

and other factors held constant during this study) is equal to internal heat production. After 2 h of anesthesia, mean steady-state temperatures were identical in both groups, demonstrating that halothane/O₂ and N₂O/fentanyl anesthesia have comparable effects on heat balance. It is unlikely that central control (thermoregulatory inhibition) caused virtually identical hypothermia during two dissimilar anesthetic regimens. However, these data do not exclude the possibility that the degree of anesthetic-induced thermoregulatory inhibition depends on anesthetic depth, not type.

In summary, we compared changes in rectal temperatures in patients undergoing eye surgery during N₂O/fentanyl or halothane/O₂ anesthesia, and found no significant differences between groups. In both, average temperatures decreased 0.6° C and reached a thermal steady state at 36.2° C after 2 h of anesthesia.

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Changes in EEG Spectral Edge Frequency Correlate with the Hemodynamic Response to Laryngoscopy and Intubation

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Laryngoscopy and intubation of the trachea frequently produce tachycardia, hypertension, and increased serum concentrations of catecholamines.^{1,2} These responses may be exaggerated or lead to in-

creased morbidity in patients with hypertension,³ coronary artery disease,⁴ intracranial aneurysm,⁵ or increased intracranial pressure.^{6,7} However, these responses may be blunted either by ensuring an adequate depth of anesthesia prior to laryngoscopy or administering an additional, nonanesthetic drug to block the sympathetic response.⁸⁻¹⁰

Because the electroencephalogram (EEG) is an indicator of the electrical activity of the cerebral cortex, it may provide a clinically useful measure of the degree of cerebral depression following a dose of anesthetic. Moreover, the EEG signal may be processed to provide a quantitative value which represents underlying electrical activity. One such parameter is the Spectral Edge Frequency (SEF),¹¹ which reflects the highest frequency present in the EEG signal and is particularly sensitive to

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TABLE 1. Hemodynamic Responses in Groups I and II (Mean \pm S.E.)

Variable	Group I (SEF < 14 Hz)	Group II (SEF \geq 14 Hz)
Systolic BP (pre-induction) (mmHg)	138 \pm 4	146 \pm 3
Systolic BP (pre-laryngoscopy) (mmHg)	125 \pm 7	119 \pm 4
Systolic BP (maximal) (mmHg)	141 \pm 8	164 \pm 6*
Systolic BP (% Δ above baseline)	12 \pm 2	40 \pm 4†
HR (pre-induction) (bpm)	78 \pm 7	83 \pm 5
HR (pre-laryngoscopy) (bpm)	82 \pm 6	84 \pm 4
HR (maximal) (bpm)	92 \pm 7	101 \pm 4
HR (% Δ above baseline)	13 \pm 6	23 \pm 4
SEF (pre-induction) (Hz)	16 \pm 2	16 \pm 1
SEF (pre-laryngoscopy) (Hz)	9 \pm 1	17 \pm .6†
Fentanyl (μ g)	94 \pm 80	72 \pm 108
Droperidol (mg)	1.5 \pm 3	1. \pm 3

* $P < 0.05$.† $P < 0.0001$.

the "anesthetic-induced fast activity" usually present during light and moderate depths of anesthesia.

The SEF compresses the EEG voltage waveform into a single number, and thus simplifies the interpretation of EEG patterns. However, this compression is at the expense of information regarding the slower EEG waveforms. Nevertheless, the SEF, in human subjects, correlates with serum levels of thiopental,¹² etomidate,¹³ and fentanyl,¹⁴ and with end-tidal concentrations of halothane.¹⁵ If the SEF reflects the electrical state of the brain as an anesthetic drug is distributed, then it may be an accurate, time-varying indicator of anesthetic depth. In the present study, we correlate SEF depression after induction of anesthesia with predominantly thiopental to the hemodynamic response to laryngoscopy and endotracheal intubation. If the degree of SEF depression prior to laryngoscopy predicts the magnitude of arterial blood pressure response following the stimulus, it may be a useful prognostic indicator, particularly for patients with coronary-artery disease or a cerebral aneurysm.

