colorimetric end-tidal CO_2 detection (1) and the negative pressure test using a syringe (2) or a self-inflating bulb (3). Recently, Kasper and Deem (3) found that negative pressure using a self-inflating bulb is a sensitive and specific test to detect esophageal intubation in emergency setting.

We are currently using a simplification of the syringe technique described by Wee (2), based on a 50-mL syringe connected to a breathing system connector's end-tidal CO_2 sampling site.

Instead of connecting the syringe to the endotracheal tube by a special connector, we attached the syringe's luer lock to a simple breathing system's connector's end-tidal CO_2 sampling hole. One end of the connector is closed hermetically with a plug (plastic or cork), and the other end is attached to the endotracheal tube (Fig. 1). This device is simple, reliable, and costs less than $1.

Tiberiu Ezri, MD*
Peter Szmuk, MD†
Daniel Geva, MD*
Departments of Anesthesiology
**Kaplan Hospital*
Rehovot, Israel
†University of Texas
Houston, TX 77030

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Transfusion Requirements in Orthotopic Liver Transplantation

To the Editor:

We read with interest the report by Frenette et al. (1) on the use of conjugated estrogen (CE) to reduce transfusion requirements during orthotopic liver transplantation (OLT). Since Neuhaus et al. (2) reported the effect of large-dose aprotinin on blood transfusion in OLT, many groups have tried to show the beneficial effects of the prophylactic use of several drugs, such as aprotinin (3), tranexamic acid (4), prostaglandin E_1 (5), antithrombin III (6), and now CE (1). However, no clear evidence supports the use of any of them in OLT. Reasons for this lack of evidence are:

- Heterogeneity of the recipient population, with varying degrees of coagulopathy (from the deranged coagulation of fulminant hepatic failure to the prothrombotic state of some cancer patients).
- Focus by authors on different outcomes (i.e., blood loss versus blood replacement), with different trigger values for blood transfusion, and coagulation factor replacement protocols guided by different monitoring techniques (i.e., thromboelastography versus conventional coagulation parameters).
- Unpredictable intraoperative factors unrelated to anesthesiologist management and basal patient status (technical complications, surgical expertise, etc).

Guidelines for good clinical research practice should be developed to enhance the quality and external validity of future clinical trials on the subject. Until then, generalized use of any specific drug remains a matter of local experience without solid scientific ground.

Angel M. Candela-Toha, MD
Adolfo Martínez-Pérez, MD
Department of Anesthesiology
Liver Transplantation Unit
Ramón y Cajal Hospital
Alcalá de Henares University
Madrid, Spain

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In Response:

We thank Drs. Martínez-Perez and Candela-Toha for their thoughtful reading of our study. They expressed some concern about previous studies from different authors and subjects. Obviously, I am not able to answer their interrogation about prior studies concerning aprotinin (2), traxemic acid (3), and prostaglandin E_1 (4). However, I can answer questions about our study concerning conjugated estrogen during orthotopic liver transplantation (OLT).

The "profusion of evidence" for the use of conjugated estrogen during OLT resides in Table 2 of our original study, which shows a statistical significant decrease in the use of blood products when conjugated estrogen was used instead of a placebo.

The concern about the heterogeneity of our study population is unfounded. No patients were in fulminant hepatic failure or had cancer before the OLT. The initial preoperative computed thromboelastography (CTEG) variables showed no statistical differences between the placebo and the estrogen group, which implies a similar coagulopathies preoperatively.

The concern about different triggers for blood transfusion monitoring techniques is also unfounded. Page 1185 of our study states, "no blood products were given empirically during OLT. All blood products were given in relation to the CTEG variables and hematocrit measurement."

The concern about unpredictable factors unrelated to anesthesia but related to surgical techniques are also unfounded. In our institution, the same group of anesthesiologist (team of two) and surgeons (team of two) were involved in every case of this study. A prospective, double-blinded study will statistically take care of any unpredictable factors.

The need for guidelines for clinical research resides in the structure of peer-reviewed medical journals. Editors of peer-reviewed journals decide which article or study need to be published. Poor-quality articles are not published in respectable peer-reviewed journals.

