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by

Z. Jagodzinsik

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EDITED TRANSLATION

FTD-ID(RS)T-0308-78 31 March 1978

MICROFICHE NR: FTD-78-C-000464

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English pages: 10

Source: Neurologia i Neurochirurgia Polska, Vol. 10, (26), No. 6, Nov-Dec 1976, pp. 793-799.

Country of origin: Poland
Translated by: LINGUISTIC SYSTEMS, INC.
F33657-76-D-0389
R. Van Emburgh
Requester: FTD/TQIC

Neur. Neurochir. Pol. 10, No. 6, 793-799 (1976)
[Polish]

CEREBRAL BLOOD FLOW IN ACCELERATION CRANIOCEREBRAL INJURY *)

by

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Interest in the reaction of cerebral blood vessels to acceleration injury of the brain arises primarily with respect to the role that is attributed to this reaction in the pathology of this type of injury, particularly the post-traumatic lack of intracranial space. As early as 1878 Daret theorized that such an injury causes paralysis of the vasomotor mechanisms in the brain, leading to hemostasis and swelling of the cerebral tissue. Somewhat later, Courtney, dealing with the problem of neurogenic regulation of cerebral vessels, noted that the effect of an injury on vessels in organs other than the brain, e.g., the intestines, causes severe distension of these vessels and suggested that a similar phenomenon occurs following injury to the brain. Circulatory stasis was expected to lead to a condition of "severe cerebral ischemia" and increased osmosis of water into the tissue, as a result of the increase in the hydrostatic gradient in the vessel walls. Based on these hypotheses, Trotter proposed that the essence of acceleration cerebral concussion (acc) might depend on the emergence of microscopic short lived foci of ischemia. On the basis of histopathological studies, Rand and Courville later suggested the action of a similar mechanism, as did Evans and Scheinker, who pointed out the presence of paralyzed capillaries and small veins in brain specimens from people who had died following a severe skull injury. They were the first to express the view that a mechanical in-

*)see last page for footnote.

jury will result in retardation of the cerebral circulatory rate.

Direct observation of vessels in a brain that had suffered an injury, in the meantime, were providing observations that were subject to various interpretations, at times even contradictory. In the majority of studies, constriction of the vessels was observed following an injury, and only in a few was distension observed. Thus, other, more adequate methods of evaluating cerebral circulation in an acceleration injury were continuously sought. One of these turned out to be measurement of the cerebral blood flow (cbf). As early as the 1890's the French researcher Polis became involved with this method. He noted that immmediately after an acceleration injury: 1) the blood flow in the jugular vein of the cat increases significantly; 2) the systemic, arterial blood pressure rises; 3) the increase in flow paralleled the rise in blood pressure, and within certain limits, was proportional to the severity of the injury.

Denny-Brown and Russel later confirmed these observations, finding that in animals, following a "subconcussive" blow to the brain, a marked increase in blood flow in the jugular vein occurs, even when the systemic blood pressure was dropping. German, et al. obtained an increase in cerebral flow of 86% relative to the control values in dogs who had suffered a skull injury.

This substantial increase in flow occurred with slight changes in the systemic blood pressure and despite the sometimes significant losses associated with the choice of tests and the operating procedure itself; nevertheless, it was always accompanied by an increase in the cerebral metabolic rate for oxygen ($CMRO_2$), on the average, 42%, in the first three hours after the injury. A dozen or so years later, Brown, et al., using an improved electromagnetic flowmeter, found that the blood flow in the internal carotid artery of monkeys that had suffered a cerebral concussion dropped by 24% in the first three seconds, then increased significantly, achieving a value 250% higher than the control level after 30 seconds. By the end of 6 min. following the injury, a return to normal had occured; Similar fluctuations were observed in the behavior of the systemic arterial blood

pressure. Mayer and Denny-Brown showed that a substantial increase in blood flow in the cortex can occur even when acceleration cerebral concussion is not accompanied by any blood pressure change. These authors related the increase in cortical flow to the initial, post-traumatic distension of the cerebral vessels.

At this point, some comments seem necessary relating to the behavior, in animals following an acceleration skull injury, of: 1) the systemic arterial blood pressure; 2) the cerebral circulatory rate; and 3) changes in the so-called cerebral blood volume. These parameters have a secondary effect on the cerebral blood flow and should always be taken into account when considering the post-traumatic changes in cerebral circulation.

As a rule, in experimental animals we observe an increase in the arterial blood pressure that represents an almost constant and instantaneous (about a 3 sec. delay) vasomotor reaction to an acceleration injury of the brain stem. This occurs not only when the injury causes direct destruction of the bulbar vasomotor center, which manifests itself by a rapid and irreversible collapse in circulation, but when strong excitation of the depressive centers of the vagus nerve suppresses the instinctive contraction of peripheral vessels, sometimes leading even to a drop in blood pressure. Denny-Brown's contribution was the demonstration that post-traumatic paralysis of the vasomotor center can also have a functional, reversible nature.