METHODS

Institutional Review Board approval was obtained for EEG monitoring in 32 consecutive adult patients undergoing extracranial cerebrovascular procedures. None of the patients had physically apparent neurological deficits at the time of study. No sedative drugs were given preoperatively. The timing of drug administration for anesthetic induction was at the discretion of the responsible anesthesiologist. All patients received d-tubocurarine (3 mg iv), oxygen, and a test dose of thiopental (50 mg iv), followed by thiopental (3–5 mg/kg

iv), succinylcholine (1.5 mg/kg iv), lidocaine (75–100 mg iv), and fentanyl and droperidol at doses determined by the anesthesiologist (table 1). Patients were ventilated with oxygen *via* a face mask until direct laryngoscopy was performed at a time determined by the responsible anesthesiologist using only standard clinical signs and hemodynamic data. The duration of laryngoscopy ranged from 15–30 s. In cases of failed tracheal intubation, only data from the first laryngoscopy were included in the study. Volatile anesthetics were not used prior to endotracheal intubation.

All patients were monitored by electrocardiogram (ECG) and an automatic oscillometric blood pressure device set to measure at 1-min intervals throughout the study. For continuous measurement of blood pressure, all but one patient also had an indwelling radial arterial catheter placed before induction of anesthesia in the arm contralateral to the blood pressure cuff.

The scalp EEG was recorded over each cerebral hemisphere using standard ECG adhesive disk electrodes placed bilaterally in a frontal-ipsilateral mastoid configuration. The skin of each patient was prepared with OmniPrep™ (DO Weaver, Denver CO), and electrode placement was adjusted until impedances were all below 5,000 ohms. EEG signals were analyzed by a Neurotrac™ (Interspec, Conshohocken, PA) which displayed the resulting spectral data in the Compressed Spectral Array (CSA) format. Data were updated every 4 s, and overlaid with the calculated SEF. The responsible anesthesiologist was blinded to the EEG. The actual algorithm incorporated in the commercial EEG monitor used in this study is in concordance with the original description of the SEF as given by author IJR.¹¹ This algorithm searches each newly calculated EEG frequency spectra for the highest frequency contiguous 2 Hz wide band of activity. Each frequency bin of the spectra within this band contains more than a threshold quantity of power (generally Total EEG Power/256). The upper frequency limit of this band of activity is designated as the Spectral Edge Frequency. It has been the experience of the author (IJR) that this pattern matching method provides additional noise immunity and increased sensitivity to anesthetic induced changes in the EEG over the 95th percentile approach^{12,15} to the calculation of SEF.

Values for the systolic arterial blood pressure (BP), heart rate (HR), and SEF for each hemisphere were recorded at the following times: 1) awake, eyes closed; 2) post-induction, immediately before laryngoscopy and intubation; and 3) at the time of maximal BP following laryngoscopy. Monitoring continued for at least 5 min following laryngoscopy. The values recorded for the SEF were the combined averages of both channels over a 16-s interval centered at the time of interest.

In a previous pilot study of 18 patients, we found that patients whose SEF was equal to or greater than 14 Hz immediately prior to laryngoscopy were likely to exhibit a significant hypertensive response to the stimulation. The patients in this study were therefore assigned to two groups prior to laryngoscopy. Group I (n = 9) had a slow EEG, SEF < 14 Hz. Group II (n = 23) had a less depressed EEG, SEF ≥ 14 Hz. Data were analyzed by *t* test, Fisher Exact test, and plotted. A *P* value < 0.05 was considered significant.

RESULTS

No significant or sustained differences in SEF were appreciated between EEG channels. There were no significant differences between groups in preinduction SEF, or in their BP, HR, or average dose of fentanyl or droperidol before laryngoscopy (table 1). Following laryngoscopy, group II differed significantly from group I in maximal systolic BP and in the percent rise in BP (from pre-laryngoscopy). Systolic blood pressure rose $40 \pm 4\%$ (SE) in group II versus only $12 \pm 2\%$ in group I (*P* < .05). Heart rate response to laryngoscopy did not differ significantly. Of the nine patients in group I, only one had an increase in systolic BP greater than 20%. Of the 23 patients in group II, twenty had increases in systolic BP of greater than 20%. A Fisher Exact test suggested a statistically significance difference (*P* = 0.0001) between the rates of hypertensive responses in the two groups. A scatterplot of the pre-laryngoscopy SEF versus the percent change in systolic BP is shown in figure 1. A scatterplot of the pre-laryngoscopy SEF versus the percent change in mean arterial pressure revealed an almost identical relationship, whereas there was no apparent relationship between the SEF and the percent change in diastolic blood pressure. All of the patients with a maximal systolic BP of greater than 180 mmHg were in group II (SEF ≥ 14 Hz). We observed no correlation ($r^2 = 0.14$) between the pre-laryngoscopy systolic BP value and the percent change in systolic BP following laryngoscopy, as illustrated in figure 2. There was also no correlation between the decrease in BP following induction and either the maximal BP or the percentage increase in BP following laryngoscopy.