Luc Frenette, MD
Department of Anesthesiology
University of Alabama
Birmingham, AL 35233-6810

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External Jugular Vein Cannulation for Central Venous Access

To the Editor:

Central venous cannulation via the external jugular vein (EJV) is a recognized technique (1–3). It is associated with minimal complications but with a relatively frequent failure rate compared with the cannulation of the internal jugular or subclavian veins (SCV) (1,3,4). The use of the Seldinger J-wire increased successful central venous cannulation via the EJV from 50% to 79%–90% (2–4). Cannulation

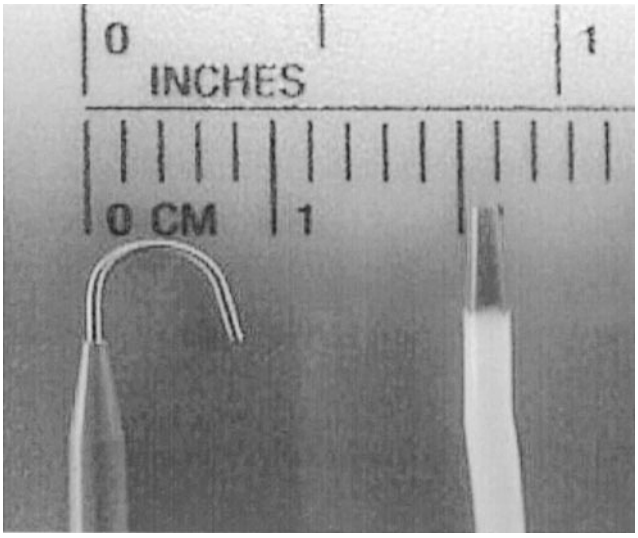

Figure 1. Differences in the width of the J-tip, J-wire, and the triple-lumen catheter.

failures are, in part, due to the difficult passage of the J-wire through the EJV-SCV junction into the thorax (3). Variations of the termination and angulation of the EJV as it enters the SCV and the distribution and morphology of the valves in the EJV contribute to this difficulty (2–7). We describe a technique that facilitates the placement of a 7F triple-lumen catheter (TLC) via the EJV when the J-wire could not transverse the EJV-SCV junction.

In 11 of 68 EJV cannulation attempts, we found that the J-wire could not transverse the EJV-SVC junction, as evidenced by resistance to further wire advancement. We withdrew the J-wire approximately 0.5 cm proximal to the junction and advanced the TLC over the J-wire. We found that the TLC negotiated through the venous junction pass the guide wire and entered the SVC without difficulty. The success of this technique may lie in the difference of the width of the tip of the J-wire (6–8 mm) and the TLC catheter (1–2 mm) (Figure 1). The diameter of EJV-SCV junction is 5.5 ± 1.6 mm (2). The tip may be too wide to negotiate through the venous junction, but the smaller TLC allows passage without difficulty.

We have found that the use of this rescue technique facilitated central vein cannulation through the EJV in 10 of 11 attempts.

Alvaro M. Segura-Vasi, MD
Melody D. Suelto, MD
Arthur M. Boudreaux, MD
Department of Anesthesiology
University of Alabama in Birmingham
Birmingham, AL 35233-6810

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Use of the Laryngeal Mask Airway in Children with Upper Respiratory Tract Infections: Beware Secretions

To the Editor:

I was interested to read the article by Tait et al. (1) on the use of the laryngeal mask airway (LMA) in children with upper respiratory tract infections. A common scenario we encounter in the United Kingdom is the child aged 2–6 yr with a chronic nasal discharge and cough with normal heart rate and temperature, but clinically "well."

Soon after the LMA became available, I used one on a 2-yr-old child for myringotomies after an inhaled induction of anesthesia. The child was well but had a nasal discharge. After blind insertion in the routine manner, as described by Dr. Tait, the child went into severe, long-lasting laryngospasm. This was remedied by removal of the LMA and laryngoscopy, which revealed yellow mucopus on the vocal chords. Suction clearance and reinsertion of the LMA enabled anesthesia and surgery to proceed uneventfully.

To prevent a recurrence of this episode, I now make a brief visual inspection of the pharynx and cords with a laryngoscope to suction any secretions before inserting a laryngeal mask into any child with a suspicion of nasal discharge. I recommend this precaution to your readers.

