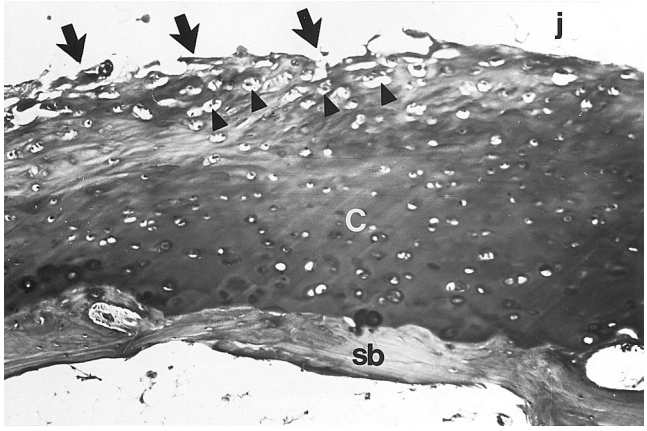

Fig. 6. Sagittal section of a cricoid cartilage (c) revealing pathologic changes of the articular cartilage that were visible previous to the experiment and are not related directly to the experiment. Beneath a roughening of the articular surface (*arrows*), chondrocyte clusters are visible near the articular surface (*arrowheads*) (40-year-old man). j =joint cavity; sb = subchondral bone. (Original magnification, 90×.)

Table 2. Comparison of the Literature

Author	Sex, Age (yr)	Side	DI	ID	DL	LL
Komorn *et al.,* 1973[34]	m, 54	r	s (3,5h)	e	po	ant/med
Prasertwanitch *et al.,* 1974[12]	f, 61	l	s (7,5h)	e	ld	ant/med
Schulz-Coulon, 1974[13]	f, 53	l	s (2h)	e	po	ant/med
Quick and Merwin, 1978[14]	m, 31	l	s (3h)	e	6	post/lat
	f, 31	l	s (4h)	d	d	post/lat
	f, 30	l	s (4h)	e	1 d	post/lat
					1 d	
Catterji *et al.,* 1984[29]	m, 34	l/r	s (0,5h)	e	1 d	ant/med
Rudert, 1984[16]	f, 37	r	s	e	nr	nr
	m, 38	r	s	e	nr	nr
	m, 58	r	s	e	nr	nr
	m, 66	r	s	e	nr	nr
	f, 36	l	s	e	nr	nr
	m, 44	r	s	e	nr	nr
	m, 56	l	s	e	nr	nr
	m, 72	r	s	e	nr	nr
	m, 41	l	s	e	nr	nr
	f, 43	r	s	e	nr	nr
Dudley *et al.,* 1984[15]	m, 27	r	s (2h)	e	po	ant/med
Brandwein *et al.,* 1986[10]	f, 70	nr	s (3h)	e	po	nr
Close *et al.,* 1987[18]	m, 59	l	l (10d)	d	nr	ant/med
Debo *et al.,* 1989[19]	m, 29	r	s	d	po	ant/med
Frink and Pattison, 1989[20]	f, 49	l	s (8h)	e	3d	post/lat
Castella *et al.,* 1991[21]	f, 77	r/l	s (4h)	e	po	ant/med
Gauss *et al.,* 1993[22]	m, 60	l	s (12h)	d	1d	nr
Sataloff *et al.,* 1994[24]	m, 53	r	nr	nr	nr	nr
	f, 26	r	nr	nr	nr	post/lat
	m, 14	l	nr	nr	nr	nr
	m, 68	r	nr	nr	nr	post/lat
	f, 29	r	nr	nr	nr	nr
	m, 39	l	nr	nr	nr	post/lat
	f, 70	l	nr	nr	nr	ant/med
	f, 50	r	nr	nr	nr	nr
	m, 56	l	nr	nr	nr	ant/med
	f, 31	r	nr	nr	nr	ant/med
	m, 58	r	nr	nr	nr	ant/lat
	m, 55	r	nr	nr	nr	ant
	f, 45	l	nr	nr	nr	ant
	f, 44	r	nr	nr	nr	ant
	m, 17	l	nr	nr	nr	ant
	m, 11	l	nr	nr	nr	ant/med
	f, 15	l	nr	nr	nr	post
Szigeti *et al.,* 1994[25]	m, 57	l	s (4h)	e	6d	ant/med

