INTRODUCTION

The excessive transfer of water from blood to brain tissue during the development of edema is dependent upon a series of interrelated circulatory, vascular and tissue factors. The circulatory factors have long since been the subject of research. The most important role is certainly played by the intravascular pressure which is forcing the water across the vascular walls into the cerebral tissue. The cerebral blood pressure (i.e. predominantly the pressure in the brain capillaries) is, on the one hand, dependent upon the systemic arterial and venous pressures, and, on the other hand, on the cerebral arterial resistance.

Cerebral blood volume may be involved in the development of brain edema, since it depends on the surface area of the cerebral vessel wall, through which water is filtered into the tissue. Although numerous studies of cerebral blood volume have been conducted with different techniques, they have never been related to the development of brain edema.

The present study was aimed at demonstrating the mutual relationships between cerebral blood volume, brain intravascular pressure, and cerebral volume changes during the development of brain edema. To determine these relationships the relevant factors have to be kept constant. This, however, is rather difficult under natural conditions of a living organism where various factors are interdependent. Therefore the study was carried out on a "bio-
logical model", a living animal's body, allowing a firm control of the conditions by the stabilization or alteration of separate processes. The "chest-head preparation" in the rabbit has been found to be a suitable experimental model. Brain edema was produced by repeated induction of cerebral venous congestion.

METHOD

The experiments were carried out on 26 adult rabbits of either sex, weighing about 3 kg, which were anesthetized with Nembutal (pentobarbital Sodium, Abbot). Besides, the animals were treated with the muscular relaxant Tricuran (Gallamine triethiodide) for artificial ventilation during the experiments (the respiratory volume was adjusted to the situation before paralysis).

Preliminary Surgical Procedure

A sagittal incision was made along the midline of the neck. A tracheostomy was performed for artificial ventilation, the right common carotid artery and both external jugular veins were exposed and ligated. Polyethylene catheters of the maximum possible diameter were then inserted into the artery for recording of systemic arterial pressure and into the veins for recording of systemic venous pressure, the i.v. administration of drugs and the taking of blood samples.

To obtain a "chest-head" preparation the circulation in the forelegs and in the hindpart of the body was interrupted. Both subclavian arteries and veins were exposed and ligated immediately outside the chest wall; subsequently the abdominal aorta and inferior vena cava were exposed just behind the diaphragm and polyethylene catheters of the maximum possible diameter were inserted into both vessels to connect them with two separate pressurized reservoir systems filled with Dextran-40 (Fig.1).

A large craniotomy (approximately 20 mm in diameter) was made over the parietal region of the cerebral hemispheres. The dura mater was not opened until the experiments started and was then removed in the area of the craniotomy. Furthermore, the fourth ventricle of the brain was opened by a suboccipital sagittal incision to drain cerebro-spinal fluid.

To prevent blood clotting heparin was injected intravenously (approx. 2,000 units/kg body weight) at the beginning of every experiment. To maintain systemic arterial pressure in the animals (which were deprived of adrenal hormones) norepinephrine was gradually infused into the circulatory system in a dose of approximately 1-2 μg for 5 minutes during the experiments.