Carotid Angiography after Experimental Head Injury in the Rat

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Summary. Carotid angiography was performed in 11 anesthetized and artificially respired rats before and after infliction of a reproducible but variable occipital head injury. Arterial narrowing of the intra- and extracerebral arteries and slowing of the circulation occurred after trauma and correlated with the impact velocity. Maximal changes were seen within a minute but tended to decrease over 20--50 min. Possible causative factors for the arterial narrowing were basal subarachnoid hemorrhage, which was an almost constant finding, stretching of the basal vessels due to extreme movement of the craniocervical junction and sympathetic discharge from the brain stem at the moment of impact. The arterial narrowing possibly explains profound ischemic changes in the brain tissue energy metabolism found in other studies after injury with the same method.

Multiple foci of ischemic brain damage have been reported in patients who died from blunt head injuries and in whom the infarct could not be attributed to contusion or brainstem herniation [1]. The lesions tend to occur in boundary zones between cerebral arteries suggesting a vascular cause for the changes. In previous experiments with a head injury technique in the rat we found that trauma causing brief concussive symptoms did not imply any change in the energy state of the brain [20] but that more severe blows caused an increasing derangement of the energy metabolism of the tissue and an increasing mortality [15]. There were also indications that the changes increased in the first few minutes, suggesting that some complicating factor was responsible for the impairment of the energy state. This factor would be cerebral vasoconstriction which has been found in angiographic studies after experimental concussion in other species [13]. A technique for cerebral angiography in the rat was previously described by one of us [7], and was now applied to the concussion technique used for the previous studies of energy metabolism in the rat brain after trauma.

Material and Methods

General Experimental Conditions

The experiments were performed on male Wistar rats (300 to 350 g) which had been prepared with tail artery and vein catheters. After induction of anesthesia with divinyl ether the animals were intubated endotracheally and paralyzed with an intravenous injection of suxamethonium chloride. They were respired artificially in a normocapnic steady state with 25 percent oxygen and 75 percent nitrous oxide with a volume- and flow-controlled respirator based upon a mechanical flow regulator [11]. The blood pressure was monitored continuously through the tail artery catheter, from which blood samples were also taken for measurements of arterial $P_{O_2}$, $P_{CO_2}$ and pH.

Angiographic Technique

The technique for catheterization of the common carotid artery through the femoral artery has been described in a previous paper [7]. Angiograms were obtained in the AP projection with the use of a small
film changer for industrial film [2]. FFD 45 cm, focus 1.0 mm. Film: Agfa Gevaert D4, no intensifying foils. Exposure data: 80 kV, 20 m As and 0.04 sec. 0.4 ml of contrast medium (Isopaque Cerebral, Nyco, Norway) was injected in the common carotid artery at a constant rate by a pressure syringe. The series consisted of 1 film per second for 8 sec. In evaluating the angiograms special attention was paid to the caliber of the common carotid, internal carotid, middle cerebral and pterygopalatine arteries as well as to the circulation time (Fig. 1).

of the animal was hit from below by an air driven piston with adjustable velocity. The impact point was the occipital crest. The blow thus gave an initial upward mainly translational acceleration of the freely movable head and extreme retroflexion at the craniospinal junction and upper part of the cervical spine. Impact velocities of 7, 8 and 9 meters per second (m/s) were used.

Experimental Procedure

After the animal had been prepared as described a control angiogram was made. The animal was then placed in the plaster bed and one impact of 7, 8 or 9 m/s was given. The physiological response to the trauma was evaluated from the blood pressure and pulse rate reaction. Angiography was again performed after the trauma at different intervals from 45 sec to 50 min. Only animals in good respiratory and general circulatory state were subject to repeated angiography. After the last angiogram the animals were sacrificed and the skull and brain were examined for gross morphological changes.

Fig. 1. Selective internal carotid artery injection in a specimen. The middle cerebral artery (→), anterior cerebral artery (→) and pterygopalatine artery (→) are demonstrated. The pterygopalatine artery is the largest branch of the internal carotid artery, but does not feed the brain. The basilar artery is also filled with contrast medium (→ ←)

Trauma Technique

Trauma of a specific acceleration was applied to the head of the animal lying fully relaxed on the back in a plaster bed thus making certain of an exact and reproducible position of the head and body. The head

Results

The trauma technique has been used in parallel series to study the immediate clinical effects, survival, morphological changes and effects on the energy metabolism of the brain. The results will be published elsewhere [15] but will be summarized briefly here.

An impact of 7 m/s gave a clear concussive reaction with an immediate blood pressure rise usually of 30 to 60 sec duration combined with bradycardia and irregular pulse. Non paralyzed animals had convulsions and apnea or bradypnea during this phase, were unresponsive for another 1 to 3 min and then usually recovered. During the period of blood pressure elevation a few animals developed pulmonary edema. In a few cases there were no morphological changes but moderate subarachnoid hemorrhage was usually found in the cisterna magna, basally over the brainstem and around the upper cervical cord.

With an impact of 9 m/s a more marked blood pressure reaction, often without bradycardia, was observed. Pulmonary edema developed in about 40 percent and was often so massive that the animal died from asphyxia. After the initial convulsive reaction the surviving animals usually were unresponsive for several minutes and during observation periods of one hour or more their motor activity was markedly reduced and respiration was irregular. Some animals had occipital fractures. They always had subarachnoid hemorrhage around the brain stem and cervical cord. Small perivascular hemorrhages were usually found microscopically in the brainstem.

The 7 m/s trauma did not significantly change the energy state as judged from the contents in the brain stem and cortex of phosphocreatine ATP, ADP, and