Minimum Alveolar Concentration–Awake of Xenon
Alone and in Combination with Isoflurane or Sevoflurane

Takahisa Goto, M.D.,* Yusinori Nakata, M.D., M.B.A.,† Yoshiki Ishiguro, M.D.,‡ Yoshinari Niimi, M.D., Ph.D.,* Kunio Suwa, M.D.,§ Shigeho Morita, M.D.¶

Background: The minimum alveolar concentration (MAC)-
awake is a traditional index of hypnotic potency of an inhala-
tional anesthetic. The MAC-awake of xenon, an inert gas with
anesthetic properties (MAC = 71%), has not been determined. It
is also unknown how xenon interacts with isoflurane or
sevoflurane on the MAC-awake.

Methods: In the first part of the study, 90 female patients
received xenon, nitrous oxide (N2O), isoflurane, or sevoflurane
supplemented with epidural anesthesia (n = 36 for xenon and
n = 18 per group for other anesthetics). In the second part, 72
additional patients received either xenon or N2O combined
with the 0.5 times MAC-awake concentration of isoflurane or
sevoflurane (0.2% and 0.3%, respectively, based on the results
of the first part; n = 18 per group). During emergence, the
concentration of an assigned anesthetic (xenon or N2O only
in the second part) was decreased in 0.1 MAC decrements every
15 min from 0.8 MAC or from 70% in the case of N2O until the
patient followed the command to either open her eyes or to
squeez and release the investigator’s hand. The concentration
midway between the value permitting the response to
command and that just preventing it was defined as the MAC-
awake.

Results: The MAC-awake were as follows: xenon, 32.6 ± 6.1% (mean ± SD) or 0.46 ± 0.09 MAC; N2O, 63.3 ± 7.1% (0.61 ± 0.07
MAC); isoflurane, 0.40 ± 0.07% (0.35 ± 0.06 MAC); and sevo-
flurane, 0.59 ± 0.10% (0.35 ± 0.06 MAC). Addition of the 0.5
MAC-awake concentrations of isoflurane and sevoflurane re-
duced the MAC-awake of xenon to 0.50 ± 0.15 and 0.51 ± 0.16
times its MAC-awake as a sole agent, but that of N2O to the values
significantly greater than 0.5 times its MAC-awake as a sole
agent (0.68 ± 0.12 and 0.66 ± 0.14 times MAC-awake; P < 0.01,
analysis of variance and Dunnett’s test).

Conclusions: The MAC-awake of xenon is 33% or 0.46 times its
MAC. In terms of the MAC-fraction, this is smaller than that for
N2O but greater than those for isoflurane and sevoflurane.
Unlike N2O, xenon interacts additively with isoflurane and sevo-
flurane on MAC-awake. (Key words: Anesthetic interaction;
general anesthesia, hypnotis.)

XENON is a gaseous anesthetic that shares many charac-
teristics with nitrous oxide (N2O). For example, they are
both gaseous, have low blood–gas partition coefficients
(0.12 and 0.43 for xenon and N2O, respectively), have
minimal alveolar concentrations (MAC) at least one order
higher than those of volatile anesthetics (71% and
104%), and have good analgesic properties.

These similarities lead us to predict that xenon, like
N2O, may be weak in its hypnotic potency. Accord-
ingly, we determined the MAC-awake of xenon, a con-
centration associated with imminent arousal and a tradi-
tionally accepted index of the hypnotic potency of an
inhaled anesthetic. Furthermore, because N2O is
known to interact infraadditively with isoflurane and
sevoflurane on the MAC-awake,6,7 we sought to deter-
mine if this is also the case with xenon.

Methods and Materials

Participants

After written informed consent was obtained, 16
women aged 30–65 yr with American Society of Anes-
thesiologists physical status I or II who were scheduled
for elective total abdominal or vaginal hysterectomies
were studied according to a protocol approved by the
Institutional Human Studies Committee of Teikyo Uni-
versity. Exclusion criteria included the history or pres-
ence of neurologic diseases, ingestion of medications
known to influence anesthetic or analgesic require-
ments, and contraindications to epidural anesthesia. All
baseline laboratory values were normal.

Anesthesia Protocols

This study consisted of two parts (table 1). The first
part was intended for determination of the MAC-awake
of two gaseous anesthetics, xenon and N2O, and two
volatile agents, isoflurane and sevoflurane. The second
part was designed to characterize the interactions be-
tween the gaseous and the volatile agents by examining
whether, as predicted from simple additivity, the 0.5
MAC-awake concentrations of volatile agents would re-
duce the MAC-awake of xenon or N2O by half when
administered together.