Following blockage of the vascular reaction (adrenalectomy, administration of yohimbine or cervical myelotomy) animals never react to an injury with a rise in blood pressure. This would seem to indicate that the sympathetic nervous system is instrumental in this reaction and that it begins in the vasomotor center of the bulb. The peripheral and intracranial part of the vascular bed behave differently here: constriction of the peripheral vessels is accompanied by distension of the intracranial vessels.

The cerebral circulation rate undergoes a rather substantial retardation as a result of acceleration cerebral concussion. Rockoff and Ommaya (20) observed in monkeys a considerable lengthening in the cerebral circulation time, 15-30 minutes after the injury. Since these authors observed narrowing of the lumen in cerebral arteries only in one animal of their series, they concluded that the reason for retardation in flow rate might be not merely contraction of cerebral vessels, but also intravascular agglutination or post-traumatic swelling of the brain, which develops sometimes even within 10 to 20 seconds after the injury (11).

Recently, similar experiments with rats were performed by Ekelund, et al., who found, in addition to retardation of the flow rate, that the vascular bed of the brain has a multiphase reaction to injury and following the initial retardation of circulation, it returns to normal, after which the circulation time again becomes longer.

Immediately after an injury, the so-called cerebral blood volume also undergoes an change. White, et al. did not find any significant change in the cerebral blood volume (cbv) in cats, following an acceleration injury inflicted in the occipital region with a pendulum. On the other hand, Langfitt, et al. demonstrated a twofold increase in cbv after a craniocerebral injury, but the technique used by them did not permit precise differentiation between the intra- and extravascular blood. Since intracranial circulation is always present during an injury, and the change in obv [Translator's Note: sic] observed by Langfitt amounted to only 0.5 ml, it is impossible to state with complete certainty on the basis of these experiments that cbv undergoes a change following injury. A number of observations of Lewis et al., in which, in cats 30 min. after an injury, the cbv increased almost fourfold - from 1.8 ml to 7 ml, which is about 50% of the intracranial volume of the animal - suggests such a possibility, however. A few authors (9, 10, 11) believe that it is precisely this increase that is the direct reason for post-traumatic increase in intracranial pressure.

In the light of the above considerations we can assume that after a cerebrocranial injury, distension of vessels usually occurs simultaneously with a retardation in circulation. Certain interpetative difficulties arise here, however, specified as follows by Langfitt, et al. At the same time with an increase in blood flow in the common carotid artery, a retardation or slowing down of circulation appears, demonstrated without any doubt by angiography of the cerebral vessels. If we adopt the changes in the lumen of the cerebral vessels as the principal mechanism that regulates the amount of cerebral blood flow, then distension of the capacitive vessels (capillaries and small veins) with simultaneous slowing down of the circulation rate indicates the presence of increased vascular resistance situated peripherally from the cerebral veins. Such a peripheral venous obstruction occurs in acceleration injury of the skull very seldom; only with a substantial and long-term increase in intracranial pressure.

The slowing down of the circulation rate itself can be explained by intravascular agglutination of erythrocytes, repeatedly described in other regions of capillary circulation that have suffered an injury. This cannot be the reason, however, for the simultaneous increase in cbv. If we assume the existence of arterial-venous connections in cerebral circulation, probably localized in the subcortical regions, the coexistence of capillary retardation (associated with the increased seepage of water on the outside and with cerebral edema) with an increase in both blood flow and cerebral blood volume would be explained by their opening. However, with such an assumption,the flowing time for a dye introduced to the cerebral circulation would have to be shortened, whereas is reality the reverse is true and the divergence between the cbf and cbv values and the cerebral circulation rate remains as before.

Another interpretation could be based on the relationship between cbf and cbv on the one hand, and intracranial pressure on the other. Earlier work (26) using a hole in the fornix of the skull showed that arachnoidea vessels expand as the intracranial pressure rises. These

studies became the basis of a hypothesis, according to which the lack of intracranial space causes changes in the tonus of walls of cerebral vessels leading to an ever greater distension of these vessels, and finally to motor paralysis. This would then be identical to the mechanism described by Duret in posttraumatic conditions. Under normal conditions, every stimulus that dilates a cerebral blood vessel will simultaneously increase the blood flow. However, with a gradually developing distension in the cerebral vascular bed, the blood volume (cbv) naturally increases, along with the intracranial pressure. If this process continues without arrest, the distension of vessels will finally be counterbalanced and suppressed by the increasing intracranial pressure.

In the final stages of motor paralysis in vessels, when they are at maximum distension, the intracranial pressure will approach the arterial blood pressure, the cerebral perfusion pressure will drop to zero and blood flow will stop. If under these conditions the blood pressure in the carotid artery and jugular vein is normal, then this stoppage in flow must depend on constriction of some part of the cerebral vascular network, probably venous outflow passages.

A relatively large amount of time and effort has been devoted to studies of the overall blood flow under conditions of both severe and chronic post-traumatic syndrome of the brain. On the other hand, not many studies have analyzed the changes in regional cerebral blood flow (rcbf) in an experimental craniocerebral injury (19). The pathogenesis of acc still contains a number of unsolved problems and for this reason we can surmise that a study of disturbances in regional cbf occurring immediately after an injury will shed some light on the mechanisms associated with, e.g., loss of consciousness after a shock, vegetative disturbances, suppression of reflexes, etc. The theoretical condition for studies on rcbf in acceleration injury was the opinion that in such an injury at least 3 fundamental factors affected the nervous system: deformation of the skull (local and remote), linear

changes in intracranial acceleration, and finally - differences in angular acceleration.

