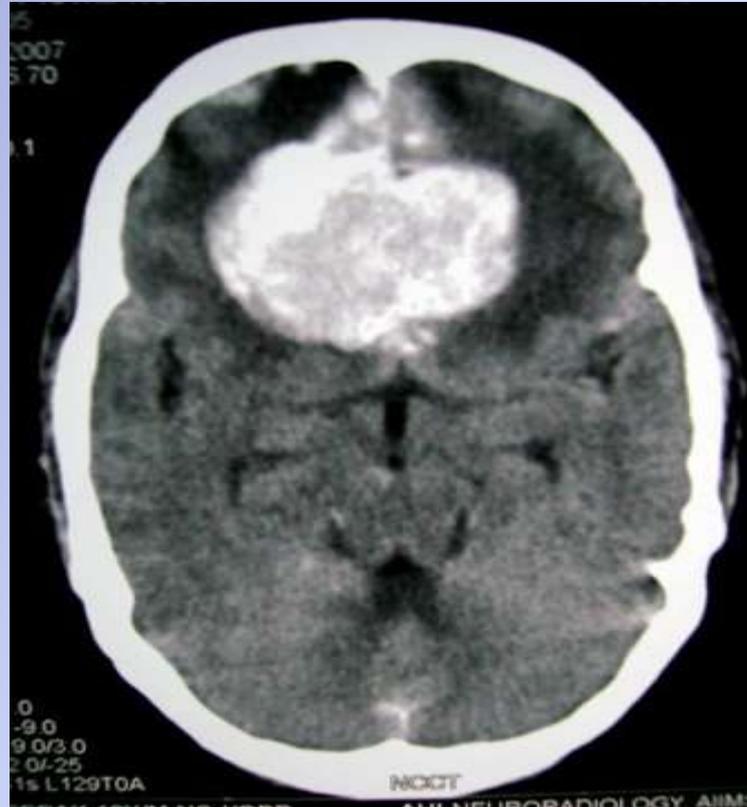


New Evidence about Intracerebral Hematomas Pathogenesis

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(Experimental data)

The present work is another attempt to study pathogenesis of hemorrhagic stroke; the analysis is based on the several well-known facts in medical practice and the experimental data. Sudden, uncontrolled changes of arterial blood pressure results in recurrent hemorrhage, ICP growth and perfusion blood pressure reduction. For arterial blood pressure correction ganglioblockers and peripheral venodilatatores should be used. Sudden decrease of intracranial pressure leads to sharp expansion of vascular diameter prior to hydrodynamic attacks (for example due to lumbar puncture). It may be one of the reasons of sudden deterioration of neurological status. Also likely to be assumed, that a strong headache due to aneurism rupture is caused by blood intrusion into the ventricular system.

Key words: Hemorrhagic stroke, pathogenesis , ICP, perfusion , ventricular system.

Introduction: Spontaneous intracerebral hematoma is an actual unsolved problem for modern neurology and Neurosurgery. Every year 20-25 new cases are registered per 100,000 people, mortality reached 45%. According to the Ayala the same ratio of hemorrhagic stroke is found in females and males, but significantly exceeds the lethal outcome in men. **Etiology:**

