THE ROLE OF THE INITIAL ARTERIAL PRESSURE IN THE DEVELOPMENT OF HEMORRHAGIC SHOCK

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An experimental model which has been used for the investigation of hemorrhagic shock consists of rats exposed to sound stimulation [3]. It was shown in this investigation that, with increased excitation of the nervous system (caffeine, parathyroidectomy), there was a considerable increase in the mortality rate from cerebral hemorrhage as the result of exposure to sound. The thyroid gland hormone had a particularly marked influence on the development of hemorrhagic shock [4]. The preliminary administration of thyroidin to animals increased the number of fatal cases after traumatization of the nervous system with a sound stimulus [4].

Further investigations [9] showed that, as the result of transient action (for up to 90 sec) of a sound stimulus, rats showed a considerable fall in the arterial pressure, a slight fall in body temperature and an increase in the blood homo globin. These changes were more marked in rats receiving thyroidin. After the prolonged action of a sound stimulus (up to 20 min) disorders of movement, areflexia and nystagmus were observed. This whole complex of symptoms and signs suggest that, as a result of trauma of the nervous system, a shock-like state develops, against which background cerebral hemorrhage may develop from the action of the sound stimulus and death of some of the animals may ensue.

We observed that the animals which died were more often those with a higher initial arterial pressure. The difference between the initial values of the arterial pressure in the animals dying as a result of the action of the sound stimulus and in those which survived was statistically significant (R = 3.8). We know from reports in the literature that the overwhelming majority of cerebral hemorrhages are the consequence of hypertension [5, 6, 8].

The aim of the present research was to study the role of the initial arterial pressure in the development of states of hemmorrhagic shock as a result of nervous trauma.

METHOD

Experiments were carried out on 206 adult (from 3 to 6 months old) white laboratory rats, mainly males, sensitive to the action of a sound stimulus.

The animals were subdivided into two groups, one of which took part in the experiment in a normal state, and the other was given preliminary treatment with thyroidin.

The thyroidin was given orally through a glass tube daily in a dose of 25-50 mg for ten days. In order to produce hemorrhagic shock in the rats a method developed in our laboratory [3] was used. The method consists essentially of the interrupted action of a sound stimulus on the rat for 15 minutes, followed by a three-minute interval, after which the sound stimulus is applied for 90 seconds. The resulting excitation is very intensive and in several cases terminates by death of the animals from cerebral hemorrhage. Before the beginning of the experiment and

TABLE 1. State of the Arterial Pressure in the Animals Dying and Surviving as the Result of Nervous Trauma

Group of animals	Surviving				Dying			
	number of animals	arterial pressure (mean)			number of animals	arterial pressure (mean)		
		before expt.	after expt.	difference before and after expt.		before expt.	after expt.	difference before and after expt.
Animals receiving thyroidin	46	118	89	—29	12	136	102	—34
Normal animals	51	116	94	—22	1	124	90	—34

TABLE 2. Relationship between the Fall in Arterial Pressure and its Initial Value and the Intensity of the Animal's Response Reaction to the Sound Stimulus

Initial arterial pressure below 120 mm Hg					Initial arterial pressure above 120 mm Hg				
character of the response reaction of the sound stimulus	number of animals	arterial pressure (mean) before expt.	arterial pressure (mean) after expt.	diff. before & after expt.	character of the response reaction of the sound stimulus	number of animals	arterial pressure (mean) before expt.	arterial pressure (mean) after expt.	diff. before & after expt.
Motor excitation with fits	66	105	83	—22	Motor excitation with fits	8	126	81	—45
Motor excitation without fits . . .	17	105	94	—11	Motor excitation without fits	5	127	95	—32

after its conclusion the arterial pressure was determined by the method of Sidney and Friedman [11].

RESULTS

By comparison with the survivors, the initial arterial pressure of the animals which died was higher, and a somewhat greater fall in its value was observed as a result of the nervous trauma (Table 1).

In order to elucidate the relationship between the arterial pressure fall and its intial value, we subdivided the ordinary experimental animals in accordance with the value of their initial arterial pressure into two groups. In one of the groups we included the animals with a pressure not exceeding 120 mm Hg, and in the other, animals in which the pressure was over 120 mm Hg. Both groups of animals were subjected to the action of continuous sound stimulus for 90 seconds.

It will be seen from the results in Table 2 that the fall in arterial pressure as a result of brief exposure to the sound depended on the magnitude of the initial pressure and also on the intensity of the animal's response reaction to the sound stimulus. The level of the arterial pressure after nervous trauma did not depend on the initial pressure, but it did depend on the intensity of the animal's response reaction to the sound stimulus.

What is the role of a high initial arterial pressure in the development of cerebral hemorrhages during the action of a sound stimulus? As a result of the action of the sound stimulus a sharp fall in the blood pressure of the animals took place, and this was more marked in those which died. Several authors have observed spasm of the cerebral vessels and a raised arterial pressure in animals at the moment of excitation, after which a sharp fall in the pressure and dilatation of the cerebral vessels ensued. L. A. Koreisha [2], for instance, points out that over-stimulation of the central nervous system during operation may cause an increase in the arterial pressure, accompanied by a sudden loss of consciousness and, occasionally, by epileptiform convulsions. The increase in the arterial pressure is quickly replaced by a catastrophic fall, with a sharp rise in the pulse rate. In the period of increased arterial pressure severe spasm of the vessels is observed, after which dilatation of both the arterial and venous systems ensues. These disturbances of neurodynamics, as this author observes, lead via dysfunction of the cardiovascular system to the escape of blood through the intact walls of the blood vessels.

On the basis of data in the literature we are inclined to consider that the sound stimulus causes spasm of the cerebral vessels in the animals, which then gives way to dilatation of these vessels and to a sharp fall in the arterial pressure. A high initial pressure is known to facilitate the onset of spasm [1, 2, 5].

The fall n the arterial pressure may thus act as an index of the functional changes taking place in the cerebral vessels. The level of the arterial pressure evidently cannot always characterize the severity of the animal's condition (see Table 2). Cases of severe shock with a comparatively normal pressure are described in the literature [10].

During morphological investigations [7] of the cerebral vessels in rats subjected to the action of a sound stimulus, dilatation of these vessels and hemorrhages into the brain were observed.

SUMMARY

As a result of the action of a sound stimulus there occurs a sharp reduction of the arterial blood pressure in rats, some of which perished from cerebral hemorrhages. Animals with a higher initial value of the arterial blood pressure show a greater percentage of deaths. The results of this experiment demonstrate the relation between the degree of arterial blood pressure drop and the value of the initial blood pressure.

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