Real-time Monitoring of Propofol in Expired Air in Humans Undergoing Total Intravenous Anesthesia

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**Background:** The physicochemical properties of propofol could allow diffusion across the alveolocapillary membrane and a measurable degree of pulmonary propofol elimination. The authors tested this hypothesis and showed that propofol can be quantified in expiratory air and that propofol breath concentrations reflect blood concentrations. This could allow real-time monitoring of relative changes in the propofol concentration in arterial blood during total intravenous anesthesia.

**Methods:** The authors measured gas-phase propofol using a mass spectrometry system based on ion–molecule reactions coupled with quadrupole mass spectrometry which provides a highly sensitive method for on-line and off-line measurements of organic and inorganic compounds in gases. In a first sequence of experiments, the authors sampled blood from neurosurgery patients undergoing total intravenous anesthesia and performed propofol headspace determination above the blood sample using an auto-sampler connected to the mass spectrometry system. In a second set of experiments, the mass spectrometry system was connected directly to neurosurgery patients undergoing target-controlled infusion via a T piece inserted between the endotracheal tube and the Y connector of the anesthesia machine, and end-expiratory propofol concentrations were measured on-line.

**Results:** A close correlation between propofol whole blood concentration and propofol headspace was found (range of Pearson $r$, 0.846–0.957; $P < 0.01$; $n = 6$). End-expiratory propofol signals mirrored whole blood values with close intraindividual correlations between both parameters (range of Pearson $r$, 0.784–0.985; $n = 11$).

**Conclusion:** Ion–molecule reaction mass spectrometry may allow the continuous and noninvasive monitoring of expiratory propofol levels in patients undergoing general anesthesia.

**Materials and Methods**

All clinical studies were approved by the institutional review board of the Ludwig-Maximilians University of Munich (protocol No. 089/04), and data protection met the standard set by German law. All patients gave written informed consent to participate in the investigation.

**The Mass Spectrometry System**

The IMR-MS system used in this study was originally designed to measure trace gas components in industrial fields such as fuel cell or reformer gas development, work space control, and environmental measurements (Airsense Mass Spectrometry Systems; V&F Medical Development GmbH, Absam, Austria). The substance is increasingly used as part of total intravenous anesthesia (TIVA) because propofol allows rapid awakening and is known to reduce the incidence of postoperative nausea and vomiting. Propofol is an aromatic compound with a distinct smell, a molecular weight of 178 Da, low water solubility, and a vapor pressure of 0.142 mmHg at 20°C. Although the partition coefficient of propofol between blood and air has not been determined, these physicochemical properties could allow the detection and even measurement of propofol in gas-phase in close proximity to a propofol-containing solution such as the blood–air interface in the lungs of patients undergoing TIVA. Indeed, the possibility of propofol quantification in expired alveolar gas has recently been demonstrated in animals, and propofol has been shown in a single patient to appear in expiratory air. Propofol measurements in expiratory air could serve as a surrogate marker for intraoperative propofol plasma levels, which are not yet possible to monitor on-line. Here we show that propofol can be measured routinely in the airspace above blood samples taken from patients during TIVA as well as on-line in expiratory air of patients undergoing propofol anesthesia using a novel ion–molecule reaction mass spectrometry (IMR-MS) system that allows direct molecular analysis of organic and inorganic compounds in gases.
A schematic diagram of the IMR-MS is shown in figure 1. In the IMR-MS analyzers, the principle of ion–molecule reactions is applied as the interaction of positively charged atomic ions with neutral sample gas molecules in two-body collision processes resulting in the formation of product ions whenever the ionization potential of the sample molecule is less than the potential energy of the incoming primary ion and hence the entropy of the process becomes positive. The excess energy of the binary reaction is first stored in the product ion as transition state and either is statistically distributed in internal degrees of freedoms (electron vibration, bond oscillations) or is used up to break the weakest bond of the ionized molecule leaving a lower molecular weight ion.

Differences in ionization potentials between primary and product ions may result in a bond rupture and hence a lower molecular weight fragment ion.

The IMR-MS uses krypton, xenon, or mercury atomic gas to form the primary ion beam via electron impact ionization (section 1, fig. 1). In our experiments, mercury ions were used. They were generated out of mercury vapor by electron impact ionization.

