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Variable Effect of Positive End-expiratory Pressure on Oxygenation in Patients with Acute Respiratory Distress Syndrome

To the Editor:—The recent article by Thille *et al.*[1] and the accompanying editorial by Rouby[2] discuss the effects of positive end-expiratory pressure (PEEP) on pulmonary recruitability in patients with adult respiratory distress syndrome (ARDS). Thille *et al.* found that alveolar recruitment was similar in pulmonary and extrapulmonary ARDS and concluded that PEEP levels should not be determined based on cause of ARDS.[1] However, the type of ARDS could not be classified in 37% of the patients in their retrospective study,[1] an important point that is further discussed in Rouby's editorial.[2] In the study by Thille *et al.*, the response to PEEP was assessed by pressure-volume curves only, and the effect of PEEP on oxygenation is not reported.

More than 30 yr ago, we described the variable effect of PEEP on oxygenation in patients with ARDS.[3,4] We found that both the short-term and the long-term effects of PEEP on oxygenation response in patients with sepsis were statistically smaller compared with that in patients without sepsis, and we concluded that ARDS associated with sepsis seems to be the result of a more severe pulmonary insult.[3] In a subgroup of trauma patients, we found again that the improvement in oxygenation after the application of PEEP in patients who developed sepsis was significantly smaller than that of patients without sepsis and without lung contusion.[4]

As imperfect as they are by today's standards, these two very old studies do have relevance to the ongoing discussion about the effectiveness of pulmonary recruitment in ARDS because, in contrast to Thille *et al.*'s findings, they support the view that the effectiveness of PEEP does depend on the etiology of ARDS. It is unfortunate that for too many years we have not made much progress in better defining the various subgroups of ARDS. The clinician at the bedside is still left with one option only, namely, apply PEEP and assess its consequences. In fact, the response to PEEP may help in making the diagnosis.

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References

1. Thille AW, Richard J-C, Maggiore SM, Ranieri VM, Brochard L: Alveolar recruitment in pulmonary and extrapulmonary acute respiratory distress syndrome: Comparison using pressure-volume curve or static compliance. ANESTHESIOLOGY 2007; 106:212–7
2. Rouby JJ: Recruitment in pulmonary and extrapulmonary acute respiratory distress syndrome: The end of am? ANESTHESIOLOGY 2007; 106:203–4
3. Cotev S, Perel A, Katzenelson R, Eimerl D: The effect of PEEP on oxygenating capacity in acute respiratory failure with sepsis. Crit Care Med 1976; 4:186–92
4. Perel A, Olshwang D, Eimerl D, Katzenelson R, Cotev S: The variable effect of PEEP in acute respiratory failure associated with multiple trauma. J Trauma 1978; 18:218–21

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In Reply:—I thank Dr. Perel for his interest in my editorial.[1] He raises the question as to whether the presence of sepsis alters the effect of positive end-expiratory pressure (PEEP) on oxygenating capacity in patients with adult respiratory distress syndrome. Based on his own studies published in the late 1970s,[2,3] he gives a positive answer and argues that such a result supports the concept that the cause of adult respiratory distress syndrome influences the effectiveness of PEEP and contradicts Thille *et al.*'s study.[4] Going all the way through his belief, he proposes to use the effect of PEEP on oxygenating capacity as a diagnostic test to determine whether adult respiratory distress syndrome is septic.

The body of evidence supporting Dr. Perel's statement is small. The retrospective study he refers to[2,3] concerns a very heterogeneous population of 57 hypoxemic patients (23 with multiple trauma, 21 with sepsis, 6 who underwent cardiopulmonary bypass, 4 with left heart failure, 2 with acid aspiration, and 1 with cervical chord injury) and suggests that when severe hypoxemia is associated with or caused by sepsis, the increase in arterial oxygenation after the administration of a small PEEP (5.7 ± 1.2 mH_2O) is less than that in the absence of sepsis. The heterogeneity of the patients studied (adult respiratory distress syndrome and cardiogenic pulmonary edema); the lack of any attempt made to optimize PEEP; and the absence of measurement of cardiac output, cardiac filling pressures, mixed venous oxygen saturation, pulmonary shunt, and alveolar recruitment preclude any analysis of the mechanisms involved in the observed difference. In addition, the second article,[3] which is similar to the first,[2] focuses on the 23 patients of the series, in whom lung injury was the result of traumatic lung contusion. It is well known that the initial phase of severe lung contusion can be characterized by severe hypoxemia, sometimes refractory to conventional means and requiring nonconventional mechanical ventilation.[5] If the patient survives the initial phase, however, there is a rapid and spectacular improvement in arterial oxygenation related to blood clearance from the alveolar space allowing early extubation and spontaneous breathing. If, for extrapulmonary reasons, the patient cannot be extubated, then a second direct insult to the lung rapidly occurs, ventilator-associated pneumonia. In fact, the late deterioration reported by Perel *et al.*[3] has nothing to do with "the cause of adult respiratory distress syndrome" but is related to the impossibility of weaning the patient from mechanical ventilation for extrapulmonary reasons.

Among the most convincing data pleading against the hypothesis that the septic lung recruits less than the nonseptic lung are those recently published by Gattinoni himself, the initiator of the theory that patients with pulmonary adult respiratory distress syndrome caused by lung infection potentially have a much higher recruitable lung capacity than patients with extrapulmonary adult respiratory distress syndrome.[6] Because of the lack of evidence supporting Dr. Perel's assertions, his clinical recommendations should be taken with caution. Unless new convincing data are provided, the non-response to PEEP should not be interpreted by clinicians in charge of patients with adult respiratory distress syndrome as an indirect sign of lung injury associated with or caused by sepsis.

