POSTER X—CLINICAL NEUROSCIENCES

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INTERACTION OF NICARDIPINE (Nic) AND HYPOCAPNIC ALKALOSIS (HA) ON CEREBRAL VASOMOTORICITY AND INTRACRANIAL PRESSURE.

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The effect of hypocapnic alkalosis (3.5 kPa) on nicardipine induced (1µg Kg⁻¹ in internal carotid) cerebral vasodilatation was studied by measuring cerebral artery diameter in 16 different locations on a carotid arteriogram (lateral incidence). Intracranial pressure (ICP) was recorded by an intraventricular catheter. 12 patients were divided in two randomized groups undergoing general anesthesia (methohexital, fentanyl, pancuronium bromide and mechanical ventilation with oxygen-enriched air (FIO₂ = 0.30) were studied. On T1 both groups had an arteriogram in normocapnia (NA). The first group (G1) was studied first in hypocapnia (T1) then following injection of Nic (T2) ; the second group (G2) was studied first after injection of Nic (T1) then in hypocapnia (T2). This study protocol received approval by our hospital ethical committee.

The results were analysed using Wilcoxon's T and W tests. Both groups were statistically similar for age, blood pressure, heart rate and PaO₂ at the three steps of the study. HA caused a 9.6 % decrease in arterial diameter (AD) (G1 T1, p<0.05) compared with baseline, and a 14.9 % decrease when preceded by an injection of Nic (G2 T2, p<0.05). However in the later case the decrease was 3 % in comparison with baseline (NS).

case the decrease was 3 % in comparison with baseline (NS). Nic increased AD by 18.5 % when preceded by HA (G1 T2, p<0.05), while the increase was 7.3 % in comparison with baselines values (p = NS). HA decreased ICP by 46 % (G1 T1, p<0.05) and by 42 % after Nic (G2 T2, p<0.05).

The effect of HA on ICP was maintained after Nic infusion. Nic did not cause an increase in ICP². Nic and HA mutually cancel their vasomotor effects as shown previously by Harris in the baboon using Nimodipine⁸.