Electroencephalography offers the opportunity to measure electrical activity of the cortical gray matter that originates from excitatory or inhibitory postsynaptic potentials of pyramidal neurons via surface electrodes placed on the skull.1–3 The interpretation of unprocessed EEG data in a clinical setting is difficult because evaluation requires time and knowledge.4 Therefore, quantitative variables, such as SEFs, MF, and relative power in various EEG frequency bands, are potentially useful for monitoring patterns in humans.
but are not reliable indicators of arousal when used alone.\(^5\)

Several specific anesthesia monitors with inherent algorithms have been developed in human medicine, providing easily readable measurements for anesthetic monitoring.\(^5\) One multivariate analysis algorithm of the EEG is based on spectral and autoregressive parameters as well as entropy factors that lead to identifying predefined EEG patterns.\(^6\) Six EEG stages (A [awake] to F [increasing burst suppression pattern to isoelectricity]) with 15 substages and a corresponding EI (from 100 [awake] to 0 [isoelectricity]) can be defined from these patterns.\(^5\) Little information is available on the usefulness of this EI in veterinary anesthetic monitoring. In a clinical setting, the EI can be used to differentiate reliably between excessively deep and moderate anesthetic depth in dogs but not between moderate and inadequately light anesthesia.\(^a\)

All EEG patterns and variables are affected by anesthetic depth, anesthetic agent used, adjuvant drugs administered, and physiologic changes such as hypothermia and hypoperfusion.\(^7\) Assessment of these influences has become increasingly important because the brain is the target of anesthesia.\(^8\) The purpose of the study reported here was to compare and analyze the effects of 3 anesthetic protocols, supramaximal nociceptive stimulation, and 3 isoflurane MAC multiples on selected quantitative EEG variables in dogs.

**Materials and Methods**

Dogs—Six adult university-owned Beagles (4 females and 2 castrated males) with a mean ± SD body weight of 16.3 ± 1.0 kg and a mean age of 4.0 ± 2.7 years were selected for the study. The dogs were housed in separate kennels and were fed commercial dry adult maintenance dog food.\(^b\) They were considered healthy on the basis of physical examination, hematologic, and blood biochemical findings. All dogs were vaccinated and received anthelmintics on a regular basis. Food but not water was withheld for 6 to 8 hours prior to each anesthetic session. The study protocol was approved by the Animal Care and Use Committee of the local district government of Lower Saxony, Germany.

Experimental protocol—Each dog underwent 3 anesthetic sessions with a washout period of at least 1 week between the sessions: first isoflurane alone, then ID and IR in random order. In all sessions, anesthesia was induced with 5% isoflurane in oxygen (5 L/min) via a face mask until endotracheal intubation was possible. In the ID session, dogs were given a loading dose of dexmedetomidine\(^c\) (3 \(\mu\)g/kg, IV) delivered via a syringe pump\(^d\) over 10 minutes. This loading dose was followed by induction and maintenance of anesthesia with isoflurane, which was combined with a CRI of dexmedetomidine (3 \(\mu\)g/kg/h, IV).\(^e\) In the IR session, a remifentanil\(^f\) CRI (18 \(\mu\)g/kg/h, IV)\(^g\) was started without a loading dose and was followed by induction and maintenance of isoflurane anesthesia. Both drugs used for CRI were diluted in saline (0.9% NaCl) solution.\(^h\)

Once anesthesia was induced, dogs were endotracheally intubated, and the endotracheal tube was connected to a circle breathing system\(^i\) operated in a semi-closed mode with an oxygen flow rate of 1 L/min. Each dog was positioned in right lateral recumbency and was mechanically ventilated\(^j\) with settings adjusted to maintain eupnoea (35 to 45 mm Hg). Esophageal temperature was kept constant (within 0.5°C of 37.6°C) with a warm air blanket.\(^k\) An indwelling IV catheter\(^l\) was placed in a cephalic vein, and balanced electrolyte solution\(^m\) was infused at 5 mL/kg/h with a volumetric pump.\(^n\) Arterial blood pressures were invasively measured via an arterial catheter\(^o\) that was placed in a dorsal pedal artery and connected to a precalibrated pressure transducer\(^p\) via noncompliant pressure lines. The level of the sternal manubrium was used as the zero reference point. An instrumentation and stabilization period of at least 1 hour was allowed. During that period, anesthesia was maintained at the expected ET\(_{50}\) of 1.0 MAC. The eyes were lubricated\(^q\) repeatedly during the experiment. An attempt was made to use fluids to maintain mean arterial blood pressure > 60 mm Hg.\(^r\)