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References

1. Rouby J-J. Recruitment in pulmonary and extrapulmonary acute respiratory distress syndrome: The end of a myth? Anesthesiology 2007;106:203–4
2. Cotev S, Perel A, Katzenelson R, Eimerl D: The effect of PEEP on oxygenating capacity in acute respiratory failure with sepsis. Crit Care Med 1976; 4:186–92
3. Perel A, Olshwang D, Eimerl D, Katzenelson R, Cotev S: The variable effect of PEEP in acute respiratory failure associated with multiple trauma. J Trauma 1978; 18:218–21
4. Thille AW, Richard JC, Maggiore SM, Ranieri VM, Brochard L: Alveolar recruitment in pulmonary and extrapulmonary acute respiratory distress syndrome: Comparison using pressure-volume curve or static compliance. Anesthesiology 2007; 106:212–7
5. Riou B, Zaier K, Kalfon P, Puybasset L, Coriat P, Rouby JJ: High-frequency jet ventilation in life-threatening bilateral pulmonary contusion. Anesthesiology 2001; 94:927–30
6. Gattinoni L, Caironi P, Cressoni M, Chiumello D, Ranieri VM, Quintel M, Russo S, Patroniti N, Cornejo R, Bugedo G: Lung recruitment in patients with the acute respiratory distress syndrome. N Engl J Med 2006; 354:1775–86

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In Reply:—We thank Dr. Perel for his interest in our article.[1] He raises the issue of the influence of sepsis on positive end-expiratory pressure (PEEP) response in terms of oxygenation in patients with adult respiratory distress syndrome. Based on his own studies published in the late 1970s,[2,3] he indicates that their result supports the concept that the cause of adult respiratory distress syndrome, or at least the presence of sepsis, influences the effectiveness of PEEP. He also suggests that the effect of PEEP on oxygenation may be part of a diagnostic test to determine whether adult respiratory distress syndrome is septic.

In our study, we did not directly address the points raised and try to reproduce these effects, for several reasons. First, Dr. Perel used small levels of PEEP with no standardization, making it difficult to reproduce these results. Second, we do not have systematic measurements of oxygenation at every level tested or assessment of the precise criteria for defining "sepsis" exactly at the time when the curves were performed. We do not think, however, that this is the right approach for at least two reasons. Based on the last 15 yr literature on PEEP, it has become clear that if PEEP is important, it is not because of its effects on oxygenation, but mainly because of its effects on lung recruitment and by making ventilation more homogeneously distributed.[4] Arterial oxygenation has many drawbacks as a pure indicator of recruitment,[5,6] and we have focused our more recent research on recruitment rather than oxygenation, mainly for this reason. The differences Dr. Perel described many years ago could be entirely explained by hemodynamic reasons independent of recruitment and could thus be misleading for clinicians. In addition, what we have shown is that there is no *a priori* difference in terms of PEEP-induced recruitment whatever the origin of adult respiratory distress syndrome.[1] In other words, the clinician has to look for the individual effect of PEEP and not base the administered therapy on *a priori* criteria. We think this is an important message for today's clinicians.

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References

1. Thille AW, Richard JC, Maggiore SM, Ranieri VM, Brochard L: Alveolar recruitment in pulmonary and extrapulmonary acute respiratory distress syndrome: Comparison using pressure-volume curve or static compliance. Anesthesiology 2007; 106:212–7
2. Cotev S, Perel A, Katzenelson R, Eimerl D: The effect of PEEP on oxygenating capacity in acute respiratory failure with sepsis. Crit Care Med 1976; 4:186–92
3. Perel A, Olshwang D, Eimerl D, Katzenelson R, Cotev S: The variable effect of PEEP in acute respiratory failure associated with multiple trauma. J Trauma 1978; 18:218–21
4. Grasso S, Fanelli V, Cafarelli A, Anaclerio R, Amabile M, Ancona G, Fiore T: Effects of high *versus* low positive end-expiratory pressures in acute respiratory distress syndrome. Am J Respir Crit Care Med 2005; 171:1002–8
5. Aboab J, Louis B, Jonson B, Brochard L: Relation between PaO_2/FIO_2 ratio and FIO_2: A mathematical description. Intensive Care Med 2006; 32:1494–7
6. Takala J: Hypoxemia due to increased venous admixture: Influence of cardiac output on oxygenation. Intensive Care Med 2007; 33:908–11

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John Quincy Adams Ward Did Not Create the Ether Monument

To the Editor:—We thoroughly enjoyed reading "A Tale of Two Paintings." However, the article presents one misleading—although minor—detail; it states that sculptor John Quincy Adams Ward (1830–1910) "created the Ether Monument."[1] Although Adams Ward sculpted the Good Samaritan group atop the Monument and the four bas-reliefs on the base, he did not design the Monument. Henry Van Brunt (1836-1903), of the Ware and Van Brunt architectural firm (Boston, MA), is considered the Monument's architect; the letter he wrote to Adams Ward, asking him to sculpt these pieces, included his early sketch of the Monument.[2]

There are, however, other individuals who might have contributed to the Ether Monument's design. William Ware (1832–1915), Van Brunt's partner, wrote in the 1866 Harvard Yearbook that his activities included the design of the Ether Monument. It is not certain whether he was referring to himself personally or to his firm with Van Brunt. John La Farge (1835–1910), a painter and friend of Van Brunt, may also have had a hand in designing the Ether Monument. In the 1970s, La Farge's grandson encountered sketches resembling the Monument and, despite some slight differences between the drawing and the Monument, surmised that his grandfather might have influenced the memorial's design.[2]

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References

1. Desai SP, Desai MA, Maddi R, Battit GE: A tale of two paintings: Depictions of the first public demonstration of ether anesthesia. Anesthesiology 2007; 106: 1046–50
2. Ortega RA: Written in granite: An illustrated history of the ether monument. Boston, Plexus Management, 2006

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In Reply:—On behalf of all the authors, I wish to thank Dr. Ortega and Ms. Hafez for this historical clarification regarding the design of the Ether Monument.

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The Use of Simulation Education in Competency Assessment: More Questions than Answers

To the Editor:—With great interest many anesthesia educators have read three manuscripts in previous issues of ANESTHESIOLOGY. Morgan, a well-known and respected researcher in simulation education, and her coauthors presented a thoughtful description of the use of simulation in the evaluation of teamwork in obstetrical practice.[1] However, in light of their conclusions that the "study does not support the use of Human Factors Rating Scale for assessment of obstetrical teams" and recommended limited use of the Global Rating Scale, taken along with Murray and Enarson's reflective editorial[2] on the difficulties of teamwork and communication skills assessment, I fear that some anesthesia educators might be tempted to throw the newly born discipline of "simulation based assessment" out with the proverbial bathwater.

Morgan *et al.*'s investigation[1] raises several issues that need to be addressed in light of our urgent need to develop authentic teaching and assessment of clinical competency in anesthesiology.[3] The most pressing is our need for reliable and valid performance assessment tools used in anesthesia education, training, and practice.