For both parts of the study, the unpremedicated pa-
tients had an epidural catheter placed at the L2–L3 in-
terspace and received 10 ml of 1.5% mepivacaine with
1:200,000 epinephrine after a 3-ml test dose. If the sen-
sory block level of T10 or higher to pin pricks was not
obtained within 15 min, the epidural catheter was
judged to be functioning inadequately, and the patient
was not included in the study.

In the first part, 90 patients were randomly assigned to

* Associate Professor, † Assistant Professor, § Professor, ¶ Professor and Chair-
man, Department of Anesthesia, Teikyo University School of Medicine, Ichihara
Hospital. ‡ Associate Professor, Departments of Health Economics and Anes-
thesia, Teikyo University School of Medicine, Tokyo.

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Address reprint requests to Dr. Goto: Department of Anesthesia, Teikyo
University Ichihara Hospital, 3426-3 Anesaki, Ichihara-shi, Chiba 299-0111 Japan.
Address electronic mail to: takigoto-med.teikyo-u.ac.jp. Individual article re-
receive one of the four anesthetics (n = 36 for xenon and n = 18 for other anesthetics; table 1). The patients assigned to xenon or isoflurane were induced with propofol 2.5 mg/kg intravenously. After the larynx and upper trachea were topically sprayed with 4% lidocaine 3 ml, the trachea was intubated with the aid of vecuronium 10 mg administered intravenously. Anesthesia was maintained using xenon 56% (0.8 MAC) in oxygen via a closed breathing system, or with 1.0–1.5% isoflurane (all concentrations of inhalational anesthetics are end-tidal unless otherwise specified). For those receiving sevoflurane or N2O, anesthesia was induced with an inhalation of 5% inspired concentration of sevoflurane. After tracheal topicalization and intubation, they received 1.2–2.0% sevoflurane or 70% N2O (plus approximately 0.5% sevoflurane to prevent intraoperative awareness) as assigned.

For at least 15 min before the end of surgery, the end-tidal concentrations of isoflurane and sevoflurane were maintained at 0.92% and 1.4%, respectively (both approximately 0.8 MAC). The supplemental sevoflurane in the N2O patients was discontinued at least 30 min before the end of surgery and was washed out using a 10-l/min fresh flow of 70% N2O in oxygen so that no more than 0.01% residual sevoflurane was detected by the analyzer during the last 10 min.

In the second part, 72 patients were randomly assigned to receive either xenon or N2O plus 0.2% isoflurane or 0.3% sevoflurane (n = 18 per group; table 1). The concentrations of isoflurane and sevoflurane were chosen to match 0.5 times their respective MAC-awake based on the results of the first part. Induction of anesthesia was achieved by an inhalation of 5% inspired concentration of sevoflurane for the N2O-plus-sevoflurane group and intravenous propofol 2.5 mg/kg for the other three groups. Subsequently, xenon 56% or N2O 70% with isoflurane 0.2% or sevoflurane 0.3% were administered until the end of surgery.

The end-tidal concentrations of carbon dioxide, N2O, isoflurane, and sevoflurane were measured using an infrared analyzer (Capnomac Ultima; Datex, Helsinki, Finland). For those who received xenon as a sole agent, an in-line infrared capnogram (Hewlett Packard, Waltham, MA) was used instead. The end-tidal concentration of xenon was continuously monitored using a xenon analyzer (Anzai Medical, Tokyo, Japan), the effective working range of which was 1–100% with the error ±1% and the 90% response time less than 1 s. These analyzers were calibrated before each use according to the manufacturers’ instructions.

All patients also received a continuous epidural infusion of 1.5% mepivacaine with 1:200,000 epinephrine at 5–10 ml/h to maintain mean arterial pressure and heart rate within 20% of the preoperative values. The lungs were mechanically ventilated to maintain the end-tidal concentration of carbon dioxide at 30–35 mmHg, and additional doses of vecuronium were administered as necessary. The body temperature measured with an esophageal sensor was maintained with the use of a warming mattress placed on the operating table and by warming the intravenous fluids.