The patented IMR ionization method can use the atomic mass scale to detect different molecules with the same molecular weight. As an example, acetaldehyde and carbon dioxide have the same mass (44). The mercury beam with an ionization potential of 10.4 eV does not ionize carbon dioxide (13.8 eV), but it ionizes acetaldehyde (10.2 eV). Switching different ion beams and hence energy levels is fast and takes 400 ms. Krypton ions (14.0 eV) do well separate nitrogen (15.6 eV—not ionized) against carbon monoxide (13.7 eV) on mass 28.

The instrument uses two octopole systems (section 2 and 3, fig. 1) operated at high frequencies to store primary as well as product ions in a confined volume against their coulomb repulsion and transmit ions to the quadrupole mass analyzing section.

The quadrupole mass separator (section 4, fig. 1), driven by direct current and alternating current, operates as an electromagnetic filter according to a parametric resonance to a specific mass over charge ratio. At a given alternating to direct current ratio, only one specific mass of ions experiences a stable trajectory through the quadrupole.

A secondary electron multiplier (section 5, fig. 1) may generate as much as $10^8$ electrons for each incoming ion. This allows the generation of an electrical pulse strong enough to be accepted by a computer counting system. The pulse rate represents the concentration of the molecular species in the gas sample brought to the instrument.

The sample gas (section 7, fig. 1) is transferred in a 2.5-m-long heated capillary system (Silcosteel®; Restek, Bellefonte, PA) at a flow rate of 50 ml/min to the instrument. A constant pressure controller feeds via a second capillary a stable amount of 1.5 ml/min into the high vacuum ionization section.

Gas response times to concentration changes are 50 ms, and gas dead times depending on the duration of the transfer capillary range between 2 and 4 s.

**Monitoring of Propofol**

When analyzing expiratory air for propofol with this instrument, we initially monitored the intact propofol molecule with a molecular mass of 178 Da (propofol 178). Early experiments did show, however, that propofol 178 interacted during the ionization process with trace amounts of residual sevoflurane, which are usually present in the anesthesia circuit. This was caused by an
interaction of the C$_4$H$_2$OF$_6$ fragment of sevoflurane that influenced mass 178, which was adjusted to a soft mass separation to gain signal strength. This resulted in a false-positive increase in propofol 178 signaling. This interaction was not seen when measuring a propofol fragment with a molecular mass of 163 Da (propofol 163), which is internally generated in the IMR-MS during ionization of the intact propofol molecule. Propofol 163 appears because during the ionization of propofol, one methyl group of the two isopropyl groups of the molecule is removed, thus resulting in a fragment of 163 Da. Figure 2 gives an example of the effects of a brief administration of sevoflurane (0.3%) on propofol 178 and propofol fragment 163 in a patient during TIVA.

Two different methods for quantification of propofol in gas phase were used in this study. One consisted of analysis of headspace gas above blood samples containing propofol and was performed in vitro and off-line. The other technique was performed in vitro and on-line using a direct connection of the IMR-MS system to the endotracheal tube via a T piece.

In Vitro (Off-line) Studies

For in vitro studies, the IMR-MS system was connected to an auto-sampling device that allowed gas sampling in the headspace immediately above liquids without direct contact with the solution. For these experiments, blood was anticoagulated with EDTA, stored, and measured in airtight 20-ml glass vials. Blood samples were kept on ice until analyzed and incubated at 37°C for 30 min in the auto-sampler before headspace gas analysis was performed.

Spiked Blood

In a first series of experiments, whole venous blood of healthy volunteers (n = 5; aged 38.0 ± 6.8 yr; 3 females) was spiked with propofol (Propofol-Lipuro® 1%; B. Braun, Melsungen, Germany) to reach concentrations of 0, 0.5, 1, 1.5, 2, 2.5, 5, and 10 µg/ml and then transferred to glass vials (0.5 ml/vial) for off-line analysis. After incubation, propofol was measured in the headspace above blood in the scaled glass vials. As a control, propofol measurements were done in the headspace above the 10% lipid carrier solution of propofol.