Morgan *et al.* found the Human Factors Rating Scale and Global Rating Scale assessment tools of limited reliability in the obstetrical setting; however, they do not further examine the sources of variance in the reliability other than from the raters themselves. Although the number of raters, the number of items, and the occasions of testing seem to be sufficient, we have no definitive analysis of this. Classic Test Theory and the use of interrater reliability and inter- and intraclass correlations for diagnosis of measurement error does not provide a view of the relative importance or interactions of these and other variances sources. Modern Test Theory, specifically Generalizability Theory, provides an analysis of multiple sources of variance and determination of optimal sampling not only for raters but also for subjects, items, and occasions of testing.[4–6] In this investigation,[1] the nonconcordance of the correlations of both Human Factors Rating Scale and Global Rating Scale suggest that something else is going on here. It could be the result of nonparallel scenarios, lack of rater training, or (more significantly), faulty construct validity of the Human Factors Rating Scale and Global Rating Scale for the anesthesia Crisis Resource Management trait. However, we do not know from this report.

On reviewing of the original development of the Human Factors Rating Scale[7] and its 2000 revision, we still do not have formal psychometric analysis of its construct validity and factor status "due to limited sample size."[8] Although its authors claim that the items cluster around "team roles, authority/command structure, stress recognition and organizational climate,"[8] these highly complex behaviors warrant more formal factor analysis before general use. We as anesthesiologists would not use a new clinical test without knowing the measure of its specificity and sensitivity; we should be equally rigorous with the degree of validity and reliability in high-stakes testing and use of resource-intense instruction. Modern Test Theory offers many advantages necessary for the authentic assessment of complex cognitive, technical, and behaviors skills in simulation-based education and performance assessment.[9]

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References

1. Morgan PJ, Pittini R, Regehr G, Marrs C, Haley MF: Evaluating teamwork in a simulated obstetric environment. ANESTHESIOLOGY 2007; 106:907–15
2. Murray D, Enarson C: Communication and teamwork: Essential to learn but difficult to measure. ANESTHESIOLOGY 2007; 106:895–6
3. Tetzlaff JE: Assessment of competency in anesthesiology. ANESTHESIOLOGY 2007; 106:812–25
4. Shavelson RJ WN: Generalizability theory: A primer. Newberry Park, SAGE Publications, 1911
5. Handbook of Complementary Methods in Education Research. Washington, DC, American Educational Research Association, 2006
6. Boulet JR, Murray D, Kras J, Woodhouse J, McAllister J, Ziv A: Reliability and validity of a simulation-based acute care skills assessment for medical students and residents. ANESTHESIOLOGY 2003; 99:1270–80
7. Helmreich RL MA, Sherman PJ, Gregoric SE, Wiener EL. The Flight Management Attitudes Questionnaire (FMAQ), technical report 93-4. Austin, NASA/UT/FAA, 1993
8. Sexton J. Operating Room Management Attitudes Questionnaire, technical report 00-2. Austin, University of Texas at Austin, 2000:2
9. Colliver JA: Effect-size measures and research in developmental and behavioral pediatrics. J Dev Behav Pediatr 2007; 28:145–50

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In Reply:—We would like to thank Dr. Edler for her interest in our editorial.[1] In an innovative study that measured teamwork during simulated obstetric emergencies, Morgan *et al.* found that the team scores were not reliable.[2] The investigators attributed the limited reliability to the use of the Human Factors Rating Scale. Dr. Edler correctly indicates that the limited reliability could be the result of several factors and that a generalizablity analysis that partitioned the variance associated with scoring method, raters, and scenario would help to clarify the findings. Regardless of the cause of the limited reliability, Morgan *et al.*'s main conclusion would still stand.

"Simulation education" or, perhaps more appropriately, simulation-based assessment, has stimulated interest in interpreting participant or team scores. The reliability and validity of scores obtained during a simulation (or any performance assessment) depend on the event and environment's authenticity, how effectively the scoring instrument captures the skills of interest, and whether the raters consistently observe and record the performance. Morgan *et al.*'s experimental design offers an important first step in evaluating teamwork in that it includes high-fidelity simulated obstetric emergencies that can be managed by a multidisciplinary team. Hopefully, rather than discouraging educators, the chal-

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lenges of developing assessment instruments that can be used to provide valid and reliable team scores will inspire additional investigations. This research would help to establish the role of simulation-based assessment as a method to investigate and evaluate team performance.

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References

1. Murray D, Enarson C: Communication and teamwork: Essential to learn but difficult to measure. ANESTHESIOLOGY 2007; 106:895–6
2. Morgan PJ, Pittini R, Regehr G, Marrs C, Haley MF: Evaluating teamwork in a simulated obstetric environment. ANESTHESIOLOGY 2007; 106:907–15

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In Reply:—We would like to thank Dr. Edler for her interest in our article.[1] First and foremost, in no way are we suggesting that simulation education be discarded, but, as Dr. Edler comments, there is a pressing need to ensure that we are assessing its impact using valid, reliable tools.

We also agree with Dr. Edler that generalizability is the way of the future and in fact is the analytic basis of our current ongoing study, designed to examine the psychometric properties of a newly developed obstetric team performance tool.

Our study was based on a sample size and using a design that would make the use of multifaceted generalizability analyses complicated at best. In fact, a generalizability analysis would likely be unstable because individuals were partially nested within team, and team was nested within scenario, and the number of actual performances was small for a generalizability analysis, which is why, ultimately, we adopted the analytic techniques we did for the study in question.

However, rater was fully crossed with all the dimensions that Dr. Elder described as other potential sources of variance, and it was the interrater reliability of the Human Factors Rating Scale that was a problem. Although we agree that other facets might add further error, there is no reason to believe that the interrater reliability would be better understood through the partitioning of other variances. With regard to the Human Factors Rating Scale, as Dr. Edler suggests, undoubtedly "something else is going on here." However, based on our findings in the study, we have become substantially less interested in figuring out that "something else" than we might otherwise have been.

Instead, we decided that it will ultimately be more profitable to start from scratch, developing a tool that is generated out of the practice and experience of healthcare teams, rather than to spend our resources investigating a better understanding of what we think will ultimately prove to be the wrong tool.

Although we have independently come to the same opinions as Dr. Edler regarding the types of studies needed, based on our preliminary data, we decided that we will be looking to tools other than the Human Factors Rating Scale as the subject of these more elaborate generalizability analyses.

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Reference

1. Morgan PJ, Pittini R, Regehr G, Marrs C, Haley MF: Evaluating teamwork in a simulated obstetric environment. ANESTHESIOLOGY 2007; 106:907–15

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Hyperoxia-induced Decrease in Organ Blood Flow

To the Editor:—In their article regarding the effects of hyperoxia on tissue perfusion, Forkner *et al.*[1] conclusively stated that administration of high oxygen concentration augments tissue oxygenation and produces no adverse effects on tissue oxygenation.

