Session on Cerebrovascular Disease
Morning Meeting
Saturday, May 3, 1980

9:00 A.M.—12:00 Noon
Ballroom E

Chairman: Howard L. Hurtig, Philadelphia, PA
Secretary: J. Philip Kistler, Belmont. MA

CVD 1—9:00

Long-Term Survival and Neurologic Status Following Resuscitation from Out-of-Hospital Cardiac Arrest

MICHAEL P. EARNEST, PHILIP R. YARNELL, GARY L. KNAPP, and SUSAN L. MERRILL, Denver, CO

Thirty-eight of 53 patients surviving from among 117 patients previously studied after out-of-hospital cardiac arrest were evaluated approximately 3½ years later.

Twenty patients were living; 18 had died. Fifty-three percent of the 38 had resumed independent social activities, but only 32% had returned to work. Only 2 of 17 patients neurologically examined were normal, while 8 of 14 patients had normal abbreviated neuropsychologic testing. Discriminant analysis showed that long-term outcome was associated with the patient being awake on admission or awakening to follow simple commands within 2 days and with good neurologic status at the time of hospital discharge. Long-term outcome was not significantly associated with other admission signs of pupillary reaction, oculovestibular reflexes, purposeful response to pain, apnea, or need for ventilation.

None of nine patients with poor neurologic function at discharge subsequently resumed working or independent living.

It is our impression from this study that: (1) patients who awake within 2 days of hospital admission and who survive subsequent cardiac and other medical complications have about 80% probability of good neurologic function several years later; (2) the other admission neurologic signs do not correlate with long-term outcome; and (3) significant long-term improvement from a poor neurologic status at hospital discharge is very uncommon.

CVD 2—9:15

CSF and Serum Creatine Kinase Isoenzyme BB Activity and Neurologic Outcome After Out-of-Hospital Cardiac Arrest

W. T. LONGSTRETH, Jr., and S. M. SUMI, Seattle WA

The ultimate quality of survival of most patients in coma after resuscitation from cardiac arrest is often dependent not on the underlying cardiac disease, but on the degree of anoxic-ischemic brain damage. To allow an early prediction of eventual neurologic outcome, the results of systematic clinical examination and electroencephalographic changes have been utilized with varying results.

We report our comparison of neurologic outcome in 55 consecutive patients resuscitated from out-of-hospital cardiac arrest with CSF creatine kinase BB activity (CKBB) in 20, and serum CKBB in 52 patients. CSF CKBB was 2 U/L in all patients with full neurologic recovery but was markedly elevated (mean 55 U/L) in those dying without neurologic recovery. Patients with intermediate neurologic outcomes ranging from dementia to persistent vegetative states had intermediate CSF CKBB values (mean 7 U/L). Serum CKBB was detected within 6 hours of cardiac arrest in 86% regardless of neurologic outcome. More than 6 hours after cardiac arrest only 4% with full recovery but 100% without neurologic recovery and death showed CKBB.

These preliminary results demonstrate a correlation between both CSF and serum CKBB and neurologic outcome after cardiac arrest.

CVD 3—9:30

Prolonged Hypoxia in Man Without Circulatory Compromise Fails to Demonstrate Cerebral Pathology

M. A. RIE, and P. G. BERNAD, Boston, MA

This study attempted to determine whether hypoxia produces neuropathologic changes in human brain. Three previously healthy young adults (16-19 years) without known cerebral or cardiac disease developed acute respiratory hypoxia (PaO_2 < 45 mm Hg) of 1-8 days duration. Patients were monitored throughout their illness with continuous recording of mean intra-arterial blood pressure (MAP) and frequent intermittent determination of cardiac output (CO), arterial (PaO_2) and mixed venous (PvO_2) oxygen tensions. Each patient was found to be responsive and following commands until the day of death.

Duration of hypoxemia (hrs)	Terminal PaO_2 (mm Hg)	Terminal CO (L/min)	Terminal MAP (mm Hg)
192	30	8.00	55
28	24	2.96	57
31	38	*	80

*CO = 10.44 L/min 4 hrs prior to death.

In all cases measures were taken to maintain systemic blood pressure and flow at supernormal levels. The mode of death in each case was sudden refractory bradycardia unresponsive to maximal respiratory, vagolytic and cardiotonic therapies. Detailed pathologic examination of each brain by two neuropathologists unfamiliar with the clinical histories failed to reveal any of the pathologic changes that are known to be associated with ischemic-anoxic brain injury.

CVD 4—9:45

Tolerance of the Perinatal Brain to Graded Hypoxemia.

ROBERT C. VANNUCCI, ELIZABETH E. NARDIS, SUSAN J. VANNUCCI, and PAULA A. CAMPBELL, Hershey, PA

To ascertain the metabolic response of the immature brain to graded hypoxemia, newborn dogs 2-6 days postnatal age were tracheostomized, paralyzed and artificially ventilated with 70% N_2O-20% O_2. Thereafter, the puppies breathed 5% O_2 for up to 1 hour, beyond which no animal survived owing to cardiovascular collapse. Baseline systemic measurements were: arterial blood pressure (MABP) = mm Hg; PaO_2 = 95 mm Hg; $PaCO_2$ = 37 mm Hg; pHa = 7.40; arterial lactate = 1.7 mmol/1. Arterial PO_2 in animals exposed to 5% O_2 for up to 30 minutes averaged 13 mm Hg and resulted in a progressive metabolic acidosis (pHa = 7.17) from lactacidemia (12.3 mmol/l). MABP fell to 49 mm Hg. Cerebral glycolytic intermediates and high-energy phosphate reserves determined following in situ brain freezing were (mmol/kg):

	Glucose	Lactate	L/P	P-creatine	ATP	ADP
Control	4.1	1.3	14	2.9	2.3	0.23
Hypoxia (15 min)	3.1*	6.6†	24†	2.3*	2.4	0.26*
(30 min)	3.0*	11.5†	33†	1.8†	2.3	0.26†

*$p < 0.05$.
†$p < 0.01$.