DI = duration of intubation; s = short; l = long; ID = intubation difficulty; e = easy; d = difficult; DL = duration until dysfunction; po = postoperative; LL = dysfunction localization; nr = no record; ant = anterior; med = medial; post = posterior; lat = lateral.

been proposed that the left arytenoid is subluxated more often because the laryngoscope is typically held in the left hand and the endotracheal tube is inserted from the right side.[14] Comparison of the literature (table 2) does not confirm this theory. Right and left arytenoid cartilage are involved in similar numbers. Rudert[16] reported 10 cases. Six concerned the right side, four concerned the left. Sataloff *et al.*[24] discussed 17 cases of arytenoid cartilage subluxation, with nine instances of right arytenoid cartilage involvement and eight cases with involvement of the left side.

Pröschel and Eysholdt[38] suggest that coughing parox-

ysms during extubation may be responsible for arytenoid cartilage subluxation, but there has been considerable doubt in the past as to whether subluxation of the CAJ is possible at all in a normal larynx. In a healthy state, the strong posterior cricoarytenoid ligament prevents dislocation of the arytenoid cartilage. This is confirmed by the present investigation (table 1). Chronic disease states such as laryngomalacia, renal insufficiency, acromegaly, or chronic glucocorticoid intake can lead to degeneration of the posterior ligament and therefore have been discussed as predisposing factors in arytenoid subluxation.[14] Sataloff *et al.*[24] were unable to confirm this in an analysis of their comprehensive case material.

Furthermore, diseases involving the CAJ itself also must be discussed as predisposing factors. These diseases include, for example, occurrence of cricoarytenoid arthritis,[39] rheumatoid arthritis,[40] or degenerative changes[41] in the CAJ. A recent study showed that in persons aged 40 years or older the incidence of osteoarthrotic changes in the CAJ is approximately 50%.[42] Such changes were also visible in the material used for the present investigation.

The symptomatology of arytenoid cartilage damage is, of course, highly specific (hoarseness, vocal ligament immobility) but also not very sensitive, for which reason the diagnosis and therapy of arytenoid cartilage dislocation is routinely delayed. Anterior subluxations of the arytenoid appear to be better tolerated in adults than in children, with marked hoarseness as the predominant symptom.[15,17,35] In pediatric and neonatal cases, respiratory compromise is the primary manifestation.[29,43] Posterior subluxations are associated with severe sore throat and odynophagia, as well as hoarseness.[19] The pain is most likely related to motion of the subluxated cartilage, which extends into the hypopharynx and is moved during swallowing.[19]

It can be concluded that arytenoid subluxation should be considered whenever any of the symptoms mentioned occur following endolaryngeal manipulation.[44] Patients showing no improvement of the laryngeal dysfunction should be presented to an ear, nose, and throat specialist.

The specialist should accomplish arytenoid cartilage relocation as soon as possible after recognition of arytenoid subluxation as allowed by patient status. In many of these cases, detachment of joint fixation using a spatula under light pressure or endoscopic reposition is successful.[14,15,20,35,38] However, in case of fractures in the area of the joint bodies, delayed treatment, or unrecognized arytenoid dysfunction, the CAJ may become fibrosed, and the terminal stage may be manifestation of bony ankylosis.

In summary, the present investigation demonstrates that laryngeal trauma caused by endotracheal intubation does not cause subluxation of a arytenoid cartilage *per se*, but rather the formation of hemarthros leads to fixation of the joint surface. We should therefore no longer speak of "arytenoid subluxation," preferring instead the terminology *postintubation cricoarytenoid joint dysfunction*.

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