Forkner *et al.* tried to convince readers that Iscoe *et al.*'s position[2] that the hyperventilation induced by hyperoxia as a result of the Haldane effect would lead to arterial hypocapnia and, hence, vasoconstriction in certain vascular beds, including those in brain, is not correct. Based on findings of very old studies, Forkner *et al.* also argued that during hyperoxia blood flow is not reduced enough to offset the higher oxygen content, and oxygen delivery is enhanced. This argument is not correct. A previous human study has shown that the magnitude of a decrease in cerebral blood flow is much more profound than that of an increase in arterial oxygen content.[3] In that study, for example, the arterial oxygen content was 18 ml/dl and cerebral blood flow was 54 ml $\cdot$ 100 $g^{-1} \cdot min^{-1}$ (the cerebral oxygen delivery was 9.7 ml $\cdot$ 100 $g^{-1} \cdot min^{-1}$) at baseline, and after breathing 100% oxygen at 1 atmosphere absolute, the arterial oxygen content increased by 13.4% but the cerebral blood flow decreased by 33%, so the cerebral oxygen delivery decreased to 7.3 ml $\cdot$ 100 $g^{-1} \cdot min^{-1}$ (20.7 ml/dl oxygen content $\times$ 36 ml $\cdot$ 100 $g^{-1} \cdot min^{-1}$), a 25% decrease in cerebral oxygen delivery. In addition, the partial pressure of arterial carbon dioxide decreased by 3 Torr with the administration of 100% oxygen. When hypocapnia was corrected by adding carbon dioxide to 100% oxygen, decreased cerebral blood flow was restored by only 8%. Although the role of hyperoxia-induced hypocapnia on decreases in cerebral blood flow may not be great, the independent cerebral vasoconstrictive effect of hyperoxia is too great in magnitude to be ignored. A hyperoxia-induced decrease in cerebral blood flow would cause a rightward shift of cerebral blood flow-perfusion pressure relationship line, so that cerebral blood flow would be much lower at a given perfusion pressure.

By employing laser Doppler velocimetry for the measurement of retinal blood velocity and fundus imaging with the Zeiss retinal vessel analyzer for the assessment of retinal vessel diameter, another previous human study[4] showed that hyperoxia by breathing greater than 92% oxygen decreases retinal blood velocity and retinal blood flow by 60% each and decreases retinal vessel diameter by 15%, suggesting that the decrease in retinal blood flow is mainly the result of decreased cerebral blood flow rather than the retinal vessel vasoconstriction. The decrease in retinal blood flow during hyperoxia could contribute to ischemic retinopathy when combined with increased intraocular pressure[5] during prone spine surgery.

A clinical study has shown that breathing 100% oxygen reduces coronary blood flow velocity by 20% and increases coronary vascular resistance by 23% in patients with coronary artery disease and that vitamin C reverses this effect, suggesting that these changes are medi-

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ated by an oxidative stressor acting on the coronary microcirculation.[6] A hyperoxia-induced decrease in coronary microcirculatory blood flow in patients with coronary artery stenosis can be detrimental.

To determine whether the routine use of high inspired oxygen concentration during the perioperative period alters the incidence of surgical site infection in a general surgical population, a double-blind, randomized controlled trial was conducted.[7] The incidence of surgical site infection was significantly higher (25%) in the group receiving inspired oxygen concentration of 80% than in the group with inspired oxygen concentration of 35% (11.3%). This is further evidence that does not support the argument of Forkner *et al.*

Supplemental oxygen benefits patients who would otherwise suffer hypoxia while breathing room air. However, in this situation, supplemental oxygen would not induce hyperoxia. Forkner *et al.* made no distinction between supplemental oxygenation and the administration of high oxygen concentration.

The effect of hyperoxia-induced hypocapnia on decreases in cerebral blood flow or other organ flow may not be great and is unlikely to be of clinical significance. However, the independent vasoconstricitive effect of hyperoxia is too great in magnitude to be ignored. One cannot say that hyperoxia is harmless.

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References

1. Forkner IF, Piantadosi CA, Scafetta N, Moon RE: Hyperoxia-induced tissue hypoxia: A danger? ANESTHESIOLOGY 2007; 106:1051–5
2. Iscoe S, Fisher JA: Hyperoxia-induced hypocapnia: An underappreciated risk. Chest 2005; 128:430–3
3. Floyd TF, Clark JM, Gelfand R, Detre JA, Ratcliffe S, Guvakov D, Lambertsen CJ, Eckenhoff RG: Independent cerebral vasoconstrictive effects of hyperoxia and accompanying arterial hypocapnia at 1 ATA. J Appl Physiol 2003; 95:2453–61
4. Luksch A, Garhöfer G, Imhof A, Polak K, Polska E, Dorner GT, Anzenhofer S, Wolzt M, Schmetterer L: Effect of inhalation of different mixtures of O_2 and CO_2 on retinal blood flow. Br J Ophthalmol 2002; 86:1143–7
5. Cheng MA, Todorov A, Tempelhoff R, McHugh T, Crowder CM, Lauryssen C: The effect of prone positioning on intraocular pressure in anesthetized patients. ANESTHESIOLOGY 2001; 95:1351–5
6. McNulty PH, Robertson BJ, Tulli MA, Hess J, Harach LA, Scott S, Sinoway LI: Effect of hyperoxia and vitamin C on coronary blood blow in patients with ischemic heart disease. J Appl Physiol 2007; 102:2040–5
7. Pryor KO, Fahey IIITJ Lien CA Goldstein PA: Surgical site infection and the routine use of perioperative hyperoxia in a general surgical population: A randomized controlled trial. JAMA 2004; 291:79–87

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In Reply:—We thank Dr. Her for his comments on our article.[1] He notes that a previous human study by Floyd *et al.* demonstrated that the magnitude of the decrease in cerebral blood flow (CBF) induced by 100% oxygen administration is more profound than that of the increase in arterial oxygen content,[2] which seems to support the notion that hyperoxia can induce tissue hypoxia. This study, which used arterial spin labeled-perfusion magnetic resonance imaging, reported a 29-33% decrease in CBF during 100% oxygen breathing. If the 30% reduction were accurate, then Dr. Her may have a point. However, this reduction in CBF is significantly greater than what has been observed in other studies, in which the reduction has been on the order of 5–15%.[3–5] Unfortunately the investigators in the Floyd *et al.* study made unwarranted assumptions. For instance, they assumed that T1 for blood was the same during air and oxygen breathing despite published data to the contrary.[6] This component of the lumped constant in their calculation of CBF leads to a 36% difference based on the T1 of blood with air *versus* oxygen breathing. Further, they assumed T1 in tissue to be similar to that in blood and suggest that violations of this assumption would cause only a small error. This is clearly not the case and is the reason for the overestimation of changes in CBF found in their study. In a recent article by Bulte *et al.*,[5] in which 100% oxygen reduced CBF less than 10%, the changes in relaxation times of blood and tissue with increased Fio_2 are confounders of the arterial spin labeled technique, and that failure to accurately account for them when calculating perfusion will lead to gross overestimation of hyperoxia-induced blood flow changes.