**Determination of MAC-awake**

Shortly before the end of surgery, all the patients received neostigmine 2.5 mg and atropine 1.0 mg intravenously to reverse residual neuromuscular blockade, and the recovery was verified by the train-of-four response to the ulnar nerve stimulation. When surgery was completed, a designated investigator blinded to the anesthetics administered asked the patient in a normal tone to open her eyes and then to squeeze and release the investigator’s hand. If the patient failed to follow both commands, the end-tidal concentration of anesthetic was reduced by the primary anesthetist in a decrement of approximately 0.1 MAC (7% for xenon, 10% for N2O, 0.12% for isoflurane, and 0.2% for sevoflurane; table 1), and the new concentration was maintained for 15 min. During the second part of the study, the concentrations of isoflurane and sevoflurane were kept constant at 0.2% and 0.3%, respectively, whereas those of xenon and N2O were reduced (table 1). In all patients, commands were given every 5 min and when clinical signs of impending arousal such as spontaneous movements or coughs were noted. This process was repeated until an alveolar con-
centation was reached at which the patient first responded to either one of the commands (eye opening or hand squeezing and releasing). As in previous studies, the concentration midway between the value permitting the first response to command and that just preventing it was defined as the MAC-awake. Mechanical ventilation was continued during the entire wake-up period. When coughing or bucking hindered effective ventilation with positive pressures, the ventilator was stopped to avoid excessive elevations in the airway pressure. Care was taken to minimize stimuli to the patients except that, in the last 15 patients in each anesthetic treatment group, we recorded the midlatency auditory evoked potentials (MLAEPS) immediately before each testing of the responsiveness to verbal commands (manuscript in preparation). For each recording, we applied 500 click stimuli of 0.1 ms at 80 dB at the frequency of 5 Hz binaurally using earphones.

The patients received no medication other than those previously stated until the end of the MAC-awake measurement. Fifteen minutes after extubation, the epidural block level to pin pricks was examined, and the patient was asked to rate her incisional pain using a 0–10 verbal rating scale, with 0 and 10 being no pain and the worst pain imaginable, respectively.

Statistical Analysis
Because two of the 18 patients in the N₂O group responded to verbal command at the highest concentration tested (70%), the MAC-awake of N₂O was calculated as in a previous study by assuming that these patients would not have responded at 80%. This assumption is based on the literature demonstrating that responses to verbal command occurred only occasionally with 80% inspired concentration of N₂O, and only 1 in 50 patients responded at 86%. In addition, we performed a logistic regression analysis to calculate the concentration of N₂O to suppress responses to verbal command in 50% of the patients (ED₅₀). This was to confirm our MAC-awake value for N₂O with an analysis that required no assumption and therefore better accounted for the two patients who responded at 70% N₂O.

To express the MAC-awake as the fraction of MAC, the MAC values of isoflurane and sevoflurane were adjusted for age in each individual patient based on the following equation:

\[
\text{MAC} = 1.18 (\text{isoflurane}) \text{ or } 1.80 (\text{sevoflurane})
\times 10 - 0.00269 (\text{age in years} - 40) (\%)
\]

The MAC of xenon was taken as 71% for all the patients because this is the only value reported in the literature and because the age of the subjects studied was similar (46 ± 6 in this study vs. 43 ± 17 in the study by Cullen et al., mean ± SD). Likewise, the MAC for N₂O was taken as 104% because this is the only reported value determined by the direct measurement although the subjects studied were somewhat younger than our patients (21–35 yr).

Results are reported as mean ± SD, mean and 95% confidence interval, or median and range as appropriate. To test whether the MAC-awake of xenon and N₂O obtained in the presence of 0.5 times the MAC-awake of isoflurane or sevoflurane were different from 0.5 times their respective MAC-awake as a sole agent, the results for xenon and N₂O from the first part were halved and compared with the results of the second part using analysis of variance and Dunnett’s test, with the former as a control. To test whether application of click stimuli for the recording of the MLAEPS affected the MAC-awake, the results of the patients in the xenon group who did and did not undergo the recording (n = 15 and 21, respectively) were compared using the unpaired t test. Other data were analyzed using single-factor factorial analysis of variance and the Student-Newman-Keuls tests or Kruskal-Wallis tests as appropriate. A P value less than 0.05 was considered statistically significant.

Results
The eight anesthetic treatment groups were comparable with respect to patient demographics and other postoperative data listed in table 2.

