Experimental Cerebral Circulatory Arrest: Effect on Electro cortical Potentials

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PONTONOUS cerebral electrical potentials have been used clinically for prognosis following destructive cerebral lesions, and a persistently isoelectric electroencephalographic recording (EEG) has been considered one criterion of cerebral nonviability both for cessation of artificial maintenance of vital functions and for subsequent somatic organ transplantation. Prominent among causes of cerebral nonviability are those related to circulatory arrest, which include shock, arterial occlusive disease, cardiopulmonary insufficiency, and cardiac arrest. Diminution of cerebral blood flow as well as greatly increased intracranial pressure causes stagnant (ischemic) hypoxia, which in turn is reflected in profound EEG changes.

These types of ischemic or anoxic cerebral insults may be reproduced experimentally and their effects on electrocorticographic (ECoG) activity recorded. In such experiments, intrathoracic vascular occlusion causing immediate circulatory arrest has been produced to determine patterns of electrocortical silence, recovery, and the sensitivity of cortical electrical function related to survival. However, the preparation is not comparable to stepwise obliteration of the four major cervical vessels since simultaneous occlusion is difficult and a certain amount of cerebral damage can occur during experimental ligations, which make exact timing of anemia difficult. Also, a potentially large amount of collateral circulation exists. In man, for example, cases have been reported in which only minimal neurological dysfunction was caused following occlusion of all major cervical vessels, although acute lengthy occlusions may not be well tolerated. In animals, ligation of the major cervical vessels frequently produces little deficit. Both patients and experimental preparations have survived with excellent cerebral electrical activity because of extensive collateral circulation.

Therefore, an experimental preparation used previously by Brockman and Jude and Marshall, et al., was employed because it results in complete, immediate, but temporary cerebral circulatory arrest.

Material and Method

We used 30 large mongrel dogs for this investigation, of which 20 were considered successful experiments. Ten dogs were not counted in the results, six because of ventricular fibrillation following lengthy occlusion periods with inability of rapid resuscitation, and four because they were used to evaluate the cerebral blood flow during occlusion.

All animals were anesthetized with pentobarbital or thiamylal, 20 to 30 mg per kilogram body weight. Gallamine supplementation was necessary occasionally. Following barbituralization and during the entire surgical procedure, the animals were intubated and ventilated with 40% oxygen and 60% room air by positive pressure. Thoracotomy was performed through the fourth right intercostal space, and umbilical tapes were loosely placed around the roots of the ascending aorta, inferior vena cava, and superior vena cava cephalad to the azygos vein according to the techniques quoted.

The azygos vein was not ligated. Umbilical tapes were threaded through a length of rubber tubing for the purpose of temporary occlusion of these vessels. Cessation of left ventricular cardiac output except for coronary flow was accomplished by near-simultaneous occlusion of both vena cavae and the ascending aorta. Venous blood returned through the azygos vein and coronary sinus.

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The vena cavae were occluded first, followed immediately by the ascending aorta. After variable periods of cessation of extra-cardio-

pulmonary circulation, the vena cavae were released approximately 10 sec prior to the aorta, and the period of occlusion was timed from aortic occlusion to aortic release. Follow-

ing vascular release, the lungs were reexpanded under direct vision and the chest closed airtight in layers.

Following chest closure, the animals were ventilated as long as necessary and until adequate spontaneous respirations re-

turned.

Deep body temperature was between 36.5° and 37.5°C in all animals. Femoral artery pressure and pulse were obtained via Statham strain gauge and transmitted to a Grass polygraph. Respiratory rate and electrocardiograph (EKG) were registered si-
multaneously on a Grass polygraph. Periodic measurements of hematocrit, pH, pCO₂, pO₂, and oxygen saturations were performed for evaluation of homeostasis during the vari-

ous experiments. The ECoG was monitored on a Grass Model-6 8-channel electroen-

ccephalograph utilizing silver ball recording electrodes 3.5 mm in diameter which were implanted snugly at the time of the acute surgical procedure into the epidural space using small twist drill holes and fixed with acrylic cement. Standard recordings were taken from frontal and parieto-occipital areas using bipolar techniques. The four main channels consisted of paired left and right electrodes. All experiments were con-

ducted with identical standardization and calibration. Control records of animals anes-

thetized with pentobarbital were slower than those anesthetized with thiamylal, but this was generally not reflected in post-occlusion records.

The ECoG rather than the EEG was em-

ployed because, particularly in the experi-

mental preparation, much muscle and other artifact as well as damping effect were elimi-

nated. The ECoG was discontinuously fol-

lowed up to several days during the post-oc-

clusion periods by detaching and then recon-

necting the wires of the permanently placed silver ball electrodes.

Results

With vascular occlusion, femoral arterial pressure dropped to baseline levels indicating no blood pressure within 2 to 5 sec. The ECoG from bifrontal and biparietal dural electrodes became and remained isoelectric within 10 to 20 sec and for the duration of vascular occlusion (Fig. 1); this rapid change served as a control for adequacy of occlusion and non-interference by the beating heart, respirations, or other artifact. Occa-

sionally, isoelectric recordings remained for considerable periods even after reinsti-

tution of cerebral blood flow and were always present following death. The isoelectric re-

cording at death during ventilation also served as a control for the demonstration of lack of electrical artifact by ventilators. Coincident with occlusion, the EKG showed changes in QRS complexes and, often, tran-

sient changes in rhythm. Following restora-

tion of circulation, these dysrhythmias re-

turned to normal sinus rhythm.

Ten animals succumbed between 6 and 48 hrs following 8 to 20 min of occlusion (Table 1). Ten animals survived 2 to 12

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**Fig. 1.** Isoelectric recording obtained during occlusion. (Horizontal bar 1.0 sec, vertical bar 50 μV; F = bifrontal recording, P = biparietal recording, FPR = right frontoparietal recording, FPL = left frontoparietal recording.)