Instead of using a straight combination of high-flow, non-rebreathing 67% N₂O and 33% oxygen to ventilate the patient during the experiment, the authors have chosen to ventilate the lungs with a Servo 900C ventilator equipped with a gas monitor for O₂, N₂, and N₂O. The recording of the respiratory gas fractional concentration depicted in Figure 1 demonstrates exactly the problem of gas mixing and maintaining 33% of the inspired *Fi*O₂ throughout the course of the experiment. During four minutes of gas mixing after the start of N₂O inhalation (between 15 and 19 min), the inspired-expired oxygen concentration differences almost disappeared, and a much higher oxygen concentration was demonstrated at the end of mixing (at 19 min) which indicated that the patient received an oxygen concentration higher than 33%. At the end of gas mixing (at 19 min), inspired and expired oxygen concentration differences started again, but with smaller differences in the beginning, which indicate that the functional residual capacity of the lung has been filled with a higher O₂ concentration similar to the inspired concentration during the mixing. This coincides very well with their statement at the end of their discussion, in which they admitted that there was a small change in *Fi*O₂ immediately after N₂O administration due to a delay in adjusting the oxygen fraction to 0.33 in several patients. Therefore, the FETO₂ and PaO₂ increase after N₂O administration was definitely not related to the N₂O administration, but was related to the higher oxygen supply during the initial mixing period.

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Reference

- 1 Nishikawa K, Kunimoto F, Isa Y, et al. Second gas effect of N₂O on oxygen uptake. Can J Anesth 2000; 47: 506–10.

REPLY:

*The answer to the question raised by Dr. Lin's letter is definitely "no". We actually measured *Fi*O₂ and FETCO₂ concentration every minute after N₂-N₂O exchange. As shown in Figure 2 (data at one and five minutes), we did not observe any statistically significant change in*

**Fi*O₂ compared to the control value (*Fi*O₂, 0.33). As we mentioned in the discussion, ETCO₂ data are a bit variable in each case, however, we never saw such high O₂ concentrations as speculated by Dr. Lin. Needless to say, had we found such high O₂ concentrations, those patients would have been excluded from the analysis.*

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Amniotic fluid embolism

To the Editor:

Sharon Davies concludes that amniotic fluid embolus (AFE) is of uncertain etiology, of variable presentation and without a standardized means of confirming the diagnosis.¹ I suggest a step further: abandon the term altogether since the evidence that amniotic fluid has anything to do with the syndrome of (sometimes) catastrophic collapse that we (sometimes) call AFE is at best tenuous, as Davies has so comprehensively summarized in her review. I have suggested the term "sudden obstetric collapse syndrome" (SOCS) as a more appropriate description, characterised by sudden, severe cardiovascular collapse in a woman who is (or has recently been) pregnant, where no other explanation is considered likely.² This definition reflects what is currently known about the condition, which is not very much, and gets us away from an obsession with amniotic fluid that has as much evidence against it as there is for it.

Steve Yentis MD FRCA
London, UK

References

- 1 Davies S. Amniotic fluid embolus: a review of the literature. Can J Anesth 2001; 48: 88–98.
- 2 Yentis SM. Sudden obstetric collapse syndrome. Int J Obstet Anesth 1999; 8: 296.

REPLY:

I would like to thank Dr. Yentis for his interest in my review article.¹ Others have also suggested that the syndrome should be renamed.² This may be somewhat premature until more is known about this entity. As stated in my article, there is no evidence in the literature for the common misconception that the entrance of amniotic fluid into the maternal circulation is a routine occurrence. We have enough clinical examples and autopsy findings in the literature to show that amniotic fluid elements such as lanugo, vernix and mucin can be found in the systemic circulation in cases of unexpected maternal death with no other pathological findings or diagnosis. These elements are not described in the lungs of pregnant patients dying from other causes. I continue to believe that amniotic fluid embolus does exist, that it is a pathological entity and that clinically it is not an all or nothing phenomena, but a spectrum of disease. When a mother is exposed to amniotic fluid elements, the outcome may well depend on the degree her pathophysiological response as well as the antigenicity of the fluid itself. Dr. Yentis' term "sudden obstetrical collapse syndrome" only