Perioperative In Vitro (Off-line) Measurements

In a next step, arterial blood was sampled from six patients receiving TIVA for routine neurosurgical procedures by a target-controlled infusion (TCI) system, and propofol headspace determination was performed using the auto-sampler as conducted in the above experiments using spiked blood. Table 1 shows clinical data of the patient population involved in these measurements. The patients were premedicated orally with 7.5 mg midazolam and received 20–50 µg sufentanil at the beginning of induction. Then, propofol (Propofol-Lipuro® 1% and 2%) was infused by a TCI pump (Orchestra® Base Primca; Fresenius Kabi, Bad Homburg, Germany) at an initial target plasma concentration of 6 µg/ml. Intubation was facilitated with cisatracurium. After intubation, anesthesia was maintained using TCI at target plasma concentrations from 2.5 to 4.0 µg/ml. Remifentanil at a rate of 0.5 µg·kg$^{-1}$·min$^{-1}$ was used as analgesic. Propofol target plasma concentrations as selected on the TCI system were registered. Target plasma concentrations were estimated by the TCI pump with the Marsh model.
The declustering potential was set at 1100 V for thymol. The multiple reaction monitoring transitions were m/z 410.4 → 192.3, 410.4 → 330.0 for propofol and 382.4 → 148.0, 382.4 → 164.0, 382.4 → 302.2 for thymol. The day-to-day precision of the analyses was 11.3%, and the intraday repeatability was 7.5%. The limit of detection was 85 ng/ml, and the limit of quantitation was 297 ng/ml.

**Perioperative In Vivo (On-line) Measurements**

For perioperative in vivo studies, the IMR-MS system was connected to the patients via a stainless steel T piece inserted between the end of the endotracheal tube and the Y connector of the respirator. Airway gas was sampled in a side-stream mode and conveyed from the T piece to the IMR-MS system using a 2.5-m-long tube heated to 90°C made of Silcosteel® with an OD of 1.60 mm and an ID of 0.51 mm. The tube was insulated with a layer of 35-mm rubber to keep the surface temperature less than 44°C to exclude possible burn injuries. Gas sampling by the IMR-MS was continuous at 50 ml/min throughout the respiratory cycle. Propofol measurements were synchronized to the end-expiratory phase of the respiratory cycle by a second standard mass spectrometer, based on electron impact ionization, integrated into the system, which allowed continuous high-frequency measurement of expiratory oxygen and carbon dioxide (at 100 ms). The carbon dioxide and propofol signals were registered simultaneously. The last propofol measurement before the decline of the carbon dioxide signal was defined as end-expiratory.

**Standardization of Propofol Signaling**

Because our IMR-MS system was not formally calibrated with a known concentration of propofol in a gas phase, the sensitivity of the system could have varied between different measurement days. To standardize our measurements and enable statistical analyses of expiratory propofol across different patients, we used mercury ion counts as an internal standard. Mercury ions are generated out of mercury vapor during the ionization process and are registered in parallel during all IMR measurements. Mercury ion counts can be used to estimate the sensitivity of the instrument. Standardization of propofol signaling was done by dividing propofol counts...
by mercury ion counts. This results in a dimensionless variable corrected for the internal sensitivity of the instrument, which can then be correlated to propofol blood levels across different individuals.

**Statistical Analysis**

The aim of this exploratory study was to show a statistically significant relation between propofol signaling in expiratory air or in the headspace above whole blood samples measured by IMR-MS and propofol whole blood concentrations as measured by liquid chromatography ion spray tandem mass spectrometry. Linear regression was used to estimate the coefficients of the linear equation that best predicts propofol whole blood concentrations as a dependent variable from expiratory propofol signaling as an independent variable. For on-line analysis, the mean of all end-expiratory propofol counts over 1 min in the respiratory gas during blood sampling at a measured propofol whole blood concentration was used. Data were analyzed for normal distribution using the Kolmogorov-Smirnov test. All statistical calculations were performed using SPSS 14.1 (SPSS, Chicago, IL). A P value less than 0.05 was regarded as statistically significant. Data are given as mean and SD or 95% confidence interval.

**Results**

**Spiked Blood**

In these experiments, blood from five healthy volunteers was spiked with known concentrations of propofol, and propofol signaling in the headspace above the blood samples was measured. Propofol signaling correlated strongly with propofol blood concentrations in each sample. The range of the Pearson correlation coefficient varied for the propofol fragment 163 from 0.982 to 0.990, P < 0.01, and for unfragmented propofol 178 from 0.825 to 0.987, P < 0.01 (table 2 and fig. 3). Neither propofol fragment 163 nor propofol 178 signals could be detected above the 10% lipid carrier solution of propofol during control experiments (data not shown).