All of these factors lead to displacements, tension and tearing of cerebral tissue, different in various regions of the brain. They also affect the vascular system of the brain, leading to changes in blood flow in separate regions of it. In studies dealing with rcbf it was mainly a matter of investigating possible differences in flow at the cortex and brain stem level; experimental concussion, however, creates particularly difficult conditions for these studies. The animal's head must be unrestrained (considerably easier to achieve before concussion, than with an abnormally immobilized neck) and at the same time all the fundamental physiological parameters must be recorded: blood pressure, respiration, pulse, gasometry, and finally, rcbf. Each somewhat more severe injury easily leads to a reaction in systemic circulation, which secondarily affects the cerebral circulation very quickly and fundamentally.

2 techniques are generally used in these studies: 1) intra-arterial administration of krypton^{85} and determination of rcbf in two homologous regions of the brain (e.g., left and right hemisphere), or 2) the rcbf values are determined after intravenous administration of an isotopic marker, e.g., antipyrine C^{14}, based on autoradiography of 20-micron brain sections.

Using the latter method the author of (14) conducted a series of tests in rats, the results of which enable us to surmise that acceleration craniocerebral injury leads (in the central nervous system) to: 1) paralysis of autoregulation in cerebral circulation as a result of which the cerebral blood flow follows passively behind the fluctuations in systemic blood pressure; 2) disturbances is flow are multiphase; 3) their predominate feature is the significant degree of ischemia that appears even within 2-3 minutes after the injury.

Studies (13, 19) on the behavior of cerebral blood flow following

an acceleration injury were also conducted with people in which, quite naturally, the time period involved had to be ~~less~~ more or ~~more~~ less distant from the moment of injury. The authors of these studies usually mention two corresponding mechanisms for post-traumatic ischemia in the brain and the drop in cbf. The first of these is the failure of the so-called "secondary inflow of blood". This phenomenon consists of the maintenance of a very low cortical flow despite the restoration of of the cerebral perfusion pressure to normal (cpp). Ames offered a theoretical explanation of this phenomenon in 1968, also calling it "no-reflow phenomenon". A second mechanism could be the critical drop in cerebral perfusion pressure owing to the increase in the lack of intracranial space to a level close to the average arterial blood pressure. This mechanism usually acts in the later periods of the injury, when the increase in intracranial pressure is dependent on the increasing swelling in the brain. In principle, the prevailing view is that edema and the lack of intracranial space following a skull injury are generally proportional to its severity. Two types of post-traumatic lack of intracranial space are usually distinguished here, dependent on: 1) the distension of cerebral vessels and an increase in the blood volume contained in them (increase in cbv), and 2) compression of vessels by hematomas or edema with subsequent drop in blood flow, anoxia, hypercapnia and swelling of gliomatous cells. When a mechanical injury, mainly in its initial stages, disturbs or paralyzes autoregulation of cerebral circulation and handicaps the tonus in walls of the resisting vessels, then the value of cbf will obviously reflect the fluctuations in systemic blood pressure passively and be parallel to them.

Overgaard notes that in patients after craniocerebral injury, with handicapping mechanisms that regulate the cerebral blood flow, it is possible to distinguish at least two types of circulatory disturbances: ischemic and hyperemic. In characterizing the first of these this author cites his own observation of patients with very low (lower than 20 ml/100 g/min) cbf values. These patients as a rule died, or if they survived the injury, then it was in a condition of complete decortification; the cbf maintained itself in them at a level of 10 ml/

100 g/min, despite the restoration of normal cpp values. Overgaard also believes that the low ischemia values of cbf are a poor prognostic symptom, and for the most part cannot be reconciled with the recovery of a normal functional condition in the cerebral cortex.

The second, most frequent type of cerebral circulatory disturbance, associated usually with post-traumatic functional losses in the neurologic sense, is the so-called "luxury perfusion syndrome". It is analogous to the "luxury perfusion" long since described, that in experimental animals is the reactive congestion in conditions of anoxia and ischemia, caused in essence by acidification of tissues by lactic acid. In Overgaard's patients this syndrome usually occured at a certain period after the injury and remained for about two weeks. It is obvious that short-lived lactate acidosis characteristic of "luxury perfusion" cannot explain the long-term increase in cbf; he believes therefore, that in these patients either new, not always observable "episodes of anoxia" appear, or the increase in systemic blood pressure causes an increase in seepage of fluids from the vascular bed into the cerebral tissue, leading to edema and secondary brain damage.

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Received: 1/20/1976

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Footnote to page 1:

*) Based on a lecture at the 7th Scientific Conference on the subject: "Cerebral Blood Flow in Human Physiology and Pathology," Vascular Commission of the Committee on Neurological Science, Polish Academy of Sciences, Bydgoszcz, 10/25/1975.

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