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β-Lactam Allergy in the Operating Theater: Reply

In Reply:

We have read with interest the letters from Meikle and Grant and from Vorobeichik *et al*. regarding our article,[1] to which we would like to respond.

First, we would like to point out that we drafted our paper for the most common clinical situation, being that the anesthesiologist is confronted with a patient labeled as having a suspected β-lactam allergy. This is often called penicillin allergy because it is the best known antibiotic to the lay person, but may well be a different antibiotic, even cefazolin itself. Physicians then tend to stay on the safe side and avoid all β-lactams in that situation. Our recommendations should encourage them to wisely choose an alternative that avoids overuse of agents that increase serious hospital infections. The referral to an allergist depends on the hospital of course, and should, as we state in our recommendation, not lead to a delay of surgery and cefazolin is often a safe alternative.

The point that Meikle and Grant make is that in patients with proven penicillin allergy, cefazolin can be administered safely. We did not specifically address this in our review, but we agree that in most cases this is true, if only because serious allergic reactions are very rare. We do not completely agree with "only avoiding cefazolin in patients with severe non-Immunoglobulin-E–mediated hypersensitivity reactions," because even though the R1 side chain is the most common cause of cross-reactivity, one cannot exclude cross-reactivity attributable to the β-lactam ring. As such, we would also include severe anaphylaxis as a contraindication for the use of cefazolin in patients with proven penicillin allergy, because this is associated with high morbidity and mortality. The ideal pathway would be to test possible safe alternatives first, but this decision depends on local protocols and may differ per patient.

In addition, we disagree with Vorobeichik *et al*. that a previous Stevens–Johnson syndrome or toxic epidermal necrolysis may be confused with a mild skin rash. Although initial symptoms may resemble a mild skin rash, the course and severity of the disease are overtly different.[2] In addition, we highlight the updated classifications of drug allergies.[3] Moreover, the authors state that it is confusing that we recommend to avoid β-lactams with proven or suspected severe allergic reactions, but also have referred to table 2 in our article,[1] showing the R-side chain cross-reactivity. However, as we clearly state in figure 1 in the article,[1] table 2 is to be used in case of a suspected mild reaction to choose an alternative antibiotic. We do not agree with the authors that allergy testing is of limited use in the context of surgical prophylaxis. Referral to an allergist, with or without testing, can prove essential in diagnosing or delabeling a suspected allergy.[4] However, we do agree that implementation of referral and testing is dependent on the country, the center, and resources available.

Finally, referring to Blumenthal *et al*.,[4] both Meikle and Grant and Vorobeichik *et al*. state that the administration of alternative antibiotics is associated with a 50% increased odds of developing a surgical site infection.[4] Although the study by Blumenthal *et al*. concerned a retrospective multivariate analysis of the risk for surgical site infections in patients labeled as being allergic to penicillin, and does not report on the efficacy of clindamycin and vancomycin as compared with cefazolin, we do agree that these are inferior to cefazolin in preventing surgical site infections. The study also supports our recommendation of proper evaluation of all patients undergoing surgery labeled as allergic to penicillin.

Competing Interests

The authors declare no competing interests.

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Respiratory Muscle Effort during Weaning: Comment

To the Editor:

We read with interest the article by Doorduin *et al.*[1] regarding weaning failure. The hypothesis was that expiratory muscle effort is higher in weaning failure patients compared to successfully weaned patients. They studied 20 patients of whom 9 had weaning failure. They found that neuromechanical efficiency of diaphragm was lower both during pressure support ventilation and during spontaneous breathing trial in the weaning failure group, despite higher neural respiratory drive. We are curious about the mechanism and possible confounders.

First, the clinical characteristics of the weaning failure group appear different. The weaning failure group had more inspiratory muscle problems at the beginning of the spontaneous breathing trial. Specifically, 56% of the weaning failure but 36% of the weaning success group received mechanical ventilation longer than 2 weeks, suggesting that the failure group had more frequent diaphragm and inspiratory muscle weakness problems.[2] Studies report that diaphragm thickness progressively decreases during mechanical ventilation, and ventilator-induced diaphragm atrophy develops in patients with prolonged mechanical ventilation. In addition, 45% of the weaning success group but 67% of the weaning failure group had cardiac arrest or surgery. Cardiac surgery frequently causes diaphragm weakness.[3] Additionally, patients with more cardiac problems might have developed pulmonary edema during weaning, and this may cause increased inspiratory muscle load. Can these characteristics explain higher neural respiratory drive but lower neuromechanical efficiency of the diaphragm in the failure group? Some previous studies suggest that abdominal muscles are recruited when the load on inspiratory muscles is increased.[4] Second, the weaning failure group had higher $Paco_2$ levels at the end of the trial. This may be another reason for expiratory muscle recruitment in the weaning failure group. Some studies report that expiratory muscles can be recruited when respiratory demand increases under conditions such as hypercapnia, hypoxia, or exercise.[5] While expiratory activity of the diaphragm was similar during the end of the spontaneous breathing trial in both groups, expiratory gastric pressure increased in the weaning failure group compared to the success group. Contribution of the expiratory muscles to the total pressure–time product significantly increased from 13 to 24% at the end of the spontaneus breathing trial in the weaning failure group but did not change in the weaning success group. Taken together, these findings suggest that increased expiratory muscle recruitment happened in the weaning failure group as a result, not a reason. In conclusion, further information is necessary to understand the exact role of expiratory muscles in weaning failure.

Competing Interests

The authors declare no competing interests.

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