**Table 2. Results of Headspace Determination above Spiked Blood**

<table>
<thead>
<tr>
<th>Volunteer</th>
<th>Propofol 163</th>
<th>Propofol 178</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.989*</td>
<td>0.876*</td>
</tr>
<tr>
<td>2</td>
<td>0.982*</td>
<td>0.825*</td>
</tr>
<tr>
<td>3</td>
<td>0.990*</td>
<td>0.969*</td>
</tr>
<tr>
<td>4</td>
<td>0.989*</td>
<td>0.966*</td>
</tr>
<tr>
<td>5</td>
<td>0.987*</td>
<td>0.987*</td>
</tr>
</tbody>
</table>

Pearson correlation coefficient between propofol concentrations in spiked blood and propofol counts in the headspace above the propofol–blood solution in five healthy volunteers.

* P < 0.01.

Propofol 163 = propofol fragment with a mass of 163 Da; propofol 178 = unfragmented propofol.

**Fig. 3. Correlation between propofol signaling in the headspace above a venous blood sample and propofol concentration within the sample. For this experiment, blood from a healthy volunteer was spiked with increasing propofol concentrations (0–10 μg/ml) and propofol counts above the sample measured using the ion–molecule reaction mass spectrometry system connected to an auto-sampler. Intact propofol 178 in the headspace is represented by circles, and the propofol fragment 163 is represented by triangles. The Pearson r for the relation between blood propofol and propofol 178/propofol 163 was 0.980, P < 0.01. Dotted lines represent 95% confidence intervals.**

**Perioperative In Vitro (Off-line) Measurements**

During perioperative *in vitro* measurements, blood from patients undergoing routine neurosurgery procedures was sampled, propofol signaling in the headspace above the blood sample was determined, and propofol concentrations in the samples were measured by liquid chromatography ion spray tandem mass spectrometry. In these experiments, we found a highly significant correlation between propofol signals in the headspace above arterial blood samples and whole blood propofol concentrations. Pearson correlation coefficients ranged from 0.846 to 0.957 (n = 6) for propofol fragment 163 and from 0.841 to 0.959 for unfragmented propofol 178 (table 3). Average Pearson correlation coefficients for propofol 163 headspace counts above blood samples and propofol blood concentrations were higher than for propofol blood concentration and the selected propofol target concentration by TCI (0.938 ± 0.017 vs. 0.873 ± 0.089).

**Perioperative In Vivo (On-line) Measurements**

For perioperative *in vivo* determination of exhaled propofol, the IMR-MS system was directly connected to the endotracheal tube via a T piece, and propofol signals were registered on-line. These on-line measurements were performed in 11 patients. In addition, at predefined propofol plasma concentrations set by TCI, blood samples were drawn, and propofol concentrations were determined by liquid chromatography ion spray tandem mass spectrometry. In 6 patients from this cohort, in addition to on-line determinations, propofol headspace...
measurements above blood samples drawn at the predefined TCI levels were performed. We then correlated propofol counts in expiratory air, propofol headspace counts above blood samples, propofol levels as estimated by TCI, and measured propofol concentrations in whole blood. Table 3 summarizes these analyses of correlation.

The propofol fragment 163 in end-expiratory air correlated strongly with measured whole blood propofol levels. Pearson correlation coefficients ranged from 0.784 to 0.985. Correlation coefficients for the unfragmented propofol 178 ranged from 0.541 to 0.995 (table 3). There was also a strong intraindividual correlation between propofol off-line (headspace) counts and propofol on-line measurements for any given propofol blood concentration with average Pearson $r$ values of 0.938 ± 0.055 for propofol 168 and 0.917 ± 0.057 for propofol 178 (fig. 4).

Intraindividual correlation coefficients were higher for the relation between expiratory propofol 163 or propofol 178 signaling and propofol blood concentrations than for TCI levels and measured propofol levels (0.938 ± 0.055 for propofol 163, 0.904 ± 0.102 for propofol 178, and 0.873 ± 0.089 for TCI levels and measured blood concentrations). Linear regression between mercury ion corrected expiratory propofol signaling and propofol blood concentrations across all individuals ($n = 11, 49$ measurements) showed a correlation of $r = 0.85$ between both variables (fig. 5).