We agree with Dr. Her that acute hypocapnia can be associated with markers of ischemia, such as impaired psychomotor performance, at least in the arterial Pco_2 range 20–25 mmHg.[7] However, our original manuscript included data showing that during spontaneous breathing, the tendency of 100% oxygen administration to cause hypocapnia is either very small or does not exist.[1] Tissue Po_2 regulates blood flow such that, although oxygen administration does induce vasoconstriction, there is a monotonically *increasing* relationship between arterial and tissue Po_2.[8–10] This has also been observed in the retina.[11] In a pig study, the administration of 100% oxygen reduced retinal blood flow by 62% but increased periarteriolar and intervascular Po_2.[12] It is therefore unlikely that hyperoxia could contribute to retinal ischemia induced by an increase in intraocular pressure, and indeed there is direct evidence to the contrary.[13]

Oxygen administration also reduces coronary blood flow; however, in the study cited by Dr. Her,[14] there is no evidence that the reduced coronary blood flow induced either ischemia or myocardial hypoxia.

With regard to Dr. Her's comment on oxygen and postoperative wound infections, evidence suggests an inverse relationship between tissue Po_2 and infection rate,[15] and the bulk of evidence supports the use of supplemental perioperative oxygen to reduce wound infection rate.[16,17] The study by Pryor and colleagues cited by Dr. Her,[18] which failed to demonstrate a beneficial effect, has been criticized on methodological grounds.[19,20]

In summary, peripheral blood flow is regulated to maintain tissue oxygenation in the face of alterations in oxygen delivery. There is no evidence that the autoregulatory decrease in tissue blood flow during hypoxia induces tissue ischemia or hypoxia.

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References

1. Forkner IF, Piantadosi CA, Scafetta N, Moon RE: Hyperoxia-induced tissue hypoxia: A danger? ANESTHESIOLOGY 2007; 106:1051–5
2. Floyd TF, Clark JM, Gelfand R, Detre JA, Ratcliffe S, Guvakov D, Lambertsen CJ, Eckenhoff RG: Independent cerebral vasoconstrictive effects of hyperoxia and accompanying arterial hypocapnia at 1 ATA. J Appl Physiol 2003; 95:2453–61
3. Kety SS, Schmidt CF: The effects of altered arterial tensions of carbon dioxide and oxygen on cerebral blood flow and cerebral oxygen consumption of normal young men. J Clin Invest 1947; 27:484–92
4. Kolbitsch C, Lorenz IH, Hormann C, Hinteregger M, Lockinger A, Moser PL, Kremser C, Schocke M, Felber S, Pfeiffer KP, Benzer A: The influence of hyperoxia on regional cerebral blood flow (rCBF), regional cerebral blood volume (rCBV) and cerebral blood flow velocity in the middle cerebral artery (CBFVMCA) in human volunteers. Magn Reson Imaging 2002; 20:535–41
5. Bulte DP, Chiarelli PA, Wise RG, Jezzard P: Cerebral perfusion response to hyperoxia. J Cereb Blood Flow Metab 2007; 27:69–75
6. Tadamura E, Hatabu H, Li W, Prasad PV, Edelman RR: Effect of oxygen inhalation on relaxation times in various tissues. J Magn Reson Imaging 1997; 7:220–5

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7. Balke B, Ellis JP Jr, Wells JG: Adaptive responses to hyperventilation. J Appl Physiol 1958; 12:269–77
8. Whalen WJ, Nair P: Skeletal muscle PO_2: effect of inhaled and topically applied O_2 and CO_2. Am J Physiol 1970; 218:973–80
9. Klitzman B, Damon DN, Gorczynski RJ, Duling BR: Augmented tissue oxygen supply during striated muscle contraction in the hamster: Relative contributions of capillary recruitment, functional dilation, and reduced tissue PO_2. Circ Res 1982; 51:711–21
10. Demchenko IT, Luchakov YI, Moskvin AN, Gutsaeva DR, Allen BW, Thalmann ED, Piantadosi CA: Cerebral blood flow and brain oxygenation in rats breathing oxygen under pressure. J Cereb Blood Flow Metab 2005; 25:1288–300
11. Wangsa-Wirawan ND, Linsenmeier RA: Retinal oxygen: Fundamental and clinical aspects. Arch Ophthalmol 2003; 121:547–57
12. Riva CE, Pournaras CJ, Tsacopoulos M: Regulation of local oxygen tension and blood flow in the inner retina during hyperoxia. J Appl Physiol 1986; 61:592–8
13. Alder VA, Cringle SJ: Intraretinal and preretinal PO_2 response to acutely raised intraocular pressure in cats. Am J Physiol 1989; 256:H1627–34
14. McNulty PH, Robertson BJ, Tulli MA, Hess J, Harach LA, Scott S, Sinoway LI: Effect of hyperoxia and vitamin C on coronary blood flow in patients with ischemic heart disease. J Appl Physiol 2007; 102:2040–5
15. Hopf HW, Hunt TK, West JM, Blomquist P, Goodson WH III Jensen JA, Jonsson K, Paty PB, Rabkin JM, Upton RA, von Smitten K, Whitney JD: Wound tissue oxygen tension predicts the risk of wound infection in surgical patients. Arch Surg 1997; 132:997–1004
16. Greif R, Akca O, Horn EP, Kurz A, Sessler DI: Supplemental perioperative oxygen to reduce the incidence of surgical-wound infection: Outcomes Research Group. N Engl J Med 2000; 342:161–7
17. Belda FJ, Aguilera L, Garcia de la Asuncion J, Alberti J, Vicente R, Ferrandiz L, Rodriguez R, Company R, Sessler DI, Aguilar G, Botello SG, Orti R: Supplemental perioperative oxygen and the risk of surgical wound infection: A randomized controlled trial. JAMA 2005; 294:2035–42
18. Pryor KO, Fahey TJ III, Lien CA, Goldstein PA: Surgical site infection and the routine use of perioperative hyperoxia in a general surgical population: A randomized controlled trial. JAMA 2004; 291:79–87
19. Dellinger EP: Increasing inspired oxygen to decrease surgical site infection: Time to shift the quality improvement research paradigm. JAMA 2005; 294:2091–2
20. Brasel K, McRitchie D, Dellinger P: Canadian Association of General Surgeons and American College of Surgeons Evidence Based Reviews in Surgery 21: The risk of surgical site infection is reduced with perioperative oxygen. Can J Surg 2007; 50:214–6

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Anesthesia-related Maternal Deaths: Where Is "Regional Anesthesia"?

