INTRATHECAL PAPAVERINE ADMINISTRATION TITLE :

DECREASES THE RISK FOR SPINAL CORD ISCHEMIA IN RELATION WITH THORACIC

AORTIC ANEURYSM SURGERY

AUTHORS: G. Godet, M.D., M. Bertrand, M.D.,

E. Kieffer, M.D., J. Chiras, M.D. P. Coriat, M.D., G. Daas, M.D., P. Viars, M.D.

AFFILIATION: Department of Anesthesiology, La Pitié

Hospital, University of Paris 6, FRANCE

Neurologic complications remain a major concern during thoracoabdominal aortic aneurysmectomy (TAAA). The extension of the aneurysm, the location of the Adamkievicz artery, the duration of aortic clamping and the intraoperative disturbances are the main risk factors for spinal cord ischemia. Only intrathecal papaverine administration (IPA) seems to be effective for decreasing such a complication (1,2), but its efficiency is not demonstrated by a prospective study in patients with a high risk for spinal cord ischemia.

This randomized study was conducted in patients undergoing TAAA without the use of a shunt or a bypass, with informed consent and authorization given by our investigation Committee. Only 69 patients with Adamkievicz artery arising in or below the aneurysm, were included in the study and assigned to receive or not to receive IPA during aortic clamping. The data presented concern 52 patients who could be evaluated in a neurologic point of view in the postoperative

No patient who received IPA developped a complication in relation with this administration. Results are reported in the Table.

	SPINAL CORD DISORDERS			PERMANENT : FLACID : PARAPLEGIA :	
		TRANSIENT	PERHANENT	i	1
N = 52	11	1 4	: : 4	3	
WITHOUT IPA n = 29	1 1 7 1	i 0	: 4 : 4	: : 3 :	i : : p < 0.(
WITH IPA	. 4	. 4	1 0	: 0	: :

We conclude that IPA decreased the incidence of paraplegia and the severity of the other neurologic complications such as isolated proximal motor deficit or sphincter disorder during TAAA.

References

1. Svensson LG. Ann. Surg. 1986, 204, 38-47 2. Svensson LG. J. Thorac. Cardiovasc. Surg. 1988, 96, 823-829

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MIDAZOLAM AMNESIA: CORRELATION TITLE: WITH HIGH-FREQUENCY EEG POWER.

RA Veselis MD, RA Reinsel PhD, R Heino MD, R Alagesan MB, RF **AUTHORS:**

Bedford MD.

AFFILIATION: Dept of Anes & CCM, Memorial Sloan-Kettering & Cornell Univ.

New York, NY 10021.

Identification of midazolam-induced amnesia by EEG monitoring would be clinically useful. This study examines EEG spectral analysis parameters during different degrees of amnesia following IV midazolam sedation.

METHOD: Following IRB approval, 8 healthy, consenting volunteers (25-36 years old) received midazolam, 0.07 mg/kg, infused at a rate of 0.5 mg/min IV, while relaxed in the supine position with eyes closed. EEG lead Fz-Al2 was monitored and subjected to power spectrum analysis before, during and after midazolam. Amnesia was detected from the highest number recalled while listening to a number series presented on a tape at the time midazolam infusion was begun. During a 90 min post-infusion period, subjects were presented with verbal and visual stimuli for later recall. Data were collected at the following time periods: 1) Baseline; 2) 30 sec before amnesia; 3) 30 sec after onset of amnesia; 4) at the end of infusion; 5) 30 min post-infusion, and 6) 90 min post-infu-

sion. The degree of amnesia was scored as follows: no recall despite prompting= 2+; recall with prompting = 1+; spontaneous recall of all events = 0. Statistical comparisons were performed using ANOVA for repeated measures: P<0.05 was regarded as significant.

RESULTS: Among a large number of EEG power spectrum parameters analyzed, the log absolute power in the betal (12.5-20 Hz) and beta2 (20.5-30 Hz) bands identified most clearly the onset and degree of amnesia (Table). Specifically, there was a marked increase in these parameters during the time that amnesia was present (Beta1 = P<0.02, Beta2 = P<.001) as compared with values measured just before amnesia began.

CONCLUSIONS: Midazolam-induced amnesia can be identified by an increase in EEG beta power. As such, when amnesia is a desirable component of monitored sedation, EEG beta power monitoring may be useful to predict its presence.

REFERENCE: Greenblatt DJ, et al: Clin Pharmacol Ther 45:356-65, 1989.