Adjustments of whole blood propofol levels by TCI led to a rapid response in propofol signaling in expired air. These changes were generally detectable within 90 s after modifications of the infusion rates were made. The system was able to detect increases of propofol blood levels of approximately 0.5 µg/ml as well as the immediate decline of blood propofol levels after stop of the infusion. Figure 6 gives an example of the response in propofol signaling in relation to changes in selected TCI concentrations.

**Discussion**

This pilot study demonstrates that molecular gas analysis by IMR-MS can be used as a noninvasive technique to monitor relative changes in plasma concentrations of propofol during TIVA in humans undergoing routine surgery. Furthermore, we showed the feasibility of detection and quantification of propofol in the headspace above blood samples containing this agent. The physicochemical properties of propofol, particularly its low water solubility, result in a measurable amount of propofol in gases in close proximity to the surface of hydrophilic solutions. Our experiments have shown that the relation between gas-phase propofol and the concentration of soluble propofol is remarkably linear. It is this linear association that could make the estimation of propofol blood levels from expiratory propofol concentrations possible, provided these findings will be reproducible in larger study samples.

There are several previous studies that have addressed this issue. Harrison et al. demonstrated propofol signaling in both expiratory air and in the headspace above serum taken from a single patient anesthetized with propofol. In this study, a proton-transfer reaction (PTR) mass spectrometer was used. Several propofol fragments
and metabolites were detected in addition to the major signal of 179 Da resulting from protonated propofol. When comparing both techniques in more detail for propofol measurements, the currently available technical layouts must be taken into consideration. PTR- and IMR-MS are generalized terms in molecular collision dynamics. PTR means the transfer of a proton onto a molecule with higher proton affinity than the incoming protonated ion. $\text{H}_2\text{O}^+$ is generally used in PTR ionization, whereas monatomic ions are used in IMR ionization. Both methods perform equally fast reactions in exothermic energy conditions and reach thereby a high sensitivity. The major differences in performance and limitations between both methods are the technical lay-

Fig. 4. Correlation between whole blood propofol concentrations as measured by liquid chromatography ion spray tandem mass spectrometry, on-line counts of the propofol 163 fragment (circles), and the corresponding off-line measurements above blood samples taken simultaneously with on-line propofol registration (triangles). Six patients were evaluated. Patient E had an unusually high number of headspace propofol 163 counts, probably due to unintended overheating of the probe during incubation. Dotted lines represent 95% confidence intervals.
out of the reaction chamber and the physical consequences thereof. The IMR instrument used in this study is operating at very low pressures (10^{-3} mbar) in the reaction chamber and hence performs true single-collision ionization processes, whereas the PTR instrument uses a high-pressure (1-mbar) multiple-collision reaction chamber. Multiple collisions of ions before their mass detection inherit a severe gas matrix dependency of the ion signal. Whenever a sample gas product ion collides with another neutral molecule with lower ionization level or higher proton affinity before it is detected, the charge and hence the molecule will be lost for detection. This fact results in the requirement to keep the background gas matrix very constant or else a complex matrix-dependent ion signal arrives at the detector. Breath gas contains some 4.5 vol% carbon dioxide and up to 5.5 vol% of water vapor next to the trace compounds under study. Although H_3O^+ ions do not directly proton-transfer to carbon dioxide, a small correction for carbon dioxide must be made as the 3-atomic gas carbon dioxide changes the diffusion coefficient in the tube and so the radial diffusion and density of the ions. H_3O^+ ions do associate with H_2O molecules readily into H_2O.H_3O^+ clusters by three-body reactions, changing thereby the original H_3O^+ ion density where trace compounds such as propofol experience a nonlinear measurement and need correction. Also, any calibration process of a trace compound must be performed with the equivalent water vapor density. Furthermore, our mass spectrometry technology allowed the addition of a second mass spectrometry system, operating in the identical sample gas environment that measured carbon dioxide at very high cycle rates of 100 ms in expiratory air. This made the identification of the end-expiratory phase and analysis of true alveolar gas possible.

A disadvantage of all mass spectrometry techniques used in gas analysis is, however, that these systems detect only mass signals (counts/s) but do not report actual concentrations (e.g., parts per billion [ppb]). Mass signals and concentrations of volatile agents in gases are strictly proportional, but to calculate concentrations for mass signals, a calibration procedure is required. This procedure could consist of analyzing a gas inert to propofol, e.g., nitrogen or synthetic air with a known concentration of propofol in the gas phase. Another way
of calibrating the mass spectrometry system is the use of a gas-phase permeation system. This system consists of an oven at constant temperature where a few milliliters of liquid propofol is enclosed in a plastic or Teflon tube. Due to the difference in partial pressures of propofol within the tube and outside the tube, a permeation process starts moving propofol molecules to the outside of the tube. As soon as a constant gas flow, created by a small pump, is moving fresh air over the tube, a dynamic equilibrium is established, and the off going gases mix with a small concentration of propofol. This permeation system is currently under development.

