

Pulmonary Postoperative Complications: Is There a Place for Anesthesia?

To the Editor:

We read with a great interest the article by Canet *et al.*¹ regarding pulmonary complications after surgery. The authors have evaluated the incidence of this frequent adverse event and its risk factors in more than 2,000 patients. They found that postoperative pulmonary complications occur in 5% of patients and identify several patient-related (*e.g.*, age, low preoperative SpO₂, acute respiratory infection during the month before surgery, preoperative anemia) and surgical-related risk factors (*e.g.*, upper abdominal or intrathoracic surgery, emergency surgery, procedure duration). It is noteworthy that anesthesia was not identified as a risk factor for postoperative pulmonary complications. Instead, the authors¹ considered only two categories for this variable (*i.e.*, general *vs.* regional anesthesia).

Some patients may receive a combination of general and regional anesthesia that aims to decrease postoperative pain and postoperative diaphragmatic dysfunction, thereby reducing risk of pulmonary complications. A large randomized controlled trial has observed that combined epidural and general anesthesia after major surgery decreases postoperative pulmonary complications.² Meta-analysis has also demonstrated that epidural analgesia that lasts more than 24 h decreases the risk of pneumonia.³ General anesthesia combined with epidural analgesia is not equivalent to general anesthesia alone.

General anesthesia is also characterized by the need for ventilatory support. However, the ventilatory “setting” may be different from one patient to another, as shown in large epidemiologic studies performed in the intensive care unit.⁴ However, similar multicenter studies are lacking for surgical patients receiving general anesthesia in the operating room.⁴ Ventilator-induced lung injury was first described in patients with acute lung injury and acute distress respiratory syndrome.⁵ Experimentally, ventilator-induced lung injury has been demonstrated in animals without previous lung injury.⁵ In the context of ventilation for anesthesia, several authors^{6,7} have observed that use of large tidal (more than 10 ml/kg) or high pressure during general anesthesia may influence pulmonary complications. Thus, ventilatory setting as well as other strategies used in operative anesthesia (*e.g.*, fluid administration, analgesia management) usually comprise the “black box” in cohort studies that evaluate risk factors for postoperative pulmonary complications. It is necessary to consider that anesthesia management (*i.e.*, ventilator settings, fluid administration, drugs, techniques used) may have a positive—but also negative—impact on the risk of postoperative pulmonary complications. More data on the practice of anesthesiologists are required.

Emmanuel Marret, M.D., Ph.D.,* Samir Jaber, M.D., Ph.D. *Tenon Teaching Hospital, Assistance Publique Hôpitaux de Paris, Université Pierre et Marie Curie-Paris, Paris, France. emmanuel.marret@tnn.aphp.fr

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In Reply:

We thank Dr. Lebard and colleagues and Drs. Marret and Jaber for their interest in our study of postoperative pulmonary complications (PPC).¹ In reply, we are glad to have the opportunity to provide information that was not included in the article itself.

Lebard *et al.* compare and contrast our study with the excellent work of McAlister *et al.*,² finding discrepancies attributable to differences of aim and design. The previous study was accomplished in a single hospital and included a more narrowly defined surgical population. Our principal aim was to calculate the incidence of PPC and predict risk in a larger sample that would be more representative of a broad general surgical population. To that end, we selected patients from 59 hospitals (which together account for 63% of the anesthesia case load in our geographic area of Catalonia, Spain) using a random sampling procedure to reflect case loads over the course of a calendar year. As Lebard *et al.* point out, the two studies defined PPC according to different criteria. Our list included more minor clinical events, yet we also found that PPC had a significant impact on postopera-