To the Editor:—We read the article by Mhyre and colleagues with interest.[1] Although in the United Kingdom problems with tracheal intubation during obstetric general anesthesia remain the leading cause of death,[2] there were no cases in their series from 1985 to 2003 suggesting that anesthesia providers in Michigan have been able to address this risk successfully. This is especially commendable as the rate of general anesthesia deaths, largely resulting from intubation problems or aspiration, was stable in the United States from 1979 to 1990.[3]

Many deaths in Mhyre *et al.*'s article were related to problems occurring some time after the induction of anesthesia. There are considerable differences between the United States and United Kingdom in anesthesia staffing both in the operating room and on the delivery suite, but the United Kingdom Confidential Enquiries into Maternal Deaths have repeatedly highlighted the vital role of the anesthesiologist in overall maternity care rather than just during surgery.[2]

However, we were surprised that there was no mention of regional anesthesia problems in the abstract or the accompanying editorial,[4] as this was the primary cause of three of four anesthetic-related deaths during cesarean delivery. Of the eight patients who died from anesthesia-related causes, three were in the first or second trimester. The five patients in their third trimester all underwent cesarean delivery. One patient who had received a spinal anesthetic died 9 h postoperatively, related to systemic morphine treatment. The type of anesthetic is irrelevant to the death as the patient would likely have had patient-controlled analgesia even if she had had general anesthesia. Only one of the four women in whom the sequence of adverse events started during surgery had a primary general anesthetic, whereas three had regional anesthesia: a respiratory event at the end of surgery with a spinal, a cardiopulmonary arrest with an epidural test dose (presumably from accidental spinal placement), and a cardiac arrest with a spinal. Deaths from obstetric regional anesthesia are rare,[5] and regional anesthesia is much safer than general anesthesia.[3,6] However, this rarity means that the risk of cardiac arrest may be forgotten and need to be relearned.[7]

We need to recognize that cardiovascular collapse[8] and unconsciousness from high block[9] may occur suddenly at any stage during regional anesthesia and lead to morbidity or death unless managed promptly and effectively.[5,9]

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References

1. Mhyre JM, Riesner MN, Polley LS, Naughton NN: A series of anesthesia-related maternal deaths in Michigan, 1985-2003. Anesthesiology 2007; 106:1096–104
2. The Confidential Enquiries into Maternal and Child Health. Why Mothers Die 2000-2002. The Sixth Report on the Confidential Enquiries into Maternal Deaths in the United Kingdom. London, RCOG Press, 2004
3. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP: Anesthesia-related deaths during obstetric delivery in the United States, 1979-1990. Anesthesiology 1997; 86:277–84
4. D'Angelo R: Anesthesia-related maternal mortality: A pat on the back or a call to arms? Anesthesiology 2007; 106:1082–4
5. Kinsella SM, Dob D, Holdcroft A. Regional anaesthesia, Crises in Childbirth: Why Mothers Survive. Lessons from the Confidential Enquiries into Maternal Deaths. D Dob, G Cooper, A Holdcroft (Eds). Oxford, Radcliffe Publishing, 2007.
6. Shibli KU, Russell IF: A survey of anaesthetic techniques used for caesarean section in the UK in 1997. Int J Obstet Anaesth 2000; 9:160–7
7. Keats AS: Anesthesia mortality: A new mechanism. Anesthesiology 1988; 68:2–4
8. Kinsella SM, Tuckey JP: Perioperative bradycardia and asystole: Relationship to vasovagal syncope and the Bezold-Jarisch reflex. Br J Anaesth 2001; 86:859–68
9. Yentis SM, Dob DP: High regional block: The failed intubation of the new millennium? Int J Obstet Anaesth 2001; 10:159–61

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In Reply:—We thank Kinsella *et al.* for their excellent comments. Our case series did include three women who suffered catastrophic complications after regional anesthesia for cesarean delivery.[1] The mechanism of death in each of these cases was unique. One patient developed high spinal from an inadvertent intrathecal pharmacologic test dose during epidural catheter placement. Despite prompt and successful conversion to general anesthesia, she experienced massive myocardial infarction. Another died from neuraxial cardiac arrest following spinal anesthesia, with cardiovascular collapse preceding delivery of her neonate. The third patient became apneic *en route* to the postanesthesia care unit after a spinal anesthetic supplemented with intravenous medications.

Regional anesthesia may be safer than general anesthesia,[2,3] but respiratory arrest or cardiovascular collapse may still occur[1–4*] during induction, maintenance, or recovery. Skills in basic airway management and cardiopulmonary resuscitation are essential for the entire team of perioperative care providers, regardless of whether the patient receives general or regional anesthesia.

* See also Lofsky S: Anesthesia-related maternal arrests on labor and delivery. Available at: www.thedoctors.com. Accessed September 6, 2007.

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References

1. Mhyre JM, Riesner MN, Polley LS, Naughton NN: A series of anesthesia-related maternal deaths in Michigan, 1985-2003. ANESTHESIOLOGY 2007; 106:1096–104
2. The Confidential Enquiries into Maternal and Child Health. Why Mothers Die 2000-2002. The Sixth Report on the Confidential Enquiries into Maternal Deaths in the United Kingdom. London, RCOG Press. 2004
3. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP: Anesthesia-related deaths during obstetric delivery in the United States, 1979-1990. ANESTHESIOLOGY 1997; 86:277–84
4. Kinsella SM, Dob D, Holdcroft A: Regional Anesthesia. In: Crises in Childbirth - Why Mothers Survive. Lessons from the Confidential Enquiries into Maternal Deaths. Edited by Dob D, Cooper G, Holdcroft A. Radcliffe Publishing Ltd., Oxford, 2007

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Surprises in Plethysmography

To the Editor:—In a recent editorial,[1] Pinsky elaborated on the use of the plethysmographic recording obtained from a pulse oximeter in assessing volume responsiveness before instituting volume resuscitation. Pinsky noted that the plethysmographic signal is dependent on the density of tissue and pulsatile blood in the pathway of the infrared and red wavelengths and that this ". . . will be a function of both perfusion pressure and vasomotor tone. As upstream vasomotor tone increases, for example, pulse oximeter plethysmographic changes would decrease for the same pulse pressure, and *vice versa* with vasodilation." The question is, however, whether it is the *density* or the *variation in density* that defines the plethysmographic variability.

