TITLE: IMPACT OF PROPOFOL ON INTRACRANIAL DYNAMICS IN HEAD TRAUMA ICU PATIENTS.

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Previous investigations revealed a beneficial effect of propofol (P) on intracranial dynamics in patients undergoing elective neurosurgery. The present study evaluated the impact of P on ICP in head injured patients with normal or compromised intracranial compliance.

Methods: Epidural ICP-probes (GAELTEC) were implanted in a total of 14 patients with head injury. While heart rate, mean arterial pressure (MAP), ICP and ETCO2 were recorded continuously, P was given in subsequent doses of 0.5, 1.0 and 2.0 mg/kg; 15 minutes elapsed after each administration. Data were evaluated 1, 2, 5 and 10 min after each application and pooled. The patients were allocated to either group I (ICP<20mmHg) or group II (ICP>20mmHg) according to their ICP baseline level. Statistical analysis was performed by one-way ANOVA. A p of less than 0.05 was regarded as significant.

Results: In group I significant decreases of MAP were detected with 1.0 and 2.0 mg/kg P, whereas ICP and CPP changed only with the highest dose studied. While in group II significant decreases of MAP and ICP appeared with 1.0 and 2.0 mg/kg P, CPP was not affected significantly (Table).

Discussion: P decreased ICP in patients with normal and compromised intracranial compliance, in particular with 2.0 mg/kg P. As the responses to the hypotensive effects of P were mild and similar in both groups, inadvertent CPP drops were not observed with any of the doses studied. Thus, P can be used safely for the sedation of ICU-patients with head injury and normal or compromised intracranial compliance.

TABLE

	group	control	Propofol mg/kg 0.5	1.0	2.0
MAP	I	87±3	86±4	82±4*	75±5*
(mmHg)	II	88±4	86±4*	83±4*	79±4*
ICP	I	10±2	10±2	8±2	8±2*
(mmHg)	II	32±7	30±8	29±8*	28±8*
CPP	I	77±4	76±4	73±4	68±5*
(mmHg)	II	56±11	56±11	54±11	51±10

means ± S.E.M., * p < 0.05

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TITLE: HUMAN EEG DOSE RESPONSE TO DESFLURANE

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Desflurane (D) a derivative of isoflurane (I) has advantageous pharmacokinetics. In swine, EEG is indistinguishable between equipotent doses of D or I. Both agents cause burst suppression above 1.0 MAC. This report describes the EEG dose response to D in human volunteers.

Methods. Twelve healthy, young males (age=23.8±2.6 SD yrs) gave informed consent to this protocol which had been approved by the local Institutional Review Board. Subdermal electrodes provided a bilateral fronto-central and parieto-occipital montage. Subjects relaxed in a darkened room for >20 min before baseline measurements. Anesthesia was induced by inhalation of D and O_2 *via* face mask. Following endotracheal intubation, normocapnea and normothermia were maintained. Each subject received .83, 1.24, 1.67 and (if blood pressure was adequate) 2.1 MAC D, and at a different time, equipotent doses of D with 60% N_2O (equilibration at each step >12min). Hypocapnea (P_{ETCO_2} ≈ 25mmHg) was also induced at 1.24 MAC and the subject exposed to loud clapping at 1 Hz in an attempt to evoke seizures.

Four channels of EEG were displayed continuously by a prototype monitor (Interspec, Ambler, PA) and recorded on analog tape. The tapes were analyzed by a Macintosh IIx using Labview (National Instruments, Austin, TX) to derive: Burst Suppression Ratio (BSR), Spectral Edge Frequency (SEF) and Burst Compensated Spectral Edge Frequency (BSEF ≡ SEF * (1.0 - BSR/100)).

Results. Epileptiform activity did not occur. Low dose (0.83 MAC) D produced prominent α and ß range activity similar to that seen with other volatile agents. Increasing doses of D progressively increased burst suppression (fig. 1); at a given MAC multiple this effect was diminished during D/N2O. BSEF declined with increasing dose (fig 2).

Discussion. D produce similar cerebral electrical, and by extension, metabolic depression as I. There was no evidence of epileptogenicity. As with other inhaled anesthetics, the depression of EEG with D may be associated with decreased metabolic demand. It remains to be determined whether D uncouples cerebral blood flow from metabolic demand.