Of course, in many cases, neither the medical/anaesthetic history nor the preoperative examinations raise suspicions for an arrhythmogenic syndrome. A characteristic case is the one reported by Hirata and colleagues, regarding a surgical patient with undiagnosed sick sinus syndrome and normal preoperative cardiac examinations, including a Holter electrocardiogram. The syndrome was unveiled after induction of general anaesthesia and was confirmed a few months after operation by a diagnostic new Holter electrocardiogram.

In patients with unexplained, suspicious intraoperative arrhythmias, even if they resolved without further complications, postoperative 24 h haemodynamic monitoring and further cardiological investigation, although associated with increased costs, would probably be useful in revealing an arrhythmogenic syndrome. If a sudden perioperative death occurs, postmortem investigation and—if indicated—familial genetic screening should be performed. In these cases, the anaesthesiologists may also play a significant role in announcing the death, explaining, informing, and even guiding the family members towards investigations which may be lifesaving for them, if a hereditary syndrome is diagnosed and thus treated early.

**Declaration of interest**

None declared.

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**FIO2 and studies on oxygenation during one-lung ventilation**

Editor—We read with interest the study by Roze and colleagues comparing the effects of two ventilation strategies on oxygenation during one-lung ventilation (OLV).

Although not explicitly stated, the authors seem to have used variable levels of FIO2 across subjects during OLV. However, to study the effects of changes in ventilation strategy (or any other intervention) on oxygenation during OLV, it may not be advisable to vary FIO2 across subjects and present data as Pao2/FIO2. It rather may be helpful to use a constant and high FIO2 in all patients and present data as Pao2.

Why is it problematic to vary FIO2 and present data as Pao2/FIO2? This is because the relationship between Pao2/FIO2 and FIO2 is not linear and may vary considerably with FIO2. The variation would be most apparent in patients with large shunts and ventilation/perfusion abnormalities, pathologies prevalent in the thoracic surgical patient population. A low FIO2 in patients with low ventilation/perfusion ratio may, for example, increase venous admixture. Thus, using variable levels of FIO2 in a patient population with respiratory disease and different shunt fractions may generate excess variation in Pao2/FIO2 values unrelated to the intervention. The cross-over design in this study may have averted gross variation with respect to the intervention but does not rule out excess interindividual variation.

Why is it better to use not only a constant but also a high FIO2 (>0.8) and present data as Pao2? This is because while using high FIO2, even small changes (increase or decrease) in shunt fraction, induced, for example, through the intervention under study, would predictably lead to large changes in Pao2. While using low FIO2, similar changes in shunt fraction may lead to comparatively smaller changes in Pao2, and thus less chances of obtaining statistically significant results. This can be readily appreciated by studying the iso-shunt lines, the graphic interrelationship between Pao2, shunt, and FIO2.

During clinical OLV, however, we too advocate using low FIO2 compatible with sufficient oxygenation.

**Declaration of interest**

None declared.

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**Reply from the authors**

Editor—We thank Karzai and Klein for their interest in our article.3 We totally agree with them regarding the interpretation of the Pao2/FIO2 ratio. It is important to clarify that this