PHYSIOLOGIC STUDIES OF ARTERIOVENOUS ANOMALIES OF THE BRAIN

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There has been considerable attention given the clinical and pathological features of the vascular anomalies and tumors of the brain without, however, considering the physiological changes in the cerebral and general circulation that accompany such lesions. Our attention was drawn to these changes by the fact that the finding of a tremendously increased cerebral blood flow in a patient, considered to be suffering from idiopathic epilepsy for 15 years, led to the establishing of the correct etiology of the patient's convulsive seizures. This patient was proven to have an arteriovenous vascular anomaly of the left cerebral hemisphere, and confirmed the findings of the first case.

METHODS AND CLINICAL MATERIAL

1. The cerebral blood flow was determined by the method of Kety and Schmidt. This method is based upon the application of a modified Fick Principle to the dynamic exchange of an inert gas between blood and brain. Simultaneous samples of blood are withdrawn from the superior jugular bulb and from the arterial system throughout the 1st minute, at the end of the 1st, 3rd, 5th, and 10th minutes after the patient has begun to breathe a mixture of 15 per cent nitrous oxide, 21 per cent oxygen, and 64 per cent nitrogen. The gaseous mixture was administered through a tightly fitting mask over the nose and mouth. Blood samples were immediately iced and analyzed within 6 hours for oxygen, carbon dioxide, and nitrous oxide contents by means of the Van Slyke-Neill manometric apparatus. The hydrogen ion concentrations were determined potentiometrically at 38°C. by means of a glass electrode. With the arteriovenous oxygen difference available, an exact determination of the cerebral oxygen consumption (metabolic activity) may be obtained by the product of this difference and the rate of cerebral blood flow.

2. Cardiac output was estimated upon the ballistocardiograph in both the horizontal and vertical positions by Dr. Isaac Starr.

3. Orthodiagrams and electrocardiograms were done by Drs. William Jeffers and John Sayen of the Robinette Foundation.

4. Total blood volumes were estimated, utilizing the dye (T 1824) dilution method of Gibson and Evelyn.

5. Mean arterial blood pressures were recorded by direct reading, with a damped mercury manometer, from the femoral artery during the determin-
ation of the cerebral blood flow. Pressures in the internal jugular bulb were determined directly.

Case 1. M.M., a 48-year-old female, had a generalized epileptic seizure in 1931 at the age of 28. Her second seizure was in 1934, when she aborted a 5-month fetus. From this time on she has had generalized grand mal attacks, at first occurring about once a month, but rapidly increasing in frequency until they occurred every 3 to 4 days. Her seizures are preceded by an aura of a “wave or cloud” sweeping up from her chest to her head, following which she loses consciousness. Numerous petit mal seizures occur between the grand mal attacks. In 1940 she began to display memory defects and emotional instability with some flight of ideas. An encephalogram was performed at another institution in 1941 but the ventricles were not filled with air. The subarachnoid pathways were visualized and found to be dilated. A diagnosis of cortical atrophy was suggested. Phenobarbital and dilantin were undoubtedly effective in controlling her convulsions, but over the course of the years her treatment was inadequate.

In November 1946, the patient was admitted to the Hospital of the University of Pennsylvania under the impression, gained from her out-patient record, that she represented a typical case of idiopathic epilepsy. Examination disclosed severe impairment of her memory for recent events and marked emotional instability. Visual field studies revealed an incongruous, incomplete left homonymous hemianopia. The usual laboratory studies of blood, urine and spinal fluid were normal. Ordinary x-ray films of the skull were not remarkable. On Nov. 18, 1946, a study of the cerebral blood flow was made, revealing it to be 143 cc./100 gm. of brain per minute. This is approximately three times the normal value found by Kety and Schmidt in a series of normal adult males. On the supposition that this was probably due to an abnormal intracranial arteriovenous shunt, arteriography was carried out and a large right-sided frontoparietal angiomatosus mass (Fig. 1) disclosed. The left arteriogram was normal.

Case 2. F.B., a 28-year-old man, was originally admitted to the Hospital of the University of Pennsylvania on Dec. 1, 1941. This patient has had right-sided Jacksonian sensory seizures since 1936. In June 1940, he had his first right-sided Jacksonian motor seizure which terminated in unconsciousness. These attacks recurred 6 or 7 times and starting in September 1941, he gradually lost strength in his right extremities, principally the upper. Neurological examination confirmed the motor weakness and in addition brought out a mild right hemihypäthesia and hemihypalgesia. No bruit could be heard at any point over the head. Ordinary studies of the blood, urine and spinal fluid were entirely normal. A roentgenogram of the skull showed a small area of calcification in the left frontoparietal region. Encephalography revealed this calcification to be just to the left of the body of the left lateral ventricle. There was a very questionable displacement of the ventricular system to the right. On Dec. 19, 1941, a left arteriogram was performed (Fig. 2). An extensive mass of large-sized blood vessels was shown to be present in the left frontoparietal area.

He was given a full course of deep x-ray therapy but continued to show progression of symptoms. He grew markedly weaker on the right and in addition began to complain of occasional headaches, and difficulty with speech. However, he had been free of convulsive seizures after being placed on anticonvulsant medication. He was readmitted to the hospital on Nov. 20, 1942, and was found to have a marked right hemiparesis, a more marked right hemihypäthesia and hypalgesia and in addition a very mild, predominantly motor, aphasia.

On Nov. 30, 1942, a left fronto-temporo-parietal craniotomy was done by Dr. Francis Grant. He stated in his operative note: “On opening the dura, the Rolandic vessel was dilated to the extent of being almost 1 cm. in width. The Sylvian vessel also seemed to be enlarged, but to a lesser degree. Just behind the enlarged Rolandic vessel, at about the level of the hand center, there seemed to be a protruding tumor. In this area there were also 3 or 4 good-sized cortical veins. The walls of the large Rolandic vein were so thin we could see the blood coursing through the vessel” (Fig. 3). It was felt that the lesion was inoperable.

He was readmitted to the hospital on Mar. 4, 1943, for ligation of the left internal carotid artery. In the interim the patient had applied pressure to the left carotid vessels in the neck