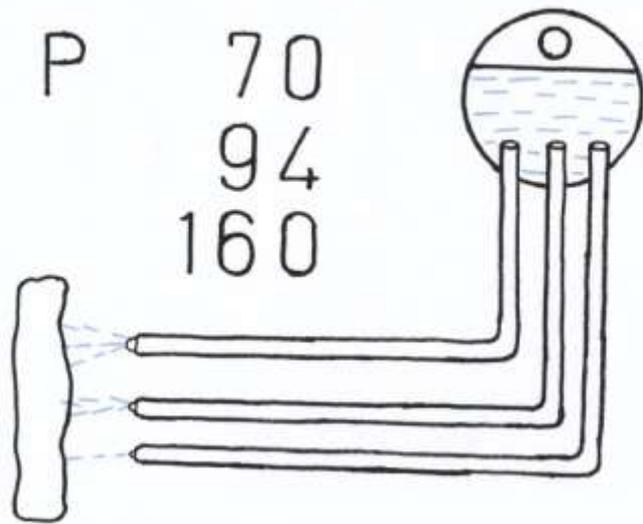
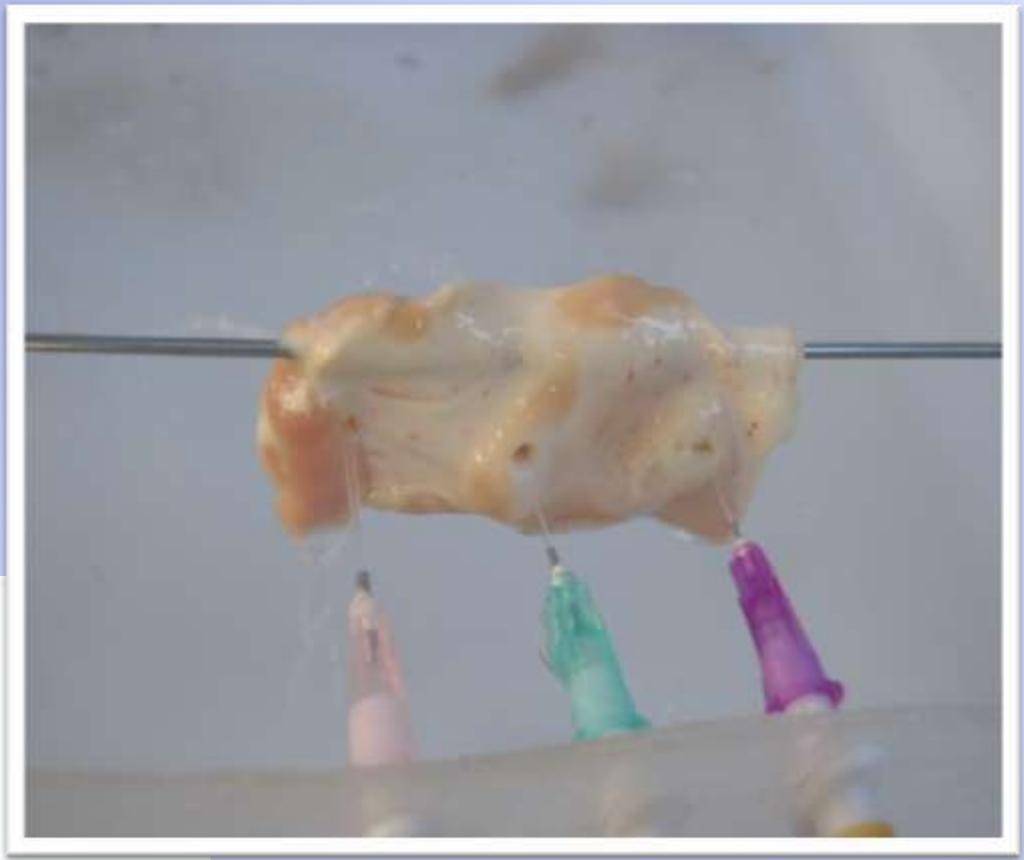
1. Arterial hypertension.
2. Arterial - venous malformation.
3. Rupture of arterial aneurisms.
4. Brain amyloid angiopathy
5. Brain neoplasm.
6. Coagulopathy.
7. Transformation of ischemic stroke in to the hemorrhagic.
8. Brain vein thrombosis.
9. Moya Moya deases.
10. Vasculitis and etc.

In especially high-risk group are patients with arterial hypertension, diabetes, alcoholism, excessive use of tobacco. Hypocolesterinemy, excessive alcohol and tobacco consumption, male gender, is associated with deep intracerebral hemorrhage. Hypocolesterinemy, diabetes melitus, old age - with lacunar hemorrhage. ruptured aneurism or arterial blood vessels are sources for intracerebral hematoma development. Around the hematoma pathomorphologic picture is represented with swelling, neuronal damage, accumulation of neutrophils and macrophages. It should be noted that the blood spreads through the white substance and distracts it. Intracerebral hematomas has tendency of growth. On the bases of observation of 103 patients Brott found out, that in 26% the volume of hematoma has increased in an hour, and in 12% - in 20 hours - the same data were delivered by Kazui. Hematomas volume growth is connected with the continuation of bleeding. Hematomas volume increase causes the deteriorating patient's condition in the first 3 hours; further deterioration of the situation is mainly due to the brain swelling.

- In most cases intracerebral hematomas are developing as a result of arterial or arteriole rupture, therefore the pressure in the hematomas cavity goes close to the pressure in the arteries or systemic arterial pressure. The pressure increasing in the hematoma cavity causes strong compression of adjacent structures and intracranial pressure rising. Oligemia is marked in the surrounding brain tissue, and this area can significantly exceed the hematoma volume.
- Based on the above-mentioned there are two very important issues.
- How to arrange management for intracerebral hematomas?
- How to avoid recurrent hemorrhage?

The present work is another attempt to study pathogenesis of hemorrhagic stroke; the analysis is based on the several well-known facts in medical practice and the experimental data.