**Sample collection**—Arterial blood samples were collected periodically into heparinized syringes, connected to esophageal temperature, and analyzed\(^s\) for blood gas concentrations. Ventilator settings were adjusted to maintain eupnoea. Expired air samples for the analysis of ET\(_{50}\) and end-tidal CO\(_2\) concentration were collected from the tracheal end of the endotracheal tube. The samples were constantly analyzed through an infrared technique of a multiparameter anesthesia monitor\(^t\) that was calibrated with a reference gas mixture\(^e\) containing 3% CO\(_2\), 33% N\(_2\)O, 2% desflurane, and N\(_2\) before each experiment.

Peripheral arterial oxygen saturation was monitored via the pulse oximetry function of the same anesthesia monitor. Heart rate was recorded via ECG.\(^u\) For a single-channel EEG registration,\(^w\) 3-needle electrodes were placed subcutaneously. The 2 recording and deriving electrodes were placed bilaterally midway between the lateral canthi of the eyes and between the ears, and the reference electrode was placed on the bridge of the nose.\(^x\) The impedance of the electrodes was checked automatically and did not exceed 6 kΩ.

**Nociceptive stimulation**—For nociceptive stimulation, 2 stimulating electrodes\(^y\) were placed subcutaneously over the middle third of the medial aspect of the ulna of the right thoracic limb approximately 4 to 5 cm apart (one proximal and the other distal). The electrodes were connected to a square pulse stimulator\(^z\) that was set at a voltage of 50 V, frequency of 50 Hz, and duration of 10 ms/stimulus.

**MAC determination**—Standardized MACs were achieved by obtaining individual MAC determinations for each dog within each anesthetic session. The investigator (AMK) made each determination. The supramaximal electrical stimulation protocol, which involved a square pulse stimulator\(^z\) and a visual evaluation of motor responses, consisted of 2 single stimuli and 2 continuous stimuli (applied over 3 seconds), with 5-second pauses between all 4 stimuli.\(^{10}\) A positive motor reaction was defined as gross purposeful movement of the head, limbs, or tail. A negative motor reaction…
was defined as breathing, swallowing, or chewing. For each concentration of ET10-11, a 15-minute equilibration period was allowed. If the individual MAC, the bracketing study design was applied. The MAC was calculated as the arithmetic mean of the ET10-11 that just permitted and just prevented movement after supramaximal stimulation. In addition to 1.0 MAC, the anesthetic concentrations of 0.75 and 1.5 MAC were achieved, and the same protocol as for MAC determination was used for nociceptive stimulation at these anesthetic depths.

EEG measurements—The EEG signal was recorded continuously at 128 samples/s with a 12-bit resolution. Filter settings of the amplifier were set at 0.5 to 45 Hz and were combined with a supplemental 50-Hz notch filter. Fast Fourier transformation of 2-second segments was performed automatically, and measurements were provided as means of 10 consecutive 2-second segments (20-second epochs). Frequency bands were defined as δ = 0.5 to 3.5 Hz, θ = 3.5 to 7.5 Hz, α = 7.5 to 12.5 Hz, and β = > 12.5 Hz. Recorded data were classified as if derived from a 35-year-old human.

Prior to EEG analysis, the raw EEG was visually evaluated for artifacts. Periods with EMG activity (areas visually and automatically identified) and burst suppression pattern were included in the EEG analysis. The EI, frequency bands (δ, θ, α, and β) and their ratios (θ/δ, α/δ, and β/δ), 95% SEF, and MF were derived automatically by the EEG monitor and reported offline. Prestimulation values for each anesthetic session and each MAC were derived from up to 1 minute before start of the stimulation. Poststimulation values were recorded directly after the end of the stimulation.

After completion of each anesthetic session, all catheters were removed. The dogs were allowed to recover and received a bolus injection of carprofen (4 mg/kg, SC).