Because of the absence of calibration in our current study, the same concentration of propofol results in a different number of counts in different individuals and between different IMR-MS machines (but in a consistent number of counts within any given individual using the same system), and we could only report intraindividual correlations between propofol blood concentration and propofol signaling. This does only allow monitoring of relative changes in propofol blood levels. To realize regression analysis across all individuals and measurements in our study, we corrected propofol signaling for differences in the sensitivity of the instrument by using mercury ion counts. Therefore, a close correlation between corrected propofol counts and propofol blood levels in the whole sample could be demonstrated. Corrected propofol signaling will not replace the formal calibration of the system because the use of mercury ions for internal calibration results in dimensionless values but not in absolute concentrations. Therefore, the above-mentioned calibration procedure can be expected to result in a further increase in precision of propofol measurements.

Grossherr et al. recently quantified propofol in end-expiratory air in two different animal species (pigs and goats) using propofol adsorption by Tenax-TA tubes, consecutive thermal desorption, and conventional measurement by gas chromatography–mass spectrometry. This technique needs complicated and long sample preparations and does not allow on-line analysis but may be useful as a calibration procedure for other methods of alveolar propofol measurements. Comparable to our study, Grossherr et al. found a linear correlation between propofol plasma concentrations (range, 0–8 μg/ml) and propofol concentrations in alveolar gas. These authors also described marked and pronounced differences in alveolar propofol concentrations between the two different animal species with a more than 10-fold difference between pigs (range, 0–1.4 ppb) and goats (0–22 ppb). This limits the applicability of animal data to human findings. Furthermore, Grossherr et al. reported interindividual differences in propofol levels in alveolar gas samples within the same species by measuring different alveolar propofol concentrations at the same plasma propofol concentration. There exist several possibilities that could explain these findings. The permeability of the alveolocapillary membrane could significantly influence the amount of propofol in exhaled breath. The function of this diffusion barrier can be altered in disease state such as pulmonary edema or emphysema, which could affect alveolar propofol concentrations. Possible other effects may include an influence of tidal volume as well as ventilation/perfusion mismatches as a consequence of decreased functional residual capacity due to general anesthesia. To be able to characterize these different effects on exhaled propofol with our IMR-MS, the above-mentioned calibration procedure is prerequisite for direct comparison of expiratory propofol concentrations between different subjects.

In our study, we monitored unfragmented propofol with a mass of 178 Da and a propofol fragment with a mass of 163 Da, which is generated by demethylation during ionization in the IMR-MS. Although basically propofol on its mother mass 178 seems to be more suitable for on-line and off-line monitoring, our initial observation of possible interferences with low concentrations of sevoflurane led to the use of propofol 163 as a surrogate marker for gas-phase propofol. The correlations between propofol blood levels and propofol 163 were at least as close as those seen with the intact molecule.

Determination of propofol 163 and 178 by IMR-MS seems to be a sensitive and quick method. IMR-MS is capable to detect small changes of propofol whole blood levels in the range of 0.5 μg/ml reliably and within less than 90 s. Measuring propofol signals in the headspace above blood samples seems to be an alternative method to estimate propofol blood levels, albeit presumably with less accurate results.

Limitations of our study—beside the use of an arbitrary concentration unit for expiratory propofol—include the fact that we were unable to account for possible effects of pulmonary and extrahepatic metabolism of propofol, changes in ventilation/perfusion relations, or diffusion capacity across different disease states or ventilation modes. Theoretically, all of these effects could influence the relation between blood and alveolar concentrations of propofol, and these open questions must be addressed in further studies. On the other hand, our method could allow the systematic delineation of these and other effects, resulting in improved validity and reliability of this technology.

In summary, our study shows that propofol is eliminated in measurable quantities by the lung and that concentrations of propofol in expiratory air correlate strongly with blood concentrations. IMR-MS may allow the continuous and noninvasive monitoring of expiratory propofol levels in patients undergoing general anesthesia.
References