Brian L. Smith, FRCA
Anaesthetic Department
Wexham Park Hospital
Wexham, Slough, Berkshire SL24HL, UK

Reference

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In Response:

We thank Dr. Smith for sharing his experience of laryngospasm after placement of a laryngeal mask airway (LMA) in a child with an upper respiratory tract infection. Although we find his comments interesting, we cannot support his recommendation of routinely performing laryngoscopy and suctioning under direct visualization before LMA insertion in children in whom nasal discharge is observed or suspected. Indeed, we believe that this practice somewhat defeats the primary purpose of placing the LMA in a child with an upper respiratory tract infection, i.e., minimizing the stimulation of a potentially irritable airway. When excessive nasal/oral secretions are a problem, the practice at our institution is to ensure an adequate depth of anesthesia and to suction the naso/oropharynx blindly using a soft-tipped suction catheter before LMA insertion. Perhaps Dr. Smith may consider this as an alternative to his routine practice.

Alan R. Tait, PhD
Uma A. Pandit, MD
Terri Voepel-Lewis, BSN, MS
Hamish M. Munro, MD, FRCA
Shobha Malviya, MD
Department of Anesthesiology,
University of Michigan Medical Center
Ann Arbor, MI 48109

Is There a Responsibility to Disclose Data Used as the Basis for a Publication?

To the Editor:

Ebert et al. (1) have published the results of their work indicating that the administration of sevoflurane for 4 h at a 1-L/min flow does not produce renal injury. I am constructing reviews that examine

the issue of toxicity from compound A secondary to sevoflurane administration. To that end, I asked Professor Ebert to share some of the data on which the above article is based. He declined to do so.

I believe that this behavior is inappropriate for an academician. The academic enterprise depends on the free exchange of information, not on secrecy. I believe that an unwillingness to reveal the specific data on which a published result is based calls into question the validity of the published results.

Edmond I Eger II
Department of Anesthesia
University of California San Francisco
San Francisco, CA 94143-0464

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In Response:

Dr. Eger has suggested that our refusal to share our study data with him is inappropriate for an academician, and he believes that this should call into question the validity of our published results on renal function after sevoflurane in volunteers (1,2). Our research group has unanimously decided not to share these data with Dr. Eger, and I am less than enthusiastic to publicly provide our reasons for refusing his request. Clearly, when one publishes a study, it must be in a format that can be duplicated. When we duplicated the research methods of Dr. Eger for exposing volunteers to sevoflurane in a low fresh gas flow, our data were devoid of the significant changes in markers of renal function/integrity that were reported by Eger et al. (3,4).

Dr. Eger and I have both privately and publicly discussed possible differences between what seem to be identical study protocols. I have refused, on more than one occasion, Dr. Eger's request that we provide him with all of our renal and compound A data from two published sevoflurane studies in human volunteers (1,2). It is my strong belief that this issue has no place in our professional journals; rather, it is a personal matter between Dr. Eger and myself. However, the hubristic nature of his now public presentation of our personal dealings gives me further justification for our refusal to share data. The basis for our unwillingness to share data is our concern that the data might be misconstrued, perhaps inappropriately combined with other data, and most certainly interpreted with a bias. Our concerns are based on misleading and biased statements and summaries made by Dr. Eger in his past public lectures and debates.

Dr. Eger is a highly paid consultant to Baxter/Ohmeda, which markets a "competitor" anesthetic, desflurane, and he has received substantial research support from Ohmeda for more than a decade. Based on this alone, Dr. Eger should disqualify himself from authoring reviews on the new volatile anesthetics. At the very least, the reader must be alerted to the possibility of significant bias in his interpretation of data in articles and reviews.

Our refusal to share our data certainly does not invalidate the results of our rigorous scientific studies. Perhaps the best approach for pursuing this matter would be for Dr. Eger to seek support for an independent research group and a blinded analytical laboratory to duplicate the methods and procedures used in the protocols of Dr. Eger and myself. If independent findings were to differ from ours, Dr. Eger would have a scientific basis from which to question our study results. We would vastly prefer this to his suggestion that our unwillingness to share our data somehow invalidates our results. It is our belief that differing study outcomes should provide the impetus for further study, rather than the ammunition to denigrate the research findings of a fellow researcher and academician.