DISCUSSION

This is the first report of a correlation between EEG and the hemodynamic response to laryngoscopy. Our results suggest that if, during anesthesia with thiopental, the SEF is <14 Hz, the blood pressure response to direct laryngoscopy may be minimal. If the SEF ≥ 14 Hz, the blood pressure response may be exaggerated and, possibly, clinically undesirable.

Management of the stress response to laryngoscopy

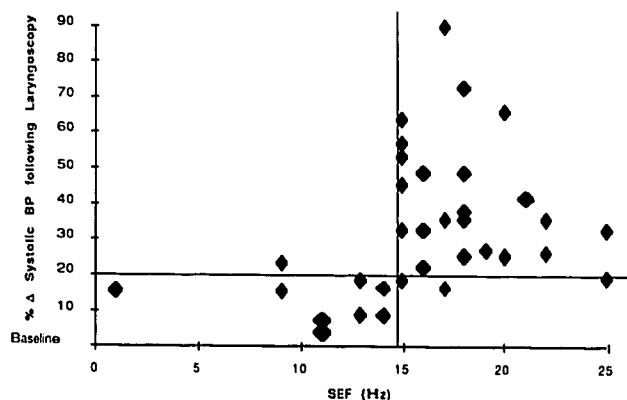


FIG. 1. A scatterplot of the SEF immediately prior to laryngoscopy and intubation for each patient, and the corresponding maximal systolic blood pressure following laryngoscopy as a percentage of the prelaryngoscopy pressure.

has received substantial attention in the literature.^{8,9,16-18} This response is a centrally mediated sympathetic reflex. Although the EEG only reflects the cerebral cortical activity, it appears to correlate with the depression in function of the deeper brain structures which is necessary to obtund the stress response during anesthesia with thiopental and adjuvants. The use of processed, but non-quantitative, EEG during fentanyl or fentanyl/clonidine reportedly demonstrates the slowing in EEG patterns (predominance of delta activity [<3.5 Hz]) consistent with an appropriate depth of anesthesia for laryngoscopy.¹⁶ Small doses of fentanyl in combination with thiopental also may be effective in blunting hemodynamic responses to laryngoscopy.⁸ Similarly, lidocaine administered intravenously⁹ or sprayed topically on retropharynx, larynx, and trachea¹⁷ may blunt these responses, although its efficacy is

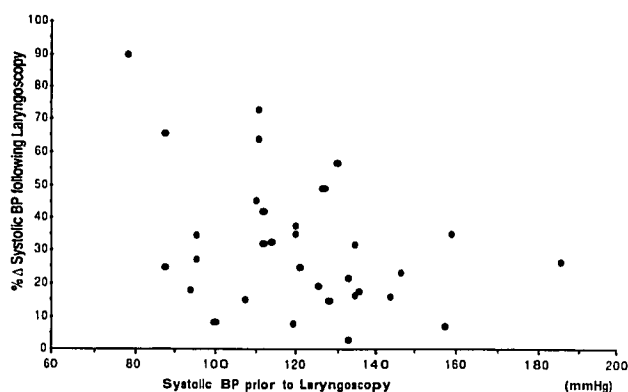


FIG. 2. The relationship between the systolic blood pressure immediately prior to laryngoscopy, and the magnitude of the increase to maximal blood pressure following the stimulus as a percentage of the prelaryngoscopy pressure.

controversial.¹⁸ Non-anesthetic drugs with predominantly peripheral actions, such as esmolol¹⁰ or nitroprusside,¹⁹ may also provide prophylaxis against the stress response without significant effect on the EEG.

Because other anesthetics create different EEG patterns, SEF criteria different from those used with thiopental may be required. In addition, the effects of vasoactive drugs, such as β -adrenergic blocking drugs or antihypertensive agents, may alter the sympathetic stress response and thus reduce the predictive value of EEG monitoring.

Monitoring the EEG appears to provide a sensitive, convenient measure of the depth of anesthesia during induction with thiopental and adjuvants. Moreover, under the conditions obtained in this study, the degree of SEF depression prior to laryngoscopy and intubation of the trachea appears to predict the magnitude of the blood pressure response following the stimulus. In contrast, in this group of patients, the systolic blood pressure prior to laryngoscopy did not predict the hemodynamic response. In patients with coronary-artery disease or cerebral aneurysms, the SEF may be used to adjust the anesthetic dose to ensure an adequate depth of anesthesia prior to stimulation.

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