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tive outcome (see our table 4) and are therefore worthy of the clinician's serious consideration. Lebard *et al.* also express surprise at the 19.5% mortality we observed in patients with PPC, finding it high; they suggest that overlapping postoperative cardiovascular complications (CVC) might have played a role. In fact, however, high mortality is not unusual in patients with PPC: mortality was 27% and 21% in two studies by Arozullah *et al.*^{3,4} Nonetheless, we did record postoperative CVC in detail in our study, finding them in 36.6% of the patients with a PPC. Thirty-day mortality in this subgroup was 33.3%, which was similar to mortality in the study of Lawrence *et al.*⁵ and in sharp contrast with the rate of 11.5% we saw in patients with a PPC but no added CVC. Meanwhile, mortality in patients with a CVC but no PPC was low in our study (3.4%). We therefore think that although the cooccurrence of a PPC and a CVC is an ominous event, the PPC still play a large role in increasing risk of death. We emphasize that, although we analyzed factors associated with PPC, it was not our aim to examine how they might have arisen. Generally speaking, if a patient first develops a PPC, the clinical course that culminates in death may also include the development of cardiovascular or other complication that will influence the outcome. Conversely, if a PPC is not the first complication to appear, its later development nonetheless will play a role.

Marret and Jaber suggest that anesthetic technique may play role in the development of PPC, and they specifically ask about the effect of combining general anesthesia with an epidural block. This subgroup accounted for 8.4% of our study population undergoing general anesthesia (n = 1,336) and comprised patients who on average were older, in a poorer state of health, and undergoing more aggressive and longer-lasting surgical procedures. In a *post hoc* analysis of our data, we compared a group of 112 patients who underwent general anesthesia with another group of 112 who received combined general-epidural anesthesia, finding no significant differences in the incidence of PPC (18.8% *vs.* 20.5%, $P = 0.867$) or pain intensity at 24 h (score of 3 or less on a visual analog pain scale, 56.3% *vs.* 67%, $P = 0.131$) (statistical results from the ARISCAT database run on March 1, 2011). Thus, there seems to be no suggestion of a beneficial effect of combined anesthesia, although we must emphasize that our study was not designed to compare anesthetic strategies. We agree with Drs. Marret and Jaber that there is a possible influence of ventilatory settings on the development of PPC. The anesthesiologists in charge of care chose the settings in all cases in our study, and although our database includes recordings of positive end-expiratory pressures, alveolar recruitment maneuvers, and hyperoxygenation, we have no reliable information on tidal volume. Finally, with regard to fluid therapy and postoperative pain, we included both in the list of potential risk factors for PPC, but neither achieved statistical significance in the bivariable analysis. We agree with Drs. Marret and Jaber that different perioperative strategies might reduce risk; nonetheless, so far, systematic analysis has found that only a few have been shown to clearly

or possibly do so,⁶ whereas others remain to be tried. We think controlled studies should now be designed to analyze the possible benefit of promising strategies given the impact of PPC on postoperative mortality. Our study has provided evidence of the magnitude of the problem in general surgical populations and the possibility of easily and reliably identifying patients at greater risk of PPC.

Jaume Canet, M.D., Ph.D.,* Lluís Gallart, M.D., Ph.D., Carmen Gomar, M.D., Ph.D., Guillem Paluzie, M.D., Jordi Vallès, M.D., Jordi Castillo, M.D., Sergi Sabaté, M.D., Ph.D., Valentín Mazo, M.D., Joaquín Sanchis, M.D., Ph.D. *Hospital Universitari Germans Trias i Pujol, Barcelona, Spain. jcanet.germanstrias@gencat.cat

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Halogenated Anesthetics and Intensive Care Unit Sedation: A Note of Caution

To the Editor:

I read with interest the article by Sackey *et al.*¹ and accompanying editorial² discussing the use of volatile anesthetics for sedation in the intensive care unit. Although the points regarding tailoring sedation to individual needs are accurate, there is a developing body of literature that suggests prolonged exposure to volatile anesthetics is unsafe, and I believe that Payen understates the case in the editorial.²

It is clear that volatile anesthetics (and all *N*-methyl-D-aspartate receptor antagonists) cause widespread neurode-

This letter was sent to the author of the referenced Editorial, who felt that a reply was not necessary.