

CORRESPONDENCE

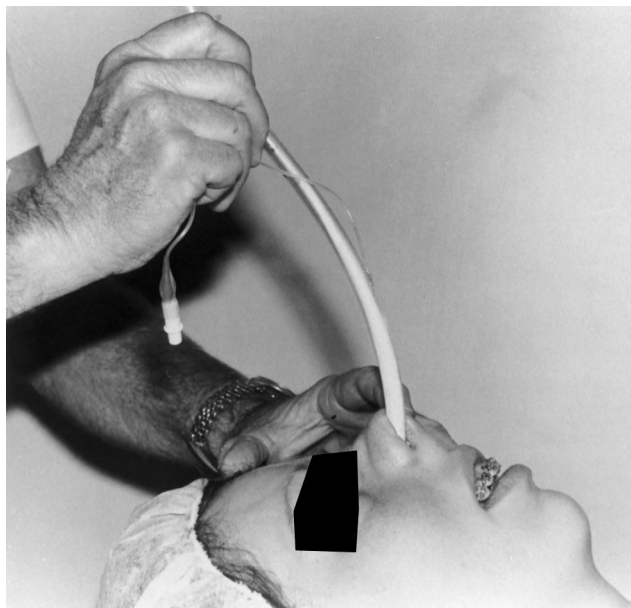


Fig. 1. Nasal tracheal intubation showing: (1) the tip of the nose being pulled cephalad as the bevel of the tube is placed through the nares, and (2) the tube being pulled cephalad as it is advanced along the floor of the nasal cavity.

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A Linguistic Crisis? Maybe. A Linguistic Crises? Definitely Not!

To the Editor:—Anatomically speaking, physicians often use the more “proper” Latin names instead of the more common lay terms. If one chooses the former, care must be taken to do so correctly.

In an otherwise clear and informative case report, the authors referred to the “left nares” and the “right nares” of a nasally intubated patient. Unless the patient in question had four or more nostrils, this usage was incorrect. One naris plus another naris makes two nares.

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Arytenoid Subluxation Caused by Laryngoscopy and Intubation

To the Editor:—I would like to question the conclusion of Paulsen *et al.*, that “laryngeal trauma caused by tracheal intubation does not cause subluxation of arytenoid cartilage.”¹ Whereas they found the forces from endotracheal tube manipulations and “manual squeezing” of the

tube cephalad (fig. 1), more likely than not directs its bevel and tip away from the turbinates and promotes their passing between the inferior turbinate and the nasal surface of the palate where the nasal passage is the largest. This, in itself, avoids turbinectomy even with a REA tube which if not pulled cephalad is likely to be directed at the turbinates.

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ment as culprit in subluxation, although blind intubation with stylets has resulted in clinical subluxation.^{2,3} However, laryngoscopy and use of stylets are typically inherent in the intubation process, and significant, if not extreme and especially localized pressure, can be transmitted to delicate structures by the tip of a laryngoscope blade. Indeed, while instructing trainees and at times personally, I have had opportunity to visualize the esophageal opening, after laryngoscope placement has lifted the larynx anteriorly. I have also felt the larynx slip backward off the laryngoscope from this position in a "crunching" manner, while facilitating visualization *via* thyroid cartilage pressure/maneuvers. Such placement of the laryngoscope would certainly allow for equal opportunity for right or left arytenoid subluxation to occur, and also under intubating conditions deemed simple.