### Table 2. Patient Demographics and Postoperative Data

<table>
<thead>
<tr>
<th></th>
<th>Xenon</th>
<th>Iso</th>
<th>Sevo</th>
<th>N₂O</th>
<th>Xe + Iso</th>
<th>Xe + Sevo</th>
<th>N₂O + Iso</th>
<th>N₂O + Sevo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>46 ± 8</td>
<td>43 ± 8</td>
<td>47 ± 9</td>
<td>47 ± 8</td>
<td>47 ± 5</td>
<td>45 ± 6</td>
<td>46 ± 8</td>
<td>43 ± 5</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>155 ± 6</td>
<td>157 ± 5</td>
<td>156 ± 3</td>
<td>154 ± 5</td>
<td>154 ± 5</td>
<td>158 ± 6</td>
<td>158 ± 6</td>
<td>156 ± 4</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>56 ± 7</td>
<td>59 ± 7</td>
<td>59 ± 11</td>
<td>55 ± 7</td>
<td>54 ± 6</td>
<td>57 ± 10</td>
<td>54 ± 6</td>
<td>54 ± 6</td>
</tr>
<tr>
<td>Anesthesia time (min)</td>
<td>119 ± 39</td>
<td>148 ± 59</td>
<td>120 ± 30</td>
<td>130 ± 49</td>
<td>146 ± 48</td>
<td>135 ± 55</td>
<td>140 ± 46</td>
<td>127 ± 40</td>
</tr>
<tr>
<td>Body temperature (°C)</td>
<td>35.9 ± 0.1</td>
<td>35.9 ± 0.2</td>
<td>35.9 ± 0.1</td>
<td>35.9 ± 0.1</td>
<td>35.8 ± 0.1</td>
<td>35.9 ± 0.2</td>
<td>35.9 ± 0.2</td>
<td>36.0 ± 0.1</td>
</tr>
<tr>
<td>Epidural dose (ml)</td>
<td>31 ± 9</td>
<td>30 ± 7</td>
<td>27 ± 6</td>
<td>31 ± 9</td>
<td>31 ± 7</td>
<td>28 ± 6</td>
<td>30 ± 6</td>
<td>29 ± 9</td>
</tr>
<tr>
<td>Pain rating</td>
<td>2 (0–5)</td>
<td>2 (0–5)</td>
<td>2 (0–5)</td>
<td>2 (0–4)</td>
<td>2 (0–4)</td>
<td>2 (0–5)</td>
<td>2 (0–5)</td>
<td>2 (0–5)</td>
</tr>
</tbody>
</table>

The body temperature was recorded at the time of the first response to verbal command. The epidural dose is the total volume of 1.5% mepivacaine with 1:200,000 epinephrine administered until 15 min after extubation. The pain rating is the numerical quantification of incisional pain by the patient using a 0–10 verbal rating scale, with 0 and 10 being no pain and the worst pain imaginable, respectively. Data are mean ± SD, or median (range).

Xe = xenon; Iso = isoflurane; Sevo = sevoflurane; N₂O = nitrous oxide.
Table 3. MAC-awake Values of Anesthetics Administered Alone

<table>
<thead>
<tr>
<th>Anesthetic</th>
<th>Xenon (± SE)</th>
<th>N2O (± SE)</th>
<th>Isoflurane (± SE)</th>
<th>Sevoflurane (± SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAC-awake (%)</td>
<td>32.6 ± 6.1</td>
<td>63.3 ± 7.1</td>
<td>0.40 ± 0.07</td>
<td>0.59 ± 0.10</td>
</tr>
<tr>
<td>(95% CI)</td>
<td>(30.5–34.6)</td>
<td>(59.8–66.9)</td>
<td>(0.37–0.43)</td>
<td>(0.54–0.64)</td>
</tr>
<tr>
<td>MAC-awake/MAC</td>
<td>0.46 ± 0.09</td>
<td>0.61 ± 0.07</td>
<td>0.35 ± 0.06</td>
<td>0.35 ± 0.06</td>
</tr>
<tr>
<td>(95% CI)</td>
<td>(0.43–0.49)</td>
<td>(0.57–0.64)</td>
<td>(0.32–0.38)</td>
<td>(0.32–0.38)</td>
</tr>
</tbody>
</table>

The minimum alveolar concentration (MAC)-awake/MAC ratios were significantly different between anesthetic groups (P < 0.001, analysis of variance and Student-Newman-Keuls tests), except between isoflurane and sevoflurane. N2O = nitrous oxide.

