TITLE: THE FRONTAL LEAD IS SUFFICIENT TO MONITOR THE EEG EFFECTS OF

MIDAZOLAM.

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Midazolam when administered in sedative doses has profound effects on the EEG power spectrum, with the increased power in high frequency bands clearly visible on a CDSA monitor(1). Often only the extreme frontal leads are used to monitor these EEG effects. We examined the EEG changes in different midline leads to determine the best lead for monitoring midazolam effects on the CNS.

METHOD: This study was approved by the local IRB, and informed consent was obtained in 10 healthy volunteers (4M, 6F) ages 25 - 36. EEG leads Fz, Cz, Pz, and Oz were attached and referenced to Al2. The power spectrum of the EEG was obtained from the analog signal and the log absolute powers in the frequency bands delta (1-3.5 Hz), theta (4-7.5 Hz), alpha (8-12 Hz), betal (12.5-20 Hz) and beta2 (20.5-30 Hz) were analyzed. EEG recordings were obtained before, during and after an infusion of midazolam I.V., 0.07 mg/kg at a rate of 0.5 mg/min. Repeated

measures ANOVA across channels and infusion conditions were obtained with significance at P < 0.05.

RESULTS: The changes in alpha (P<0.0004) and beta (P<0.0001) log power, and the beta1/alpha power ratio (B/A R) (P<0.007) were significantly different between leads in different conditions. The maximal EEG effect was seen at the end of infusion with beta power changes most prominent in the frontal lead, and alpha power changes most prominent in the occipital lead. The B/A R changed fourfold in both frontal and occipital leads.

EEG PARAMETER

LEAD:	BASELINE	END of INFUSION
beta1 Fz	2.50 ±	$0.53 3.73 \pm 0.36$
betal Oz	$2.58 \pm$	$0.67 2.76 \pm 0.58$
alpha Fz	$4.30 \pm$	$0.69 3.54 \pm 0.87$
alpha Oz	$5.01 \pm$	1.23 3.61 \pm 0.69
B/A R Fz	0.20 <u>+</u>	$0.14 0.96 \pm 0.74$
B/A R Oz	$0.12 \pm$	0.14 0.43 <u>+</u> 0.39
*values are	mean <u>+</u>	1 Standard Deviation
(units		uV2 or ratio)

CONCLUSIONS: The frontal lead directly monitors the maximal beta power changes, and will adequately reflect alpha activity, which occurs maximally in the occipital area, when the betal/alpha ratio is used.

1. Greenblatt DJ, et al: Clin Pharmacol Ther

45:356-65, 1989.

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Title: RESPIRATORY COMPLICATIONS OF VENOUS AIR EMBOLISM DURING

NEUROSURGERY

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Venous air embolism is frequently encountered during neurosurgical operations in the sitting position. During such procedures minor air embolism is common, and is reported in 30-40% of the cases. Major air embolism which can be fatal is rare. Development of pulmonary edema has been reported following venous air embolism.

In the present prospective study, 100 cases of neurosurgical operations in the sitting position were studied for respiratory sequelae of venous air embolism: arterial oxygenation and chest x-rays were compared in the preoperative and immediate postoperative period.

In all cases monitoring included arterial oxygen saturation, arterial blood gases, Doppler sounds

and end-tidal CO₁. Retrieval of air through a right atrial catheter was attempted in all instances in which venous air embolism was diagnosed.

In a total of 49 cases venous air embolism occurred, with lowering of PaO₂, end-tidal CO₂ and increase in PaCO₃.

Out of the 49 cases in which venous air embolism occurred, in 6 instances (12%) the PaO, in the immediate postoperative period was lower than the predicted, for a given FiO. The radiologist who read the X-rays was not aware of which patients had venous air embolism. In 8 instances (16%) radiological changes were found, ranging from increased vascular markings to incipient pulmonary edema. All of the above findings seemed unrelated to the severity of the intraoperative venous air embolism, and disappeared within 24 hours. In conclusion venous air embolism is associated with respiratory changes demonstrated by arterial blood-gas analysis and chest x-ray, but such changes are rarely severe, and usually transient.