Finally, while multiple joint fractures and ligament injuries were demonstrated histologically after trauma on multiple fresh postmortem larynxes in Paulsen's study, the dead tissues provided no opportunity for *in vivo* posttraumatic developments, including muscular spasm, bleeding, swelling, or the combination thereof, which may have led to sustained *in vivo* subluxation. Finally, their own endoscopic view of a subluxated arytenoid (fig. 1)¹ and the suggested incidence of 1/1000 after direct laryngoscopic intubation, speaks for the existence of this clinical entity under *intra vitam* forces and conditions.^{4,5}

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In Reply:—The background of our study was to show a new understanding of the pathomechanism leading to arytenoid dislocation. Based on our experiments we concluded that intubation trauma of the cricoarytenoid joint does not cause subluxation *per se*, but rather that formation of a hemarthros or serosynovitis lead to fixation of the joint surfaces in an abnormal position.¹

We agree that our study may exclude forces from singular endotracheal tube placement as the culprit in subluxation and we also agree that in a living patient the larynx undergoes movements during insertion of a laryngoscope which we were unable to simulate in our experiments. But this is not the point. Analyzing the anatomy of the human cricoarytenoid joint in several studies²⁻⁴ we were able to show that the joint can be compared with diarthrodial joints at the limbs and that the joint capsule consists of unexpected large and intensively vascularized synovial folds projecting into the joint cavity. Laxity of the joint capsule and the large synovial folds are predisposing factors for intubation trauma of the cricoarytenoid joint, potentially leading to hemarthrosis and finally to cricoarytenoid joint dysfunction.⁵

Naturally, a postmortem study is not able to show what happens in a living patient but our experiments give us hints about the probable pathomechanism of arytenoid dislocation. Our concept with the occurrence of joint cavity hemorrhage or serosynovitis and after muscle contractures is able to explain all contradictions in the literature regarding arytenoid subluxation.

How would Professor Kempen explain cases of arytenoid dysfunction that occurred some days after easy intubation. In these cases phonation was normal directly after extubation and arytenoid disloca-

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tion primary occurred after some days.^{6,7} How will he explain the experience of laryngologists⁸ treating dislocated arytenoids who report that the arytenoid was movable when touching it with a spatula under light pressure but moved back in its starting position after release.

We do not contradict Professor Kempen that arytenoid subluxation may exist under *intra vitam* forces and conditions. But our experiments speak against this concept and allow the conclusion that arytenoid dislocation is based on the pathomechanism mentioned above¹ and therefore we should speak of "postintubation cricoarytenoid joint dysfunction."

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Potency of Cisatracurium: A Correction

We would like to make a minor but needed correction to a study we recently published in *ANESTHESIOLOGY*.¹ Historically, labels of vials or ampules containing neuromuscular blocking drugs indicate the total weight of the salt of the drug per milliliter. For example, vecuronium labeling indicates that when the drug is reconstituted, each milliliter contains 1 mg of vecuronium bromide. The GRAM molecular weight of this salt is 637.7. The GRAM molecular weight of bromine is 79.9. Thus each milliliter of vecuronium only contains 0.87 mg of the active moiety or base. The same labeling convention also applies to atracurium, rocuronium, mivacurium, and succinylcholine.

Dr. Francois Donati (Departement d'Anesthesie, Center Hospitalier de l'Universite de Montreal, Quebec, Canada) recently pointed out to us that cisatracurium is labeled differently. Each milliliter of cisatracurium contains 2.0 mg of the active moiety not the salt. Thus the molar potency of the drug is considerably lower (0.066 $\mu\text{M}/\text{kg}$ vs. 0.050 $\mu\text{M}/\text{kg}$) than we indicated in our article. We have consequently recalculated all our published data regarding the relationship between molar potency and onset time. We stand by our conclusion that there is a linear relationship between the log of onset time and the log of molar potency. However, the coefficient of determination (R²) of this relationship is somewhat less impressive than before. The R² for potency *versus* time to 50% effect (see fig. 3 in our article)¹ is now 0.958 not 0.984. Similarly, the R² for potency *versus* time to 90% effect is now 0.951 not 0.977.

It appears that the pharmaceutical industry has recently changed its labeling convention without notifying the consumers of its products. Early studies with rapacuronium used the old labeling convention.² However, the current label expresses the drug concentration in milligrams of the active moiety or base per milliliter. To the best of our

knowledge cisatracurium and rapacuronium are the only two drugs so labeled.

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