CLASSIFICATION

I. BY CEREBRAL BLOOD CIRCULATION POOL (REGION OF BLOOD CIRCULATION)

A. Carotid pool B. Vertebrobasilar pool

II. BY LOCALIZATION OF THE PATHOLOGIC PROCESS

A. Extracranial segments of the carotid and (or) vertebral arteries

B. Intracranial segments of the carotid and (or) vertebral arteries

III. BY ATTITUDE TO THE ARTERY WALL

A. Intravascular pathology

B. Extravascular pathology

C. Complex extra-intravascular (perivascular) pathology

D. Primary and (or) secondary pathologic factors

IV. BY PATHOGENESIS

A. Congenital (inborn) pathology, variants

Artery elongation

Artery tortuosity

Artery kinking

Artery coiling

Hypoplasia (aplasia) of the carotid (vertebral) artery

Ectopia of the entrance and exit of the vertebral artery /ostial lateral dislocation of the vertebral artery, high entrance in the vertebral artery canal, exit from posterior wall of the vertebral artery etc. /

Anomaly name by Kimmerly

Anomaly name by Chiari (malformation)

Anomaly name by Klippel - Pheily

Circle of Willis anomaly / trifurcation etc./

Vascular malformation /artery fusigorm and mycotic aneurysm, arteriovenous malformation (AVM), carotid-cavernous malformation, etc./ Miscellaneous

B. Developmental (acquired) pathology, variants

Vessel compression / stenosis, occlusion / by the bone, cartilage and ligaments (ex.: vertebral artery in its canal), by muscle (ex.: syndrome of anterior scalene muscle), by fibrous tissue (ex.: inflammation, proliferation, hyperplasia, etc.)

Atherosclerosis (atherosclerotic vascular stenosis or occlusion)

Thrombosis /more often by floated thrombi from large neck artery/

Embolism /more often cardioembolic stroke/

Thromboembolism

Vessel damage / intracavernous carotid aneurysms with traumatic fistulas; traumatic stretching with dissection (rupture), perivascular hematoma, full or partial vascular wall rupture; aneurysm (as a result penetrating depressed fractures, gunchot wounds, knives, iatrogenic, closed head injury, tethering, supraclinoid carotid artery local injury), etc./

Vessel disfunction (vasospasm, vasodilation, vasoparalysis, etc./ Vasculopathy and Vasculitis / Moyamoya Disease (is a chronic progressive occlusive cerebrovascular disease characterized angiographically by the proximal stenosis or occlusion of multiple intracranial arteries); Takayasu's arteritis (presented often with progressively severe vertebrobasilar transient ischemic attacks), cerebral amyloid angiopathy, fibromuscular dysplasia, etc./

Venous pathology /occlusion, thrombosis, disfunction, venous back pressure/ Others causes / neoplastic (cavernous angioma), infectious, coagulopathy (hypercoagulable states), post operative, drug related, hypoxia, hypotension, hypertension, medical (surgical) reduction of intracranial pressure, effects of alcohol and smoking, etc./

V. BY TYPE OF THE ACUTE CEREBRAL BLOOD CIRCULATION DISTURBANCE

A. Ishemic stroke (about 80% of cases)
B. Hemorrhagic stroke (cerebral hemorrhage - 5-10% of cases)
C. Subarachnoid hemorrhage (10-15% of cases)

VI. BY TYPE OF THE ACUTE CEREBRAL ISCHEMIA /ISHEMIC STROKE/

1 by duration / transient disturbance of the blood circulation - transient ischemic attacks **(TIA)** (regression of the neurological deficit during 24 hours from the moment of it's appearance); **small stroke** (regression of the neurological deficit during 3 weeks - did not included in the International classification of diseases and death reasons); **large** (massive) **stroke** (focal neurological deficit continue more than 3 weeks)/;

2.**by severity of patients condition /small stroke** (non expressive neurological deficit, which are full regressive during 3 weeks from the moment of it's appearance); **moderate stroke** (without clinical signs of brain edema, without consciousness deterioration, with focal neurological symptoms determination in the clinical signs); **severe stroke** (expressive general (common) head symptoms with consciousness deterioration, signs of brain edema, vegeto-trophic disturbance, severe focal deficit, often dislocation symptoms);

3.**by localization of the brain infarct** /is founded on the focal neurological signs topical characteristic correlation to determine arterial pools (middle, anterior and posterior cerebral arteries, basilar artery and their distal branches)/;

4. **pathogenic classification** / regional infarct (often as a result of large artery thromboembolic stroke; symptoms, as a rule, correlate to main vessels pools circulatory disturbances); **"border region" infarct** or in terminal branches large cerebral artery (as a result of quickly decrease of the perfusion pressure on large cerebral artery peripheral branches); **lacunar infarcts** in the region of thalamus, capsule internal or brainstem, and [a]lso small multiple focuses in the cerebral hemisphere white substance (as a result repeated niicro emboli or local circulatory disturbances in the region of small arteries angiopathy)/.

VII. BY PERIOD OF THE STROKE / Gusev E.I., 1997/

A. Acutest period - first 3-5 days from the stroke
B. Acute period - up to 21 day from the stroke
C. Early recovery period - up to 6 months after stroke
D. Late recovery period - up to 2 years after stroke