Osborn et al. imply. The LMA-S™ should not be confused with the LMA-P™. It is not a single-use LMA-P™ because substantial details are designed and constructed in a different way to overcome weaknesses of other LMA’s, as the producers of the LMA-S™ promote their device. The LMA-S™’s clinical performance can only be evaluated in clinical trials. First published comparisons with a reasonable sample size between the LMA-P™ and LMA-S™ showed clinically important differences in the seal pressure between both devices.

We affirm our statement that acute airway obstruction of LMA-S™ can occur at any time, and backup strategies for the failure of the backup device LMA-P™ have to be considered.

Lorenz G. Theiler, M.D.,* Maren Kleine-Brueggeney, M.D., Robert Greif, M.D., M.M.E. *University Hospital Bern, Bern, Switzerland. lorenz.theiler@insel.ch

To the Editor.—I read with interest the article by Kheterpal et al.1 However, I am concerned that they did not control for hypothermia in their analysis. Hypothermia is considered to be a risk factor for morbid cardiac events.2,3 Without controlling for this variable, the risk assigned to their nine variables may be different than what was reported. For example, suppose the elderly patients became hypothermic more readily than the nonelderly patients. If this was the case, then the risk factor of being elderly may be overestimated, as it could have been the hypothermia and not the age that caused the problem in the elderly patient. I suspect that accurate core temperatures were not measured in most, if not all, patients who did not receive general anesthesia. However, the study population seems large enough to allow for a separate analysis of patients who did have their core temperature recorded. Do the authors have any temperature analysis that was not reported in the article?

Jonathan V. Roth, M.D., Albert Einstein Medical Center, Philadelphia, Pennsylvania. rothj@einstein.edu

References

In Reply.—We thank Dr. Roth for his interest in our article and insightful commentary. Previous literature has demonstrated an association between intraoperative hypothermia and cardiac adverse events. However, we did not evaluate this clinical element in our analysis for several reasons. First, previous data regarding hypothermia and cardiac adverse events is limited to high-risk patients who had a preexisting diagnosis of coronary artery disease or several known risk factors for coronary artery disease undergoing high-risk thoracic, intraperitoneal, or vascular procedures.4 Although our dataset included some high-risk patients, only 9.6% had a previous cardiac intervention and only 22% were undergoing high-risk surgery.5 As a result, the studied population was dissimilar to previous work, and we were skeptical of being able to identify an association between hypothermia and cardiac adverse events in this more representative population. Second, although our studied dataset was large, we were only able to observe 83 events. As a result, we had to limit the number of independent variables evaluated in the logistic regression full-model fit to reduce the impact of model overfitting.3 Hypothermia was one of several independent variables that we were unable to assess because of this statistical analysis constraint.

Finally, the absence of a consistent way to separate “hypothermic” versus “normothermic” groups in an observational dataset presented the final challenge. There are several ways to define hypothermia. First, we could evaluate median temperatures within 10-min epochs, similar to the presented hypotension analysis. Second, some may advocate that a single temperature measurement below 36°C would qualify as “hypothermic.” Third, others may suggest that we employ the absence of active warming to be consistent with prospective, controlled studies.

We agree that intraoperative hypothermia should be evaluated in future studies. We look forward to conducting large, multicenter observational dataset analyses that may offer us the statistical power necessary to do so.

Anesthesiology 2009; 111:453–4

Hypothermia Should Also Have Been Considered to Be a Predictor of Adverse Perioperative Cardiac Events

Anesthesiology 2009; 111:453

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References

(Accepted for publication April 23, 2009.)
To the Editor—Recently, a patient was discovered to have a markedly aberrant right carotid artery. A subsequent informal survey of our colleagues revealed that many anesthesia providers are unaware of the origins and incidence of this anatomical variant, and do not routinely assess patients for a symptomatic or clinically silent finding.

Our patient was a 73-yr-old male with a history of hypertension, diabetes mellitus, and obstructive sleep apnea requiring the use of continuous positive airway pressure at night, who was seen in our anesthesia preoperative clinic. He was scheduled for wide local excision and partial left auriculocutoma, sentinel lymph node biopsy, and possible parotidectomy and left neck dissection because of recurrent lentigo malignant melanoma of the left ear. He had few subjective complaints, with the notable exception of difficulty swallowing both liquids and solids and intermittent episodes of choking. The airway exam revealed a Mallampati III status. No pulsatile mass was seen, but the patient had notable and asymmetric fullness to his right posterior and lateral pharyngeal wall.

Because of the planned procedure, there was a fortuitous opportunity to review computed tomographic studies of the head and neck (fig. 1). A markedly retropharyngeal course of the right carotid artery causing ventral and medial displacement of the tissues of the palate was noted.

The internal carotid arteries have their embryonic origination in the third aortic arch and the dorsal aorta. During normal embryonic development, the dorsal aortic root descends into the chest during the eighth week of fetal life, which lengthens and straightens the course of the carotid artery. It has been suggested that incomplete straightening and persistence of the embryonic angulation can result in the presence of aberrant carotid arteries in the retropharyngeal space.1 A variety of anatomic presentations are possible, which tend to become more pronounced with the physiologic effects of aging. These include simple medial displacement of the artery, as well as kinking and outright coiling of the vessel. In older patients, the incidence has been estimated to be as high as 2%. This corresponds to our experience, as we have been confronted with a midline or retropharyngeal carotid artery three times in several hundred dissections of the head and neck for cancer.

Although there are several reviews of the retropharyngeal carotid artery in both the radiology and laryngology/head and neck literature going back to the 1920s,2–6, we could find no information on the subject in the anesthesiology literature, which perhaps explains the lack of awareness on the part of our colleagues. This is unfortunate, as a retropharyngeal carotid artery has a number of clinically relevant implications for the anesthesiologist as well as the surgeon.

First, rupture of the oropharyngeal mucosa as well as lacerations to the submucosal soft tissue as a complication of tracheal intubation is anatomic presentations are possible, which tend to become more pronounced with the physiologic effects of aging. These include simple medial displacement of the artery, as well as kinking and outright coiling of the vessel. In older patients, the incidence has been estimated to be as high as 2%. This corresponds to our experience, as we have been confronted with a midline or retropharyngeal carotid artery three times in several hundred dissections of the head and neck for cancer.

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