Thomas J. Ebert, MD, PhD
Department of Anesthesiology
The Medical College of Wisconsin
Veterans Affairs Medical Center
Milwaukee, WI 53295

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An Unusual Cause of a Double-Lumen Endotracheal Tube Obstruction

To the Editor:

A 40-yr-old woman was scheduled for left pulmonary hydatit cyst resection. Preoperative fiberoptic bronchoscopy disclosed no airway malformation and a normal trachea. After the induction of anesthesia, a 37F left endobronchial tube was inserted into the trachea after minimal resistance at the cricopharyngeus, and the tube was rotated 45° counterclockwise. The bronchial and tracheal cuff were inflated with 3 and 6 mL of air, respectively. Chest auscultation revealed a decrease of left breath sounds. Manual ventilation was difficult after clamping of the bronchial tube because of high airway resistance. An inability to pass a suction of catheter beyond the tip of the tracheal tube suggested tube obstruction. The double-lumen tube (DLT) was pulled back and the trachea was extubated. On examination of the DLT, obstruction of the distal aperture of the tracheal lumen caused by the carinal hook was noted (Fig. 1).

Potential problems with carinal hook have been observed (1). A similar incident was reported in a recently published case (2). A possible mechanism is that the laryngeal lumen was too small for the tip of the DLT and that the hook was bent by the lateral wall of the larnynx and trapped in the distal orifice of the tracheal lumen. Lack of lubrication of the DLT and the use of an inappropriate size of the DLT could have contributed to the incident.

We recommend that fiberoptic bronchoscopy of both lumens of the DLT be routinely performed when the DLT is used, and choice of an appropriate DLT size seems important.

Bouaggad Abderrahmane, MD
Abassi Omar, MD
Department of Anesthesiology
Ibn Rochd University Medical
Center of Casablanca, Morocco 20000

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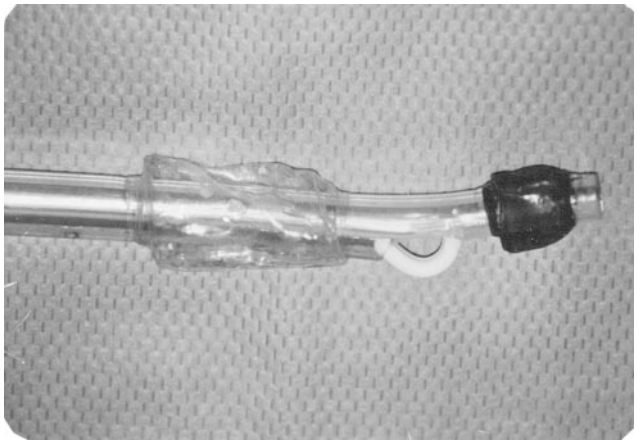

Figure 1. Closeup of the Double-lumen tube. The carinal hook is bent into the distal tracheal aperture.

Vasopressin and Postcardiopulmonary Bypass Refractory Hypotension

To the Editor:

Overand and Teply (1) recently described a patient with intractable hypotension after cardiopulmonary bypass (CPB) who improved dramatically after the administration of vasopressin (VP). We wish to make a few comments regarding experimental and clinical data suggesting that VP is a promising therapeutic tool. First, in addition to its potent vasoconstricting effects, VP stimulates the endothelial release of prostaglandins and nitric oxide (NO) in the pulmonary, coronary, and mesenteric vascular beds (2,3). Hence, the regional heterogeneity in VP receptor density and the balanced relaxant/constrictive effects of VP contribute to maintain cardiac circulatory output with minimal risk of myocardial ischemia. Second, although deficient release of VP has been documented in some patients with septic shock or with a ventricular assist device, reversal of vasodilatory hypotension with VP has been achieved regardless of the plasma concentration of VP (4). Third, VP inhibits interleukin-1 β-induced NO production in vascular smooth muscle, leading to restoration of vascular reactivity to endogenous contractile mediators such as endothelin, thromboxane A_2, and catecholamines (5). Taken together, these data lend support to the hypothesis that VP can partially reverse the adrenergic hyporesponsiveness associated with sepsis and circulatory bypass. Likewise, because the renin-angiotensin system modulates the central release of VP (6), we hypothesize that the impaired adrenergic response often observed in patients chronically treated with angiotensin-converting enzyme inhibitors (7) can be normalized with subpressor doses of VP.

Finally, in the case of combined surgical procedure (aortic and mitral valve replacement) and prolonged CPB (>6 h), one may question whether ultrafiltration could prevent the occurrence of vasodilatory hypotension through attenuation of the systemic inflammatory response elicited by CPB and surgical trauma. Intraoperative ultrafiltration reduces the circulating levels of several biological markers of inflammatory cascade and could contribute to lesser postoperative blood loss, better hemodynamic control, and earlier extubation after cardiac surgery (8).

