Title : CONTROL OF BRAIN BOUNCE DURING MICROVASCULAR ANASTOMOSIS

Authors : D. Lobo, M.B., B.S. F.F.A.R.C.S., F. Brauer, M.D.

Affiliation: Department of Anesthesiology, Loma Linda University, Loma Linda, California 92350

Introduction. Brain bounce is oscillation of the brain surface seen most noticeably during small window craniectomy. It may be synchronous with the arterial pressure wave or with the positive pressure phase of controlled ventilation (ventilator heave). Though some brain movement is normal, when magnified under a microscope at 10X to 40X power, brain bounce can make surgery on small arteries difficult or impossible.

During cerebral revascularization by superficial temporal-middle cerebral anastomosis, brain bounce presents some unique problems in anesthetic management. The subjects have cerebral ischemia, and their autoregulation is frequently impaired, leading to a more frequent occurrence of bounce. The craniectomy is a small one, so that even moderate volume changes in the cranial contents are magnified at the operative site. To minimize constriction of the middle cerebral artery and to preserve perfusion in an ischemic brain, lowering of either $PaCO_2$ or arterial pressure is undesirable. Therefore, few weapons remain to cope with a phenomenon which can wholly frustrate surgery.

Clinical material and observations. During the period 1973 to 1979 we studied brain bounce and tested different control measures in 165 patients undergoing cerebral revascularization by superficial temporal-middle cerebral anastomosis. Early in the series 73% of patients showed troublesome bounce. Since 1976 the incidence has fallen to 37%. For all patients the same basic anesthetic drugs were used: thiopental, nitrous oxide, narcotics, and pancuronium, with blood pressure and $PaCO_2$ kept close to normal. The recent lower incidence of bounce we attribute to meticulous adjustment of ventilation to give low tidal volume, low inspiratory flow rate and minimal expiratory resistance.

Effects of added drugs. To make surgery feasible in patients in whom arterial bounce persists we have tested a number of therapeutic drugs. Halothane and enflurane sometimes reduced observable bounce, but not predictably, and at the cost of hypotension which was sometimes of undesirable degree. Intravenous thiopental reduced bounce satisfactorily in most patients, but the large doses required also contributed to unwanted post-surgical somnolence. Nitroprusside infusion reduced brain bounce but caused frequent tachycardia and episodic hypotensive overshoot. Though these drugs all offered some measure of control, their use was discontinued in this series because they would interfere with parallel studies of brain enzymes.

Based on reported properties of nitroglycerin (NTG) (1) this drug was given by infusion to 12 recent patients who had brain bounce. The dose range was low (30 to 60 ug/min). Five patients had no fall (0-5%) in mean arterial pressure (MAP). Three patients whose MAP had been elevated fell 5% to 12% while receiving NTG. One patient's MAP fell 12.5% but quickly rose after fluid repletion without reducing NTG infusion rate. Heart rate increased by 8% (mean). Emergence from anesthesia was unaffected. In all 12 patients brain bounce disappeared or diminished to tolerable levels during NTG infusion.

Discussion. Brain bounce results from a complex interplay between several physiologic variables, including arterial blood pressure, stroke volume, venous pressure, cerebral vascular elastance and brain tissue turgor. In the closed cranium these factors produce pressure changes, whereas in the open system after craniectomy they produce volume changes in the cranial contents (2). Since normal arterial pressure and $PaCO_2$ must be maintained in these patients, venous pressure is more susceptible to our manipulation either by adjusting ventilation patterns or by low dose NTG infusion. Amongst several adjuvant vasoactive drugs, NTG came closest to providing a reduced elastance of capacitance vessels at dose levels which interfered little with arterial pressure. It has enabled us to consistently provide satisfactory operating conditions.

References.

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