Statistical analysis—Data are reported as mean ± SD, unless otherwise indicated. The signed rank test was used to compare quantitative EEG data before and after nociceptive stimulation and among different anesthetic depths. Spearman rank correlation coefficients (r) and linear regression were used to compare all variables among the various MAC multiples. Values of P < 0.05 were considered significant.

Results

Blood gas analysis results and oxygen saturation as determined by pulse oximetry remained within clinically accepted ranges during all 3 anesthetic sessions in the 6 Beagles. At 1.5 times the MAC, hypotension (mean arterial blood pressure, 50 to 60 mm Hg) was detected in 3 dogs during the isoflurane-alone session and in 1 other dog during the IR session.

The 95% SEF before noxious stimulation was significantly correlated with increasing MAC multiples when dogs received isoflurane alone (r = –0.59; P = 0.010) and during the ID session (r = –0.82; P < 0.001) but not during the IR session (r = –0.22; P = 0.387; Figure 1). The EI decreased with increasing anesthetic depth when dogs received isoflurane alone (r = –0.89; P < 0.001); the correlation of the EI with MAC multiples was lower during the ID (r = –0.71; P < 0.001) and IR (r = –0.13; P = 0.590) sessions (Figure 2). The internal EEG algorithm was unable to calculate the EI in 2 recordings during the isoflurane-alone session as well as in 3 recordings during the IR session. Additional correlations involving other EEG variables were summarized (Table 1).
Significant differences in prestimulation median values among MAC multiples of the different anesthetic sessions were mainly evident for 95% SEF and were summarized for the quantitative EEG variables ($\delta$, $\theta$, $\alpha$, $\beta$, $\gamma$, and 95% SEF) infrequently differed significantly among the 3 anesthetic sessions at 1.5 times the isoflurane MAC, whereas significant differences were evident between prestimulation and poststimulation frequency band patterns (data not shown).

Nociceptive stimulation resulted in significant increases in $\beta$ band presence, $\beta$, MF, and 95% SEF and a significant decrease in $\delta$ band presence at 0.75 and 1.0 times the MAC during the ID session. At 1.5 times the MAC, $\delta$ band presence decreased significantly, whereas significant increases were evident in $\theta$ and $\alpha$ band presence, $\theta$, $\alpha$, $\delta$, $\beta$, and MF during the ID session (Table 3). During the IR session, the presence of $\delta$ and $\alpha$ bands decreased significantly at 0.75 times the MAC with stimulation, whereas 95% SEF increased significantly at 1.0 times the MAC (Table 4).

Mean ± SD values for 1.0 times the isoflurane MAC for the isoflurane-alone, ID, and IR sessions were 1.7 ± 0.3%, 1.0 ± 0.1%, and 1.0 ± 0.1%, respectively. Only during the isoflurane-alone session was a burst suppression pattern, with activity bursts of up to 10 seconds, present in 33% (1.5 times the MAC) and 17% (1.0 times the MAC) of prestimulation EEG recordings as well as in 17% (0.75 times the MAC) of poststimulation recordings. In 25% (isoflurane), 50% (ID), and 3% (IR) of all EEG recordings, EMG activity was present, which prevailed at lower MACs and increased with stimulation.

Table 1—Spearman rank correlations of quantitative EEG variables with isoflurane MACs for 6 Beagles that were anesthetized with isoflurane alone, with isoflurane and a CRI of dexmedetomidine (3 $\mu g/kg/h$, IV), and with isoflurane and a CRI of remifentanil (18 $\mu g/kg/h$, IV).

<table>
<thead>
<tr>
<th>Variable</th>
<th>0.75 MAC</th>
<th>1.0 MAC</th>
<th>1.5 MAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\delta$ (rel%)</td>
<td>59.51 (66.77–70.63)</td>
<td>57.93 (53.53–62.16)</td>
<td>55.87 (51.67–60.08)</td>
</tr>
<tr>
<td>$\theta$ (rel%)</td>
<td>17.06 (16.01–22.07)</td>
<td>25.20 (19.58–29.96)</td>
<td>25.76 (16.02–43.38)</td>
</tr>
<tr>
<td>$\alpha$ (rel%)</td>
<td>9.00 (6.50–12.68)</td>
<td>13.45 (7.51–17.91)</td>
<td>12.62 (5.61–27.66)</td>
</tr>
<tr>
<td>$\beta$ (rel%)</td>
<td>7.65 (7.53–13.67)</td>
<td>15.15 (14.16–15.16)</td>
<td>12.39 (9.50–18.89)</td>
</tr>
<tr>
<td>MF (Hz)</td>
<td>3.00 (2.00–4.00)</td>
<td>11.90 (8.00–17.00)</td>
<td>3.75 (2.50–6.50)</td>
</tr>
</tbody>
</table>

| 95% SEF (Hz) | 15.80 (13.00–20.50) | 33.25* (29.00–40.00) | 25.25 (13.50–33.00) |