Neurosurgeons are aware, that the spontaneous intracerebral hematoma (non-aneurismal) almost always has a pronounced border with the brain substance, It is very rare to find the brain imbibition with blood. To create an experimental model of hemorrhagic stroke it is not enough to inject a certain amount of blood into the brain tissue, but it is necessary to destruct the brain tissue by different methods (mechanical, electrical, hydraulic). Less effectiveness of surgical treatment of hemorrhagic stroke. Removal of Large sized hematomas did not improve or improves slightly the patient's condition. It raises great interest the way of getting the blood into the ventricular system, the experiment below is an attempt to explain this fact. The brain tissue (thickness 0.7-1 cm) was placed in front of the three tubes with different diameters - 1.2 mm, 0.7 mm, 0.5 mm, at the distance of 0,7-1 cm, after opening the taps the water jet causes the brain tissue mechanical destruction, its intensity depends on the tube diameter and height of the water column. (For example, water jet from the 1.2 mm tube under 94 mm pressure destructs 1 cm thick brain tissue fragment in about 10 minutes , water jet from the 0.5 mm tube creates 1-2 mm deep cave in an hour).



Mechanical destruction of the brain tissue due to aneurysm rupture strongly depends on the blood vessel defect diameter and arterial blood pressure. Perhaps, this is the only way of blood spreading into the ventricular system. It should be mentioned, that the patient's neurological condition first of all depends on intensity of mechanical destruction of the brain tissue with the blood jet and then on the compression of surrounding tissue by the hematoma.

Modeling of indicators for vascular diameter, systemic arterial blood pressure, arterial blood volume and intracranial pressure.

- D- Diameter of the tube.

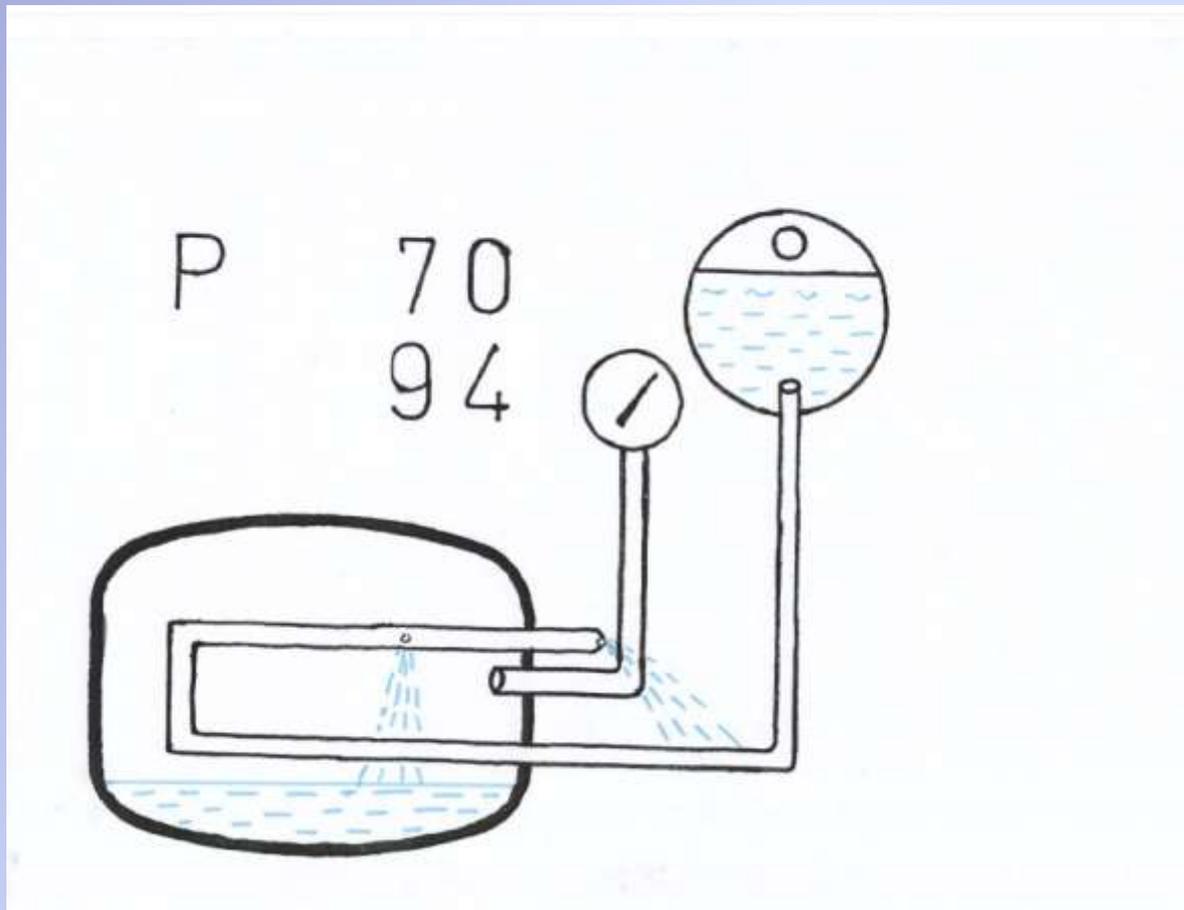
P- Systemic pressure

P's –pressure level inside the container, which stops water jet.

