TITLE: COMPARISON OF CO2 REACTIVITY FOR MIDDLE CEREBRAL ARTERY VELOCITIES AND 133-XE CEREBRAL BLOOD FLOW VALUES DURING ANESTHESIA

AUTHORS: W.L. Young, M.D., A. Massaro, M.D., E. Ornstein, Ph.D., M.D., N. Ostapkovich, R.EEGT., I. Prohovnik, Ph.D., J.P. Mohr, M.D.
AFFILIATION: Dept. of Anes., Neurology, and Pyschiatry College of Physicians & Surgeons, Columbia University, New York, NY 10032

Transcranial Doppler (TCD) ultrasound may be of value for studying and monitoring cerebral hemodynamic responses during general anesthesia. We compared reactivity of flow velocities in the middle cerebral artery to 133-Xe washout cerebral blood flow (CBF) values to changes in carbon dioxide tension.

METHODS After institutional approval and informed consent, 5 patients undergoing resection of cerebral arteriovenous malformation (AVM) were studied under 0.75% isoflurane (ISO) anesthesia in 3:2 N2O/O2. Data was taken from the hemisphere contralateral to the AVM. TCD[1] and CBF[2] were measured as previously described. After induction, TCD mean (MV), systolic (SV) and diastolic (DV) velocities were measured during a rise in PetCO2 from approximately 25 to 35 mmHg. During surgery, CBF was measured during equivalent PetCO2 levels. The slope of the response for MV, pulsatility index (PI = ([SV-DV]/MV) and CBF were plotted and extrapolated values at PetCO2 of 25 and 35 mmHg were calculated and compared by ANOVA and linear regression.

RESULTS. The means for MV, PI and CBF are shown in Figure 1. Although the slopes of the CBF responses *vs.* PI responses correlated (r = 0.862), there was no trend obvious from the MV slope correlation (r = 0.204).

DISCUSSION. This pilot study suggests that within-subject agreement of TCD and CBF values are strong and both predictably change as a function of PetCO2. CBF determinations may be used as a sort of "2-point calibration" for interpretation of TCD indices. However, caution should be used in applying TCD values when making between-subject comparisons of changes in cerebral hemodynamics without an index of true tissue perfusion.

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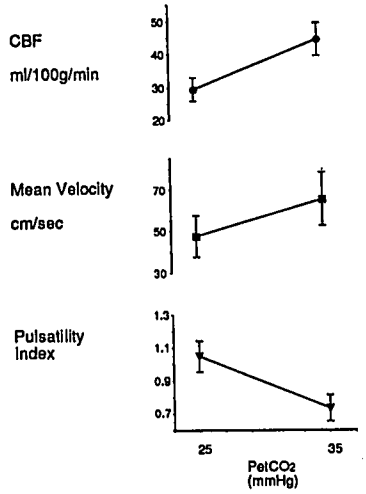

Figure 1. Changes ($p < 0.05$) in cerebral blood flow (CBF) and middle cerebral artery TCD mean velocity and pulsatility index (n = 5) as a function of PetCO2.

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TITLE: POSITIVE *IN VITRO* HALOTHANE CONTRACTURE TEST IN PIGS EXHIBITING NONRIGID MALIGNANT HYPERTHERMIA (MH)

AUTHORS: J.E. Fletcher, Ph.D. F.J. Huggins, M.S., L. Davidson, B.S. P.A. Conti, V.M.D. and H. Rosenberg, M.D.
AFFILIATION: Dept. Anesthesiology, Hahnemann Univ., Philadelphia, PA 19102-1192 U.S.A.

Anesthesia-induced MH has primarily been associated with muscle rigidity. Indeed, this has been a major reason for suspecting altered Ca^{2+} regulation as the primary defect in MH. However, nonrigid forms of MH have been reported in humans. We are not aware of any reports of nonrigid MH in MH susceptible swine. The present report examines the contracture test in muscle removed from nine swine immediately prior to exhibiting nonrigid MH and four control swine from the same breed. The pigs were initially diagnosed for MH susceptibility by the barnyard challenge (halothane 6%), CPK values and H blood typing. Biopsies were removed with a non-triggering anesthetic (ketamine and N_2O). The halothane portion of the North American MH Group Protocol was performed, as previously described.[1] Subsequently the swine were administered halothane 3% (5 min), followed by halothane 2% for the duration of the study (43 additional min). Succinylcholine (SCh) was added at 1 mg/kg (5 doses spaced 5 min apart) starting 20 min after halothane was reduced to 2%. All MH swine were confirmed as MH susceptible in the *in vitro* contracture test. The mean magnitude of contractures to halothane in the diagnostic test for the MH group (Table 1) was similar to that previously reported for other swine from this strain that exhibited rigid MH on halothane/SCh challenge.[1] During the barnyard challenge (first challenge), the swine exhibited pronounced muscle rigidity. The second *in vivo* challenge did not elicit rigidity, despite the administration of both halothane and SCh. Although the swine in the present study were flaccid and only exhibited slight temperature elevation during the second *in vivo* challenge (Table 2), they did exhibit some signs characteristic of MH, including elevated lactate, reduced HCO_3, a highly negative base excess and elevated K^+ levels (Table 2). We are not certain why the swine did not exhibit rigidity during the second challenge, but speculate that the restricted diet given between the two challenges (400 g chow/day) was responsible. An important finding was that the contracture test appears to test the presence of the MH defect, not the severity of the syndrome. More importantly, these results suggest that rigid and nonrigid MH could be the consequence of the same genetic defect.

References
1 Anesthesiology 72 71 1990

TABLE 1. Contracture (g) response to halothane 3%.

Cont (n=4)	0.04 ± 0.03
MH (n=9)	1.06 ± 0.16 (greater than Cont; P<.002)

TABLE 2. Metabolic changes after 48 min exposure to halothane and SCh (mean ± SEM).

	n	Lactate	HCO_3	Base Excess	$[K^+]$	Temperature Increase °C
Cont	4	3±1	29±1	- 1±2	5.8±0.1	+0.7±0.1
MH	9	13±2[b]	23±2[a]	-14±1[c]	7.2±0.3[b]	0.0±0.2[b]

[a]P<.05; [b]P<.01; [c]P<.001 by two tailed t-test.