I have for a couple of years routinely monitored pulse oximetry and plethysmography (Datex-GE, Helsinki, Finland) bilaterally in the lower extremities during abdominal aortic aneurysm repair and unilaterally in peripheral vascular reconstructive surgery. The plethysmographic signal is the first and failsafe herald of peripheral perfusion, and the lack of it likewise immediately warns of thromboembolic complications after declamping.

During one such procedure involving popliteal artery reconstruction under general anesthesia and spontaneous ventilation using a laryngeal mask, the plethysmographic signal after declamping is shown in figure 1, with the insert showing highly irregular beat-to-beat variation in amplitude.

The variation corresponds to variation in amplitude seen in the arterial pressure recording of the radial artery. The "silent" period at approximately 150 s represents manual compression of vascular graft. A perivascular Doppler flow probe was attached to the graft measuring a flow of 100 ml/min. The surgeon decided to assess the flow reserve of the receiving vascular bed, and papaverine 40 mg was injected intraarterially proximally to the graft. The Doppler signal instantaneously increased to 200 ml/min, and the ensuing plethysmographic recording is shown in figure 2. The plethysmogram was all but abolished in response to the injection of papaverine. The arterial pressure remained largely unaffected by the peripheral vasodilation.

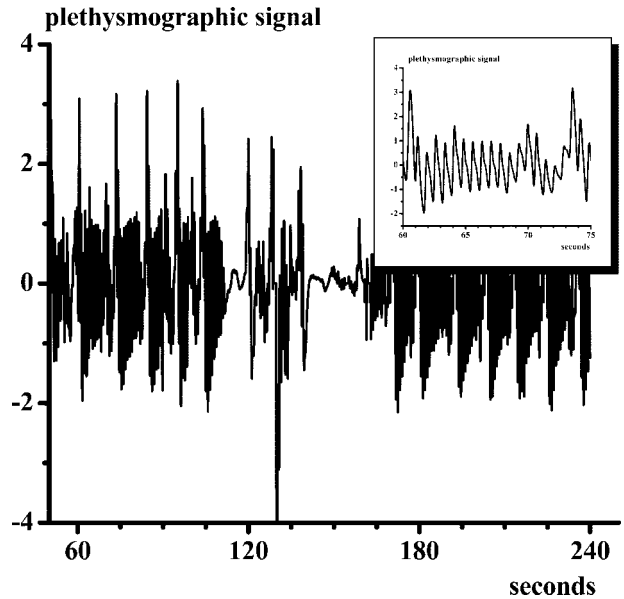

Fig. 1. Registration of plethysmographic signal shortly after declamping of graft. "Silent" period at 150 s represents manual compression of graft. Insert demonstrates the initial highly irregular plethysmographic signal from the reperfused extremity.

Initially, the response baffled me as I was expecting an increase with vasodilation, as stated by Pinsky. On second thought, the explanation became evident: the plethysmographic signal in Datex-GE pulse oximeters is highpass filtered, which resets the oscillations to vary between

The above letter was sent to the authors of the referenced report. The authors did not feel that a response was required.—James C. Eisenach, M.D., Editor-in-Chief.

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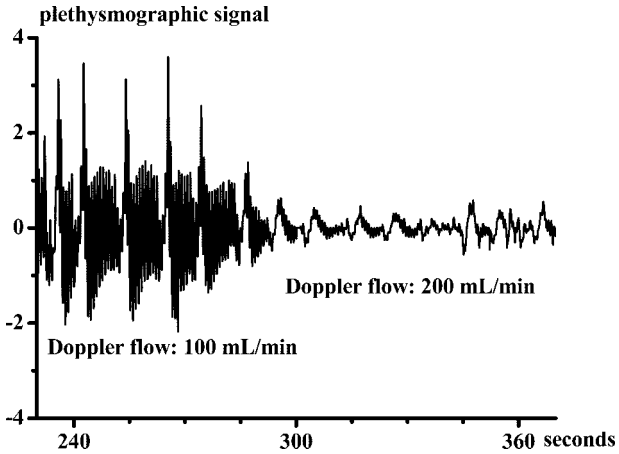

Fig. 2. Registration of plethysmographic signal and Doppler flow after injection of papaverin 40 mg intraarterially after reperfusion of extremity. The papaverin causes an increase in graft blood flow, but the dilation and diminished pulsatile oscillations of the receiving vascular bed decrease the plethysmographic signal.

negative and positive values, and the signal is normalized by the direct current value. By vasodilating the vascular bed in the foot and increasing the direct current component (tissue and diastolic blood) of the plethysmographic signal, the oscillations of the arterial wall diminished even though the vessels carried a higher flow, decreasing the alternating current (systolic blood) component of the plethysmographic signal. This observation was later repeated in another patient.

In conclusion, knowledge of the software algorithm of the pulse oximeter is essential to the interpretation of changes. In addition, without proper estimate of flow in and diameter of the vessel, any change in plethysmographic signal after a pharmacological intervention or fluid challenge must be interpreted with utmost caution.

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Reference

1. Pinsky MR. At the threshold of noninvasive functional hemodynamic monitoring. ANESTHESIOLOGY 2007; 106:1084–5

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Herniation of the Laryngeal Mask Airway Classic

To the Editor:—I read with interest Wrobel and Ziegeler's article[1] that airway obstruction occurred during the use of a reusable laryngeal mask airway classic (LMA-Classic; The Laryngeal Mask Company, Henley-on-Thames, United Kingdom). In their report, a clear airway was obtained after insertion of a size 4 LMA-Classic and after inflation of the cuff with 20 ml air. During anesthesia, 10 ml air was added to the cuff to prevent airleak around the device, but the airway was obstructed. Close examination of the device after its removal showed that there was a cuff herniation that had not been detected at preuse checking.