Marc Licker, MD
Alexandre Schweizer, MD
Division of Anesthesiology
University Hospital
CH-1211 Geneva 14, Switzerland

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A Simple Technique for Oral Fiberoptic Bronchoscopy: "No More Needles, Doc"

To the Editor:

As fiberoptic bronchoscopy becomes part of the armamentarium of all anesthesiologists for securing the airway, multiple techniques have been described and utilized. These techniques involve antisialogog treatment; nebulization of local anesthetic solutions; topicalization of the airway with sprays, pledgets, and cotton swabs, as well as invasive peripheral nerve blocks of the nasal and oral pharynx. Most of these require significant time to prepare and administer and may be quite uncomfortable and anxiety-provoking for the patient. We all know how necessary thorough anesthesia of the airway is for instrumentation and intubation in the awake patient. The following describes a simple and timely method of anesthetizing the oropharynx for oral fiberoptic intubation.

It is important to administer an antisialogog, i.e. glycopyrrolate, before beginning for the local anesthetic to be effectively delivered to the oropharyngeal mucosa. To anesthetize the tongue and oral cavity, 10% lidocaine spray is used with the tongue protruded, approximately two to three sprays (each spray contains 10 mg of lidocaine). A large hollow oral airway with the distal third coated with 2% lidocaine jelly is placed a third of the way into the patient's mouth. This is repeated twice more, each time with approximately 2 mL of jelly applied to the end and moved further into the mouth. With each application, the patient is asked to open wide with the tongue out and to take the airway in as far as is tolerable. This elicits patient cooperation and gives them a measure of control in their care and your efforts. With the last one-third application and the airway fully in the mouth, the patient is instructed to inhale and exhale through it as if the airway were a straw. As the patient inhales, two to three puffs of 10% Lidocaine are sprayed into the hollow center of the airway and are drawn to the glottis. This is then replaced with an intubating oral airway, e.g. Ovassapian, with the endotracheal tube engaged, and bronchoscopy proceeds as routine. Often, the vocal cords are anesthetized by the above measures and no further topicalization is necessary; however, a small amount (2 mL of 2% lidocaine) may be sprayed through the injection port of the bronchoscope just before advancing past the cords. The full time for this process is approximately 8 mins. Patients tolerate this procedure well even without sedation; however, sedation (most frequently with midazolam or midazolam/fentanyl combinations) is recommended for patient comfort and anxiolysis.

This method is especially useful in patients who are needle shy, whose oropharyngeal anatomy is difficult to access for peripheral nerve blocks, and those with surgical or radiation altered anatomy. This technique may be used, in addition to the operative indication, for difficult airway procurement on the ward, in the intensive care unit, and timely and comfortable endoscopy and bronchoscopy suite experiences.

Maria E. Fagan Shier, MD
Department of Anesthesiology
Long Beach Veterans Affairs Medical Center and
University of California at Irvine
Orange, CA 92668

Hypoxic Ventilatory Response: The Effects of CO_2 and of Sustained Hypoxia

To the Editor:

I have two comments to make on the article by Sjögren et al. (1) on the ventilatory responses to acute and sustained hypoxia during isoflurane anesthesia.

Figure 4 of their article claims to show hypoxic-hypercapnic interaction in the awake state and the effect of isoflurane on this. To demonstrate such an interaction, it is necessary experimentally to increase $ETco_2$ from resting levels in the same subject and to measure the hypoxic ventilatory response (HVR) at both the lower and the higher level of CO_2. Sjögren et al. have not done this, but have simply plotted values for HVR from individual subjects against their ambient air-breathing $ETco_2$ values. The interpretation of this figure for their awake subjects, as they have drawn it, is that subjects with a high ambient $ETco_2$ have a high HVR, and it therefore has nothing to do with the notion of hypoxic-hypercapnic interaction. Furthermore, this proposed relationship between $ETco_2$ and HVR does not actually hold true for a larger population. Figure 1 shows