$\rho$ = Relative percentage.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Prestimulation</th>
<th>Poststimulation</th>
<th>Prestimulation</th>
<th>Poststimulation</th>
<th>Prestimulation</th>
<th>Poststimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\delta$ (rel%)</td>
<td>51.41 (35.27–61.32)</td>
<td>14.15* (7.99–26.39)</td>
<td>69.58 (50.28–76.61)</td>
<td>18.51* (16.40–30.50)</td>
<td>68.85 (57.85–73.70)</td>
<td>50.75* (27.83–62.95)</td>
</tr>
<tr>
<td>$\alpha$ (rel%)</td>
<td>12.89 (10.00–14.12)</td>
<td>7.84* (5.62–10.05)</td>
<td>8.11 (5.66–14.78)</td>
<td>25.80* (16.40–48.02)</td>
<td>8.34 (6.28–14.84)</td>
<td>14.93 (12.60–29.06)</td>
</tr>
<tr>
<td>MF (Hz)</td>
<td>3.50 (2.50–5.50)</td>
<td>19.00* (7.50–70.00)</td>
<td>2.50 (2.00–3.50)</td>
<td>10.75* (5.50–15.50)</td>
<td>2.50 (2.00–3.00)</td>
<td>3.75* (3.00–5.00)</td>
</tr>
</tbody>
</table>

95% SEF (Hz) | 19.75 (16.00–25.50) | 34.25* (31.50–38.00) | 14.00 (12.00–24.00) | 24.00* (18.50–30.50) | 12.25 (10.50–13.50) | 13.25 (12.50–13.50) |

Table 2—Median (range) changes in quantitative EEG variables with isoflurane MAC multiples and nociceptive stimulation in 6 Beagles anesthetized with isoflurane alone.

Table 3—Median (range) changes in quantitative EEG variables with isoflurane MAC multiples and nociceptive stimulation in 6 Beagles during the ID anesthesia session.
Discussion

The purpose of the present study was to compare and analyze the effects of 3 anesthetic protocols, supramaximal nociceptive stimulation, and 3 isoflurane MAC multiples on the quantitative EEG variables, δ, θ, α, and β frequency band presence; θ; δ; α; δ; β; δ; MF; 95% SEF; and EI in dogs. Cerebrocortical activity differed among the 3 anesthetic protocols at identical MAC multiples. The strongest depression of brain activity with deepening of anesthesia was evident when dogs received isoflurane alone.

Humans and dogs anesthetized with isoflurane reportedly have a concentration-dependent transient EEG activation (desynchronization), followed by EEG slowing, burst suppression pattern, and finally isoelectricity. In dogs anesthetized with isoflurane alone, an EEG burst suppression pattern was noticed most frequently at 1.5 MAC, which is in agreement with observations in humans. Thus, isoflurane exerts a strong dose-dependent hypnotic effect, which was also evident in dogs in the present study.

Significant changes in the presence of β and δ frequency bands, MF, and 95% SEF after stimulation, observed in dogs anesthetized with only isoflurane at 0.75 times the MAC, resembled classic EEG arousal reactions defined as desynchronization, a shift from lower to higher frequency ranges, and a decrease in amplitude. As was reported for an isoflurane study in goats, no clearly identifiable arousal was apparent at 1.5 times the MAC. Isoflurane can effectively block sympathetic nervous responses to noxious stimulation. Its analgesic properties, regardless of the isoflurane MAC multiple used, appeared to be strong enough to prevent brain activation in the study dogs. Remifentanil administration also resulted in the least number of EEG changes with deepening of anesthesia. Opioids have a dose-dependent suppressive effect on EEGs but do not usually result in maximal cortical suppression, even at higher doses than those used in the present study. This suppressive effect might be the reason for the weak correlation between EI and MAC observed during the IR sessions. Only slight differences in variable values between awareness and unconsciousness have been reported for humans anesthetized with remifentanil combined with an inhalant anesthetic. Opioid administration can cause excitatory patterns in an EEG, depending on anesthetic depth, which could explain the observed higher overall amount of brain activity (eg, as reflected by the 95% SEF during the IR versus isoflurane-alone session). Determination of MAC has become an established method for the evaluation of anesthetic potency.

