Tracheal intubation in patients with odontogenous abscesses: plans and more plans

Reply from the authors

Editor—We appreciate the chance to respond to the letter of Cassellos and Ball in response to our paper.1 The authors are concerned that our approach may not be generally applicable for patients with this dangerous condition.

We agree with the authors’ concern, but it was at no point our intention to present a general plan for all patients with this condition. Our intention was to compare two devices, which might be used for intubation in this critical situation and possibly improve future management of this condition.

We excluded, as pointed out in our Methods section, patients with a mouth opening of < 1.4 cm and patients with a reduced apnoea tolerance. Patients with odontogenous abscesses can present conditions for tracheal intubation that vary from very easy and unproblematic to almost impossible. Therefore, as we pointed out in our Discussion section, all these cases require a thorough planning before induction as presented by Darshane and colleagues2 in their ‘responsive contingency planning’.

Based on the history of the patient, clinical examination, and information from the responsible surgeon about spread and location of the respective abscess, the planning has to go from plan A to B or C or even further down the alphabet. For example, patients with a mouth opening of < 1.4 cm would be intubated directly under local anaesthesia and pre-served spontaneous ventilation. Moreover, it depends also on the local resources and experience. In ‘advanced’ cases, we have an experienced surgeon from the department of oral and cranio-maxillofacial surgeon in the room for emergency intervention. The higher the risk of the patient, the more we have to plan and find individualized plans and solutions. In selected cases, part of the plan might be to place prophylactically a percutaneous transtracheal catheter. However, we do not suggest doing this for every odentogenous abscess and we should not neglect complications of this technique and the difficulties we might have in some patients (with distorted anatomy) in placing such a catheter.

It is also true that we have to make a thorough plan for extubation, but this was not the focus of our study.

Overall, we can agree to the conclusion of Cassel and Ball that videolaryngoscopy may be safe and effective in this challenging group of patients in the context of a robust, reliable airway management strategy.

Declaration of interest

None declared.

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End-expiratory occlusion test: please use the appropriate tools!

Editor—We would like to comment on the article ‘End-expiratory occlusion manoeuvre does not accurately predict fluid responsiveness in the operating theatre’ published by Guinot and colleagues.1 In this study, the authors carry two main messages. The first is that the changes in stroke volume, measured by the CardioQ device during an end-expiratory occlusion (EEO) test, cannot predict fluid responsiveness, as assessed by the same device. The second message is that the changes in end-tidal carbon dioxide (ETCO₂) during an EEO test do not predict fluid responsiveness either. These two messages are wrong for methodological and conceptual reasons.

Concerning the changes in stroke volume measured by oesophageal Doppler during the EEO test, the authors missed the physiological point that the CardioQ device is unable to correctly track the changes in stroke volume induced either by EEO or by volume expansion. Indeed, this device does not measure the changes in aortic diameter that physiologically accompany the changes in arterial pressure. Neglecting these changes, the device underestimates the changes in cardiac output resulting from changes in preload. This has been clearly demonstrated.2 This issue might have particularly influenced the findings in this study, given the changes in arterial pressure observed during EEO and after volume expansion.

Note that eventually, the EEO-induced changes in stroke volume predicted fluid responsiveness with an AUC under the ROC curve of 0.78 (95% confidence interval: 0.63–0.89). In fact, this result should be considered as fair (as declared by the authors themselves in the Results section, by the way), considering the methodological drawbacks of the test.

More importantly, the authors attempted to test the reliability of the EEO-induced changes in ETCO₂. This concept does not make sense to us. Changes in ETCO₂ can reflect the changes in cardiac output only if the ventilation is stable. Of course, interrupting mechanical ventilation for 15 s stops carbon dioxide...
removal and induces a strong upstroke in $e_{CO_2}$ when ventilation is restarted. So, the relatively small changes in cardiac output that could occur in the same time will not be detected.

Finally, this raises a major methodological question. As the methods are described, it seems that the authors measured $e_{CO_2}$ not after restarting ventilation, but at the end of EEO. It is hard to understand how it is possible to measure $e_{CO_2}$ on the exhaled gas... during an occlusion of the respiratory circuit. Overall, contradicting previous publications should require to use accurate methods and to construct one’s reasoning on the most obvious physiological basis.