Table 3 demonstrates the MAC-awake values and their MAC-fractions of four anesthetics studied. The MAC-awake expressed in fractions of MAC were significantly different between isoflurane and sevoflurane. The MAC-awake for xenon in the patients who did and did not undergo the recording of the MLAEPs were 31.3 ± 5.0% and 33.5 ± 6.7% (NS), respectively, suggesting that click stimuli used to elicit the MLAEPs did not affect the results. The MAC-awake for N2O (63.3%) was almost identical to the ED50 derived using a logistic regression analysis (62.7%), confirming that our MAC-awake value for N2O was close to the true value despite our assumption that the two patients who responded while breathing 70% N2O would not have responded at 80%.

Table 4 demonstrates the MAC-awake values of xenon and N2O when administered together with the 0.5 MAC-awake concentrations of isoflurane or sevoflurane. The values for N2O were significantly greater than 0.5 times its MAC-awake determined when used as a sole agent in the first part (P < 0.01), suggesting infraadditive interactions between N2O and these volatile anesthetics. In contrast, the values for xenon were equal to 0.5 times its MAC-awake as a sole agent, consistent with simple additivity.

**Discussion**

The MAC-awake of xenon was 33%. In terms of the MAC fraction, the MAC-awake for xenon (0.46 MAC) was smaller than that for N2O (0.61 MAC) but greater than those for isoflurane and sevoflurane (both 0.35 MAC). We have also demonstrated that xenon interacted additively with both isoflurane and sevoflurane on MAC-awake, whereas N2O was slightly infraadditive.

Several issues need to be addressed when considering the validity of our results. The first issue is the impacts of epidural analgesia on the MAC-awake. We used epidural analgesia from the intraoperative period because of our fear that, without it, the extremely low blood-gas partition coefficient of xenon (0.12) would have allowed the patient to perceive pain immediately after the MAC-awake measurement was finished and xenon was discontinued. To our knowledge, only two previous studies conducted by the same group of investigators, have specifically examined the effect of epidural analgesia on MAC-awake. Although both reported 30–40% reductions in the MAC-awake of isoflurane by epidural analgesia, the general applicability of these results may be questioned because the values determined without epidural analgesia (i.e., regular MAC-awake, 0.30–0.32%) were markedly lower than those reported by others (e.g., 0.36, 0.41, and 0.4411**). Furthermore, one of these studies used nonverbal stimuli (music) to awaken the patients, which is atypical.

In contrast, close proximity of our MAC-awake values (0.40% for isoflurane and 0.59% for sevoflurane) to those determined previously without using epidural analgesia (0.36–0.44% for isoflurane10,11,15 and 0.62–0.67% for sevoflurane7,10,15,17,18) strongly suggests that epidural analgesia only minimally affects the MAC-awake. However, because this is based on indirect comparisons, the true magnitude of the effect remains to be elucidated.

The second issue is whether the different anesthetic induction techniques we used (propofol vs. sevoflurane) affected our results. We used inhalational induction in the sevoflurane group, as was used in previous studies, because, as previously described, we wished to estimate the effect of epidural analgesia on the MAC-awake by comparing our results with those of the previous investigators.

We believe that propofol used for induction only minimally affected our results. The pharmacokinetic calculation†† using the parameters by Schnider et al. predicts that, in a patient who is 155 cm tall and weighs 55 kg (close to the average values of our patients), a 2.5-mg/kg bolus administration of propofol would produce a plasma concentration of 0.06 μg/ml in 2 h. Because the plasma concentration associated with a 50% chance of awakening is approximately 3 μg/ml,20,21 0.06 μg/ml is equivalent to 0.02 (= 0.06/3) times MAC-awake. Assuming additivity between propofol and xenon or isoflurane, the true MAC-awake values are estimated to be 33.3% [= 32.6/(1 – 0.02)] for xenon and

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*Because this investigation11 studied young humans (age, 22–30 yr) whereas others (including the present study) investigated older subjects (age = 40 yr), the value was age-adjusted by multiplying the reported MAC-awake/MAC by the MAC of isoflurane for the latter age range (1.15%).