In the present study, 1.0 times the MAC of isoflurane corresponded to a concentration in oxygen of 1.7 ± 0.3%, which is within the range of values reported for various breeds of dogs (8.18 ± 0.15% to 1.80 ± 0.21%). In addition to breed-dependent differences, many other aspects (eg, individual dog sensitivities to inhalant anesthetic agents, observer perception and experience, most likely, the synergistic effect of isoflurane combined with dexmedetomidine resulted in this strong hypnotic effect. However, the reactions to stimulation were strongest in dogs during the ID session at all MAC multiples. In rats, dexmedetomidine induces endogenous sleep pathways, resulting in an arousable sedation. With dexmedetomidine-remifentanil sedation, humans have a preserved cortical responsiveness to external acoustic stimuli, compared with those that receive midazolam-remifentanil. These findings in rats and humans revealed specific arousable cortical characteristics of dexmedetomidine, which were also evident in the present study.

Inclusion of remifentanil in the anesthetic protocol resulted in blunting of almost all brain activation after stimulation. Remifentanil acts at µ-opioid receptors, which are distributed throughout the CNS, such as the cerebral cortex and the spinal dorsal horn tissue, and can effectively block sympathetic nervous responses to noxious stimulation. Its analgesic properties, regardless of the isoflurane MAC multiple used, appeared to be strong enough to prevent brain activation in the study dogs. Remifentanil administration also resulted in the least number of EEG changes with deepening of anesthesia. Opioids have a dose-dependent suppressive effect on EEGs but do not usually result in maximal cortical suppression, even at higher doses than those used in the present study. This suppressive effect might be the reason for the weak correlation between EI and MAC observed during the IR sessions. Only slight differences in variable values between awareness and unconsciousness have been reported for humans anesthetized with remifentanil combined with an inhalant anesthetic. Opioid administration can cause excitatory patterns in an EEG, depending on anesthetic depth, which could explain the observed higher overall amount of brain activity (eg, as reflected by the 95% SEF during the IR versus isoflurane-alone session).

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underlying criteria for defining positive and negative reactions, and stimulation technique) influence the results. The effects of traditional clamping versus electrical stimulation for noxious stimulation in MAC determination reportedly do not differ significantly, but electrical stimulation can yield higher MAC values, which might also explain the rather high MAC values obtained in the present study.

The MAC method was used in the present study to attain a quantitatively similar anesthetic depth in all sessions. However, MAC may not be an ideal method for determining anesthetic depth: at the same MAC multiples, there were distinct EEG activity differences among the anesthetic protocols, which emphasize the limitations of the use of MAC, as the underlying reason for the suppression of immobility may have been mainly a spinal effect that did not reflect brain activity.

Inclusion of remifentanil or dexmedetomidine in the anesthetic protocol reduced the MAC of isoflurane by 41% in the study dogs. Opioids and α₂-adrenergic receptor agonists have MAC-sparing effects in dogs and other species such as humans or rats because of the drugs' analgesic and sedative effects. Remifentanil exerts a strong analgesic effect via μ, opioid receptors, whereas dexmedetomidine administration results in a decrease in the MAC, probably by strongly suppressing α₂-adrenergic receptors at the spinal level. Isoflurane MAC reductions in dogs by 59 ± 10% for remifentanil and by 59 ± 7% for dexmedetomidine administered in the same dosages as in the present study have been achieved in other studies. Even with a remifentanil infusion at 15 µg/kg/h, IV, the MAC of isoflurane decreases by 51% in dogs. Reasons for the lower decreases in the present study might be differences among dogs or the experimental setup as well as other influences.