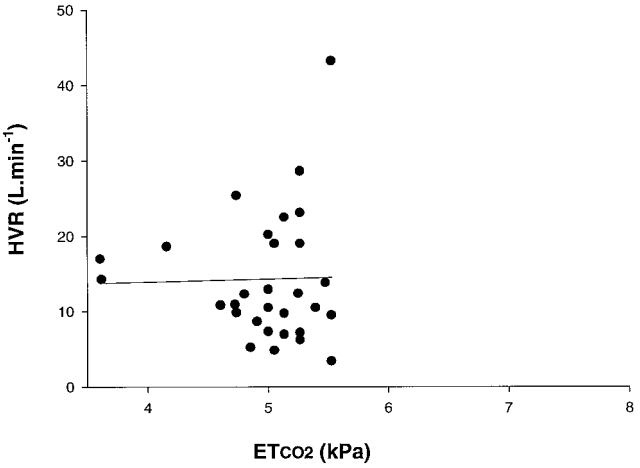

Figure 1. Individual values of hypoxic ventilatory response (HVR) at the y axis and individual values of $ETco_2$ at the x axis. The scale of the x axis is the same as that used by Sjögren et al. (1), for more direct comparison. Data are taken from 24 awake, normal subjects at rest who participated in studies described in references (2–5) and from 8 subjects whose results are unpublished. The regression line through these data points is also shown and, in contrast to the awake subjects of Sjögren et al. (1), is close to 0.

results I have obtained from 32 awake normal subjects. Twenty-four of these subjects participated in previously published studies (2–5), whereas data from eight subjects are unpublished. The figure also shows the regression line through these data points. This result from a larger group of subjects provides no support for Sjögren et al.'s novel hypothesis.

My second comment concerns the assertion that attenuation of peripheral chemoreceptor sensitivity with sustained hypoxia in adult humans has little support in the literature. Sjögren et al. refer to two studies that used methods to block the hypoxic stimulation of the peripheral chemoreceptors in an attempt to uncover a central hypoxic depression of ventilation (6,7). However, a more direct method has been used by Honda (8) and Honda et al. (9), who studied subjects with carotid body resection. The prediction was that, if central depression of ventilation existed, these subjects should demonstrate a decrease in basal ventilation with sustained hypoxia. Their results were that ventilation remained constant during hypoxic exposure, providing no support for the notion that a central depression of ventilation occurs (8,9).

Jaideep J. Pandit, MA, BMBCH, DPHIL, FRCA
Department of Anesthesiology
University of Michigan Medical Center
Ann Arbor, MI 48109

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In Response:

Dr. Pandit has made two comments regarding our article to which we would like to respond.

It was not our aim to investigate the question of O_2/CO_2 interaction during hypoxia in this study. We agree with Dr. Pandit about the need to increase end-tidal CO_2 concentrations when studying this interaction specifically. However, in our study, we found that although the individual end-tidal CO_2 concentrations were higher during anesthesia than when awake, the hypoxic ventilatory responses (HVR) were lower when patients were anesthetized, rather than awake. In our conclusions, we therefore suggest that this elimination is induced by isoflurane. The figure in Dr. Pandits letter does not seem to be comparable to our figure, as his y-axis illustrates minute ventilation (L/min) and ours illustrated HVR ($L \cdot min^{-1} \cdot \%^{-1}$). Furthermore, it seems strange that higher end-tidal CO_2 in Dr. Pandit's figure did not result in a larger minute ventilation, but this could be explained by the fact that the 32 subjects to whom he refers (excluding the 8 subjects from unpublished data) are from four different studies, with four different investigational protocols, addressing four different aims! Regarding the remark on the small number of subjects in our investigation, we do recognize that there is, in clinical studies of hypoxia, a problem regarding the number of subjects, not only from a statistical, but also from an ethical, point of view.

In his second comment, Dr. Pandit notes more recent investigations discussing the problem of whether the hypoxic ventilatory decline during sustained hypoxia is of a primarily central or peripheral origin. This is a matter of great controversy in the literature. The review by Honda (1) refers to an investigation including two patients with bilaterally resected carotid body and one patient with a unilateral resection. This study showed that the patient with a unilateral resection did exhibit a biphasic HVR, whereas the two patients with a bilateral resection did not. However, other studies suggest that the input from peripheral chemoreceptors in the carotid body could be modulated in the central nervous system to effect the hypoxic ventilatory depression seen during sustained hypoxia (2). Together with our two cited references (3,4), we therefore suggest that there are probably more than one factor responsible for this ventilatory depression. However, this may be a subject for further studies.

Dick Sjögren, MD
Sten G. E. Lindahl, MD, PhD, FRCA
Alf Sollevi, MD, PhD
Department of Anesthesiology and Intensive Care
Karolinska Hospital and Institute
171 76 Stockholm, Sweden

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