**Declaration of interest**

X.M. and J.-L.T. are members of the Medical Advisory Board of Pulsion Medical Systems.

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**Clinical practice**

**Reply from the authors**

Editor—We would like to thank Prof. Monnet for his critical comments that raised two questions regarding our methodological construction. We wished to evaluate end-expiratory occlusion (EEO) in a certain population undergoing surgery. For this purpose, we evaluated stroke volume (SV) and cardiac output (CO) with a device that has been used and validated in this setting. Additionally, we wished to evaluate variations of $e_{CO_2}$ because $e_{CO_2}$ is used every day and could be an interesting tool.

First, we used oesophageal Doppler (CardioQ) to assess CO, SV, and their changes during EEO manoeuvre and fluid expansion. We agree that CardioQ does not measure aortic diameter and its ability to measure CO can be questioned; we have already discussed this limitation in our paper.1 Despite these concerns, several studies have demonstrated that CardioQ provides good ability to track changes in SV,2–4 and can be used to improve outcomes in the operating theatre.5 6 Each cardiac output device has its limits. Given that aortic diameter varies with aortic pressure, Prof. Monnet demonstrated that aortic blood flow measurement could be influenced by this variable.7 We would like to note that oesophageal Doppler used by Monnet and colleagues may not be equivalent to these used in our study.8 Prof. Monnet used Hemosonic 100 (Arrow International, Reading, PA, USA) that may have some inaccuracy of aortic diameter evaluation, which may in turn affect the accuracy of measurement of SV.9 In addition, they did not compare the variations of aortic blood flow measured by Hemosonic to those obtained with a gold standard device, such as thermodilution. Evaluation of CO by CardioQ is based on a nomogram created by calibration of left ventricular SV as measured by the pulmonary artery catheter against descending aortic blood flow velocity and stroke distance as measured by CardioQ. Thus, its nomogram provides a calibration factor to translate the descending aortic blood flow velocity to total CO over a wide range of patient conditions.10 Some clinical situations (such as acute circulatory failure, sepsis, epidural analgesia) may change the relation between aortic diameter and aortic pressure, and percentage split of flow between the upper body and lower body.11 In contrast to the intensive care unit population, none of our patients experienced these events. Regardless of the limitations discussed above, trend monitoring of SV should theoretically be possible as long as conditions of validation remain unaltered. In our study, these conditions remained constant; moreover, none of our patients suffered from severe arterial hypotension and/or were treated with vasoactive drugs during the study period. For these reasons, we believe that CardioQ was able to measure and track changes in SV during the different steps. To note, an abstract evaluating EEO manoeuvre with ODM had similar results.12

The second point raised by Prof. Monnet was the reliability of the EEO-induced changes in $e_{CO_2}$. The rate of increase in arterial dioxide carbon in anaesthetized patients is biphasic with a first linear phase above 3–5 mm Hg min⁻¹.13 Based on this fact, we hypothesized that an apnoea of 15 s could be assimilated to a short time period not sufficient to create a strong increase in $e_{CO_2}$, and that the main changes in $e_{CO_2}$ could be due to changes in SV. As we observed an increase in $e_{CO_2}$ with no significant correlation between changes of SV and $e_{CO_2}$ ($r=0.27$, $P=0.088$), apnoea may have in part increased $e_{CO_2}$. Nevertheless, $e_{CO_2}$ did not track changes of SV for the reason that haemodynamic status in our population was not that of patients with acute circulatory failure.

From a physiological point of view, we wonder if our population did not increase oxygen consumption (VO₂) with the increase in oxygen delivery (DO₂), whereas the population in the intensive care unit or haemodynamically unstable patients do increase VO₂ and $e_{CO_2}$ with an increase in DO₂.14 15 There are relatively old studies that have evaluated the relationship between VO₂, DO₂, and $e_{CO_2}$.16 17 These studies demonstrated that when DO₂ increases, $e_{CO_2}$ and VO₂ increase together to the critical DO₂, then after this point, even large increases in DO₂ are not followed by any increase in $e_{CO_2}$ and VO₂.16 17 We believe that was the reason why we did not observe any changes of $e_{CO_2}$ during fluid expansion, whereas SV did.1

**Declaration of interest**

None declared.