The present study had some limitations. Although the 3 anesthetic protocols were not applied in complete random order to the 6 dogs, the washout periods of 1 week between anesthetic sessions should have minimized any bias introduced by this order. In addition, the EI might be a good indicator of anesthetic depth, given that it is easy to read, but it could not be fully evaluated in the present study because the EI had not been calculated at all time points in all dogs. Influences upon the EEG variables through EMG activity were not evaluated in the present study because the EI had not been calculated at all time points in all dogs. Influences upon the EEG variables through EMG activity were not expected, considering that EI values are reportedly not affected by an increase in EMG activity. On the other hand, an EEG burst suppression pattern may affect univariate descriptors such as SEF and MF derived from the power spectrum analysis. Data from an EEG recorded during burst suppression periods may fail to classify these periods as an increase in anesthetic depth. A burst suppression ratio could be calculated to quantify the influence. Because the algorithm underlying the EI includes an internal suppression detection, which cannot be reconstructed by the user, an unknown possible interference of that algorithm should be kept in mind.

Hypotension (mean arterial blood pressure, 50 to 60 mm Hg), with its possible depression of the EEG, needs to be considered when interpreting findings obtained during the isoflurane-alone and IR sessions at 1.5 times the isoflurane MAC. However, an influence on EEG recordings through accumulation of drugs would not be expected. Isoflurane is primarily eliminated via the lungs, with only 0.2% metabolized in humans. On the other hand, remifentanil is rapidly metabolized by nonspecific esterases in blood and tissue, with a context-sensitive half-time of 3 minutes that is independent of the duration of an infusion. In a pharmacokinetic study involving isoflurane-anesthetized Beagles, no cumulative effects were observed when a steady-state serum dexmedetomidine concentration (approx 2 ng/mL) was achieved through a CRI of dexmedetomidine administered for 7 hours at the same dosage as in the present study.

In the present study, anesthesia with isoflurane alone resulted in the greatest overall EEG depression with the strongest EI correlation of all 3 protocols evaluated. A weak correlation between EI and MAC multiples was evident when an opioid was included in the protocol. At the same MAC values, inclusion of remifentanil in the protocol resulted in the most depressed EEG responses to nociceptive stimulation, whereas the strongest arousal reactions were seen with dexmedetomidine. We could not identify a sole EEG parameter that differentiated among MAC multiples in dogs. The EEG parameters evaluated would likely not provide sufficient information for monitoring anesthetic depth in anesthetized dogs.

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b. GranCarno Adult, Animonda Petfood GmbH, Osnabrück, Germany.

c. Forene/Forene, Abbott AG, Baar, Switzerland.

d. Dexdomitor, Orion Corp, Espoo, Finland.

e. Perfusor Im, B Braun Melsungen AG, Melsungen, Germany.

f. Ultiva, GlaxoSmithKline, Boronia, VIC, Australia.


h. B Braun Melsungen AG, Melsungen, Germany.

i. Dräger Trajan 808, Drägerwerk AG & Co KGaA, Lübeck, Germany.

j. Alphavent, Drägerwerk AG & Co KGaA, Lübeck, Germany.

k. Bair Hugger, Caromed, Liebefeld-Bern, Switzerland.

l. Vasofix Braunüle, B Braun Melsungen AG, Melsungen, Germany.

m. Infusomat IM, B Braun Melsungen AG, Melsungen, Germany.

n. BD Careflow, Becton Dickinson, Franklin Lakes, NJ.

o. PMSET ART Safedraw (Basic Flexi), Becton Dickinson, Franklin Lakes, NJ.


q. Venofundin 6% Infusionslösung, B Braun Melsungen AG, Melsungen, Germany.


t. Datex Ohmeda Compact Monitor, GE Healthcare, Fairfield, Conn.

u. QUICK CALTM Calibration Gas, GE Healthcare, Fairfield, Conn.

v. Televet 100, Rösch & Associates Information Engineering GmbH, Frankfurt am Main, Germany.

w. Narcotted-Compact, version 5.0, MT MonitorTechnik GmbH & Co KG, Bad Bramstedt, Germany.

x. Disposable EasyGrip Monopolar Needle Electrode, 50 mm, 26 gauge, ViaSys Healthcare, San Diego, Calif.

y. Grass S48 Square Pulse Stimulator, Astro-Med, West Warwick, RI.
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43. Schallek W, Walz D. Effects of drug-induced hypotension on the