W - water volume poured out per minute.

D1=1,9mm	P=70mmHg	350ml/wT	Ps= 35,2mmHg
D2=1,2mm	P=70mmHg	120ml/wT	Ps= 59 mmHg
D1=1,9mm	P=94mmHg	420ml/wT	Ps= 36,6mmHg
D2=1,2mm	P=94mmHg	140ml/wT	Ps= 78,5mmHg

- scheme represents ruptured vascular experimental models.
- Considering the following model we have to take into account following factors.





- The scheme represents hermetic closed container, in which there is inserted latex tube with the wall hole (0.5 mm). Pressure inside the container is measured with manometer. Experiment was held at different pressure (70 and 94 mm Hg). After opening the taps water jet streams through the distal tube and into the container, but when the pressure inside the container reaches a certain level the water stream from the damaged pipe stops. Pressure level inside the container enough to stop the flow depends on the height of the water column. The higher is the water column the higher pressure inside the container is required to stop the water-flow.
- Very interesting fact was noticed during the sudden change of pressure. In case of sudden decrease of the high systemic pressure, the pressure inside the container causes total occlusion of the tube, the so-called “vessel shock” (This can be considered similar to the position change of parts of the body, cough etc.).

Results:

After stopping of water jet inside the container the pressure in the tube goes close to the systemic pressure. So increased systemic blood pressure and permanent water jet from the tube hole causes the proportional increase of pressure inside the container.

Increase of the pressure at the end of the distal tube determines rise of pressure inside the container and reduces water volume in the tube.

- Increasing of systemic pressure proportionally induces rise of pressure inside the container, but due to decreasing of systemic pressure, the pressure inside the container practically does not change.

Sudden increase of pressure inside the container directly influence on the tube diameter and develops the sharp collapse of the tube, so-called “vessel shock”.

Discussion:

- Sudden, uncontrolled changes of arterial blood pressure results in recurrent hemorrhage, ICP growth and perfusion blood pressure reduction.
- For arterial blood pressure correction ganglioblockers and peripheral venodilatatores should be used.
- Sudden decrease of intracranial pressure leads to sharp expansion of vascular diameter prior to hydrodynamic attacks (for example due to lumbar puncture). It may be one of the reasons of sudden deterioration of neurological status.
- Also likely to be assumed, that a strong headache due to aneurism rupture is caused by blood intrusion into the ventricular system.

ახალი მონაცემები ინტრაცერებრული ჰემატომების პათოგენეზის შესახებ.

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ეს ნაშრომი წარმოადგენს ჰემორაგიული ინსულტის პათოგენეზის შესწავლის კიდევ ერთ მცდელობას, ანალიზი ემყარება საექიმო პრაქტიკაში კარგად ცნობილ რამოდენიმე ფაქტს და ექსპერიმენტულ მონაცემებს. მიუხედავად იმისა, რომ აღნიშნული ექსპერიმენტი ქალას ღრუში მიმდინარე პათოლოგიური პროცესების მხოლოდ შორეული ანალოგიაა, შეიძლება ვივარაუდოთ, რომ პაციენტთა მკურნალობა ინტრაცერებრული სისხლჩაქცევების დროს უნდა წარიმართოს დაბალი საშუალო არტერიული წნევის და დაბალი ცენტრალური ვენური წნევის ფონზე. ვლევის შედეგებიდან გამომდინარე შესაძლებელია ვინსჯელოთ, რომ არტერიული წნევის უეცარ, უკონტროლო ცვლილებას მოჰყვება რეჰემორაგია, ინტრაკრანიალური წნევის ზრდა, პერფუზიული წნევის შემცირება. არტერიული წნევის კორექციების დროს გამოყენებულ უნდა იყოს განგლიობლოკერები და პერიფერიული ვენოდილატატორები. ინტრაკრანიალური წნევის უეცარი შემცირება იწვევს სისხლძარღვთა სანათურის მკვეთრ გაფართოებას ჰიდროდინამიკურ დარტყმამდეც კი (მაგ. ლუმბალური პუნქციის დროს.) რაც შეიძლება წარმოადგენდეს ნევროლოგიური სტატუსის უეცარი გაუარესების ერთერთ მიზეზს. დიდი ალბათობით შეიძლება ვივარაუდოთ, რომ ძლიერი თავის ტკივილი ანეგრიზმის გასკდომის დროს აღმოცენდება მაშინ, როცა სისხლის ჭავლი შეაღწევს პარკუჭოვან სისტემაში.