†† The computer software used for this calculation was obtained courtesy of Dr. Steven L. Shafer, Department of Anesthesia, Stanford University, Stanford, California.
Table 4. MAC-awake of Xenon and N₂O Administered in Combination with the 0.5-MAC-awake Concentrations of Isoflurane or Sevoflurane

<table>
<thead>
<tr>
<th></th>
<th>+Isoflurane</th>
<th>+Sevoflurane</th>
<th>Value Predicted Assuming Additivity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Xenon (%)</strong></td>
<td>16.3 ± 4.9</td>
<td>16.7 ± 5.3</td>
<td>16.3 ± 3.0</td>
</tr>
<tr>
<td>(Fraction of MAC-awake as a sole agent)</td>
<td>(0.50 ± 0.15)</td>
<td>(0.51 ± 0.16)</td>
<td>(0.5)</td>
</tr>
<tr>
<td><strong>N₂O (%)</strong></td>
<td>43.0 ± 7.7</td>
<td>41.7 ± 8.1</td>
<td>31.7 ± 3.5</td>
</tr>
<tr>
<td>(Fraction of MAC-awake as a sole agent)</td>
<td>(0.68 ± 0.12)</td>
<td>(0.66 ± 0.14)</td>
<td>(0.5)</td>
</tr>
</tbody>
</table>

The values predicted assuming additivity were calculated by multiplying the minimum alveolar concentration (MAC)-awake of xenon and nitrous oxide (N₂O) presented in Table 3 by 0.5 because the assumption of additivity between xenon or N₂O and isoflurane or sevoflurane predicts that the addition of the 0.5 MAC-awake concentrations of these volatile anesthetics would halve the MAC-awake of xenon and N₂O. These predicted values were then compared to those determined in the presence of isoflurane or sevoflurane.

*P < 0.01 versus the value predicted assuming additivity, analysis of variance and Dunnett test, indicating infraadditivity between N₂O and isoflurane or sevoflurane.

The results regarding the interaction between xenon and the volatile anesthetics isoflurane and sevoflurane on MAC-awake are consistent with simple additivity. In contrast, N₂O was infraadditive with these volatile anesthetics, confirming the results of other investigators. However, this MAC value has been questioned24 because it is based on a single study published more than 30 years ago without the use of a xenon concentration monitor.

We have estimated the MAC of xenon as 63.1% in patients of similar age as those in this study (Nakata et al., submitted). If this value is correct, the MAC-awake for xenon would be 0.52 MAC (95% confidence interval, 0.48–0.55 MAC). This is again significantly smaller than that of N₂O and is greater than those for isoflurane and sevoflurane (P < 0.001). Interestingly, this is equal to those reported for halothane or methoxyflurane (both 0.52 MAC).9

Our results regarding the interaction between xenon and the volatile anesthetics isoflurane and sevoflurane on MAC-awake are consistent with simple additivity. In contrast, N₂O was infraadditive with these volatile anesthetics, confirming the results of other investigators.6,7 Because an eye-opening on verbal command is a supraspinally mediated response, these findings lead us to speculate that xenon and N₂O might differ in their effects on the supraspinal neural activities. Several lines of evidence indirectly support this notion. For example, xenon slows the electroencephalogram,25 whereas N₂O increases the fast-wave components26 and opposes the isoflurane-induced burst suppression.27 Moreover, xenon reduces the cerebral metabolic rate,28 whereas N₂O increases it in specific regions of the brain.29 A clinical implication of the MAC-awake value of xenon (33%) is that xenon may be more likely to produce hypnosis at clinically used concentrations than would N₂O, the MAC-awake of which is 63%. In fact, 49% xenon prevented responses to verbal command in all of our 36 patients. This predicts that, at the confidence limit of 95%, less than 8% of the population would respond at the end-tidal concentration of 49% xenon. However, many clinicians would find an 8% risk unacceptable, and the number of the patients we studied is too small to draw any firm conclusion as to what concentration of xenon is needed to ensure adequate hypnosis in a majority of patients.

Finally, the hypnotic potency of xenon we demonstrated may offer a potentially valuable clinical utility for
this gas; it may be useful in patients who are hemodynamically too unstable to tolerate other anesthetics and are therefore at high risk of intraoperative awareness. There is evidence that xenon only minimally depresses the cardiovascular system in healthy humans\(^3^0,3^1\) and in animals with cardiac dysfunction.\(^3^2\) Furthermore, xenon does not alter the function of the isolated heart or major cation currents in the cardiac myocyte.\(^3^3\) The combination of hypnotic actions and a lack of significant hemo-
dynamic depression in one agent may make xenon a unique choice in our armamentarium of anesthetics because most currently available hypnotics are potent cardio-
vascular depressants.

Xenon was provided by Daido Hoxan, Inc., Tokyo, Japan.

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