In a previous study,[2] which Wrobel and Ziegeler did not mention in their report, I showed that cuff herniation of the LMA-Classic may not be apparent if the cuff is inflated with the volume less than the recommended maximal volume (*e.g.*, 30 ml for size 4) but may only become apparent when inflated with a volume of air 50% greater (45 ml). I also reported that if the cuff is inflated with smaller volume of air (*e.g.*, 20 ml), there may be a dimple in the herniated part of the cuff, suggesting that there is a weakness of the cuff. [2] Wrobel and Ziegeler[1] stated that there was no problem at preuse checking, but the cuff was checked by inflating it with 20 ml air (Marc Wrobel, M.D., Department for Anesthesiology and Intensive Care Medicine, University of Saarland, Homberg/Saar, Germany; personal written commnunication, 2007). In figure 1 of their report,[1] cuff herniation was apparent when the cuff was inflated with 40 ml air (Marc Wrobel, M.D., Department for Anesthesiology and Intensive Care Medicine, University of Saarland, Homberg/Saar, Germany, personal written communication, 2007), and when the cuff was inflated with smaller volume of air (fig. 2 of their report), there was a dimple in the herniated part of the cuff.[1] These features are strikingly similar to results obtained in my previous article.[2] Therefore, if the integrity of the cuff had been checked by inflating the cuff with 45 ml air in their case, airway obstruction resulting from the damaged LMA-Classic might have been avoided.

Wrobel and Ziegeler suggested that the use of a silicone lubricant may damage the cuff. [1] I pointed out that sterilization of the device in an autoclave with the cuff not tightly deflated or before any fluid has been infused into the cuff may also damage the cuff.[2] Any reusable device, particularly a well-used one, should undergo a proper preuse checking, and I suggest the following.

1. Before each use, the absence of cuff herniation (and any other deformities) should be confirmed by inflating the cuff with a volume of air 50% greater than the recommended maximal volume. This practice is now recommended by the manufacturer.
2. Before use, the absence of dimple in the cuff should be confirmed by inflating the cuff with 15-20 ml air.
3. After insertion of the LMA-Classic, the cuff should be inflated with approximately 15 ml air,[3] and if airleak occurs around the cuff, air may be added until the cuff volume reaches the recommended maximal value.
4. If there is still airleak, the device should be removed, and a larger one should be used. The integrity of the removed device should be rechecked.

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The above letter was sent to the authors of the referenced report. The authors did not feel that a response was required.—James C. Eisenach, M.D., Editor-in-Chief.

References

1. Wrobel M, Ziegeler S: Airway obstruction due to cuff inflation of a classic reusable laryngeal mask airway. ANESTHESIOLOGY 2007; 106:1255–6
2. Asai T, Koga K, Morris S: Damage to the laryngeal mask by residual fluid in the cuff. Anaesthesia 1997; 52:977–81
3. Asai T, Brimacombe J: Cuff volume and size selection with the laryngeal mask (Review). Anaesthesia 2000; 55:1179–84

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A Complication Associated with the Use of a Drug Injection Catheter through a Fiberscope

To the Editor:—Fiberoptic intubation is a very useful technique for patients whose tracheas are difficult to intubate using conventional methods. However, insertion of a fiberscope and insertion of an endotracheal tube over the fiberscope into the trachea are two major difficulties.[1] We encountered an unusual difficulty during removal of the fiberscope after successful tracheal intubation. Our search revealed no such difficulty reported in the literature.

A 30-yr-old man with atlanto-axial dislocation came for trans-oral decompression and posterior fixation. An awake intubation was planned. After adequate oral and intravenous sedation, bilateral superior laryngeal nerve blocks, and topical anesthesia to the airway, a bite block was inserted.

Endotracheal tube (8.5, polyvinyl chloride) was loaded over the fiberscope (Fujinon F B 120-p, Fujinon Corporation, Saitoma, Japan). A long catheter (drug spray cannula) was inserted through forceps port of the fiberscope for the administration of local anesthesia in the event of inadequate anesthesia. This catheter comes with a fiberscope set.

The fiberscope and enodotracheal tube were inserted through the mouth. Excellent local anesthesia was observed while introducing the fiberscope and the tube into the trachea, which was performed within 30–40 s. The patient remained comfortable during and after intubation of the endotracheal tube without any sign of distress. Administration of local anesthesia was not required. With successful intubation, we tried to remove the fiberscope. Initially we felt some resistance. After applying slight force, the fiberscope came out but the injection catheter remained inside the tube. We were unable to remove the catheter with moderate force. To determine the reason, we reinserted the fiberscope into the trachea over the catheter. We noticed that the catheter had gone beyond the tip of the endotracheal tube, making a U shape over the edge of it. We removed the tube along with the fiberscope and the catheter. The catheter was not used during the second attempt of awake fiberoptic intubation, which was accomplished without difficulty. Subsequent endoscopic examination of the distal trachea and major bronchi revealed normal anatomy.

We hypothesized that during advancement of the endotracheal tube over the fiberscope, the injecting catheter migrated beyond the tip of the fiberscope. With further advancement, it became U shaped over the edge of the tube, and one arm of it got trapped between the tube and the trachea, which caused it to be stalked within the tube (figure 1).

Support was provided solely from institutional and/or departmental sources.

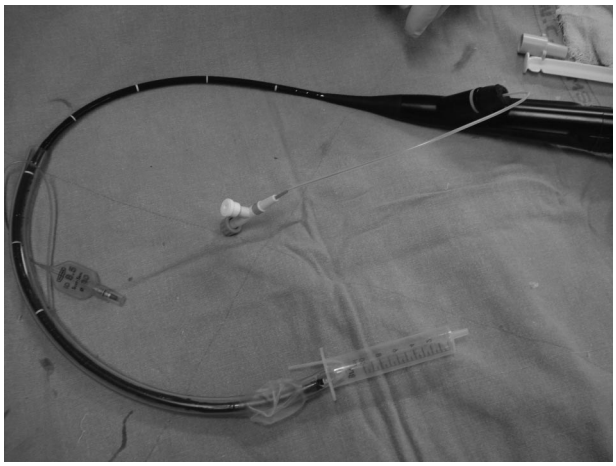

Fig. 1. The drug injection catheter protruding out from the fiberscope and making a U shape over the tip of the endotracheal tube simulating the condition *in situ*.

Inadvertent advancement of the tip of the fiberscope through a side hole (Murphy eye) of an endotracheal tube, causing difficulty in intubation has been reported.[2]

To eliminate this kind of problem with the use of an injection catheter, we suggest a simple measure. The catheter should be fixed with adhesive tape or a rubber band at the inlet of the forceps port so that it will not protrude from the tip of fiberscope *in situ*.

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References

1. Asai T, Shingu K.. Difficulty in advancing a tracheal tube over a fibreoptic bronchoscope: incidence, causes and solutions. Br J Anaesth 2004; 92:870–81
2. Nichols KP, Zornow MH: A potential complication of fiberoptic intubation. ANESTHESIOLOGY 1989; 70:562–3

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