Stroke

Ravshanova M.M
Alieva N.S.

ABSTRACT

Stroke is a type acute cerebrovascular accident (ONMK) and is characterized by a sudden (within minutes, less often - hours) by the appearance focal neurological symptoms (movement verbal, speech, sensitive, coordinating natorium, visual and other disorders) and/or general brain disorders (suppression consciousness, headache, vomiting, etc.), which persist for more than 24 hours or result in to the death of the patient in a short period time due to the cause of cerebrovascular lar origin.

Keywords: stroke, diagnostic, treatment, general practice

There are two clinical and pathogenetic forms of stroke: 1) ischemic stroke (cerebral infarction) is caused by acute focal cerebral ischemia, leading to a heart attack (area of ischemic necrosis) of the brain; 2) hemorrhagic stroke (non- traumatic intracerebral hemorrhage) is caused by rupture of an intracerebral vessel and penetration of blood into the parenchyma of the brain or rupture of an arterial aneurysm with subarachnoid hemorrhage. Stroke also includes transient disorders of cerebral circulation, characterized by the sudden appearance of focal neurological symptoms that develop in a patient with cardiovascular disease (arterial hypertension, atherosclerosis, atrial fibrillation, vasculitis, etc.), last several minutes, less often hours, but no more 24 hours, and end with a complete restoration of impaired brain functions.

Transient disorders of cerebral circulation include: 1) transient ischemic attack (TIA), which develops as a result of short- term local cerebral ischemia and is characterized by sudden transient neurological disorders with focal symptoms; 2) hypertensive cerebral crisis, which is a condition associated with an acute, usually significant, increase in arterial pressure (BP) and accompanied by the appearance of common cerebral (less often focal) neurological symptoms secondary to hypertension. The most severe form of hypertensive crisis is acute hypertonic encephalopathy, the basis of the pathogenesis of which is cerebral edema.

Brain infarction, as a rule, is the result of the interaction of many etiopathogenetic factors, which can be divided into local and systemic:

1) local: morphological changes of brachycephalic or intracerebral arteries (pathological disease, etc.), atherosclerotic lesions of the vessels of the aortic arch and cerebral arteries, heart pore as a source of thromboembolic infarcts of the brain, fibromuscular dysplasia of the walls of brachycephalic and cerebral arteries, dissection of brachycephalic arteries, vasculitis (arteritis), changes in the cervical spine with the formation of extravasal compression of the vertebral arteries, anomalies in the structure of the vessels of the neck and brain (hypoplasia of the vertebral artery, tri- furcation of the internal carotid artery), etc.

2) systemic factors: disorders of central and cerebral hemodynamics (a sharp change in blood pressure or a decrease in cardiac output , etc.), hereditary and acquired coagulopathies, polycythemia, certain forms of leukemia, hypovolemia, psychoemotional stress/distress, etc., hypercoagulation/ hyperaggregational side effect of a number of medications (oral contraceptives, etc.). Among the main causes of intracerebral hemorrhage , the following can be distinguished: 1) prolonged arterial hypertension with a crisis course - the cause of more than 50% of hemorrhagic intracerebral strokes; 2) cerebral amyloid angiopathy - about 10-12%; 3) taking anticoagulants - about 10%; 4) brain tumors - about 8%; 5) all other causes account for about 20%.

Risk factors The most important modifiable (correctable) factors that increase the risk of stroke are: arterial hypertension of any origin, heart disease, atrial fibrillation, lipid metabolism disorders, diabetes mellitus, pathology of the main arteries of the head, hemostatic disorders. The main unmodified (uncorrectable) risk factors include gender, age, ethnicity , heredity. There are also risk factors associated with a violation of a healthy lifestyle: smoking, overweight , low level of physical activity, poor nutrition (in particular, insufficient consumption of fruits and vegetables, abuse of alcoholic beverages), prolonged psycho-emotional stress or acute stress. The prevalence of the main risk factors in our country is quite high: 59.8% of adult men and 9.1% of women smoke, have arterial hypertension 39.9 and 41.1%; hypercholesterolemia — 56.9 and 55.0%; obesity — 11.8 and 26.5%, respectively ; 12.0% of men and 3.0% of women consume alcohol excessively [2].

An increase in systolic and diastolic blood pressure in arterial hypertension is the most significant risk factor for both ischemic and hemorrhagic stroke. In patients with severe arterial hypertension, stroke develops 7-10 times more often than in people with normal blood pressure. With an increase in diastolic blood pressure for every 10 mm Hg , the risk of stroke increases by 1.95 times. Stroke mortality doubles with an increase in systolic blood pressure for every 10 mmHg, starting from 115 mmHg.

Reducing the increased level of diastolic blood pressure by 5 mm Hg and systolic blood pressure by 12 mm Hg reduces the risk of stroke by 34 and 36% respectively. The most significant cardiac risk factor for ischemic stroke is atrial fibrillation (atrial fibrillation), in which the risk of stroke increases by 3-4 times. With ischemic heart disease, the risk of stroke increases by 2 times, with left ventricular myocardial hypertrophy detected on an ECG - by 3 times, with heart failure — by 3-4 times. Smoking increases the risk of ischemic stroke by 2-4 times, subarachnoid hemorrhage by 3-4 times, ischemic heart disease by 3-6 times. The degree of risk depends on the intensity (number of cigarettes smoked per day) and duration (number of years) ku- reniya. Systematic abuse of alcohol increases the risk of stroke, especially hemorrhagic, by 2 times [3].

Carotid system of cerebral blood supply (arteries: carotid, orbital, medial cerebral, anterior cerebral). 1. Motor disorders: hemiparesis/hemiplegia on the side opposite to the lesion of the brain. Weakness, awkwardness and stiffness in the arm and/or leg. Often there is a combination of damage to the arm and the lower facial muscles. Motor disorders develop on the opposite side of the body in relation to the affected artery. In weakened limbs, there is a gradual increase in muscle tone according to the pirated type: the tone prevails in the flexors of the forearm and fingers, as well as in the extensors of the lower leg, the phenomenon of the "folding knife". When the deep parts of the frontal lobe are affected, which are supplied with blood from the anterior cerebral artery basin, muscle tone in the paralyzed limbs can increase according to an extra- pyramidal (plastic) type: tone is higher in the flexor muscles of the arm and leg, the phenomenon of "gear wheel", plastic instability and prolonged involuntary stagnation- holding the limb in a certain position. On the hemiparesis side, there is necessarily an increase (revival) of deep reflexes and an expansion of their reflexogenic zone. Pathological reflexes appear that are

absent in the norm: in the hand - the Rossolimo reflex, in the leg — the Babinsky reflex.

2. Sensitivity disorders: sensory disorders in the form of a decrease/absence of pain and other types of sensitivity (numbness , hypesthesia, anesthesia), paresthesia. Hemitip is more often noted (half of the face, trunk, arm and leg) on the side opposite from the brain lesion. With extensive stroke foci in the area of deep brain structures (inner capsule) , there is a combination of hemiparesis and hemianesthesia on the opposite side.

3. Speech disorders: difficulty in finding the right words, slurred and indistinct speech, difficulties in understanding the speech of others (sensory and motor aphasia). Often , speech disorders are combined with a violation of writing (dysgraphy) and reading (dyslexia). Dysarthria in the form of blurred and inarticulate speech, slowness of speech, impaired pronunciation of words and articulation may be noted when the branches of the anterior cerebral artery are damaged.

4. Visual disturbances: blurred vision within the visual fields of both eyes (lower quadrant hemianopia occurs when the upper parts of the visual pathway are damaged in the temporal lobe, upper quadrant hemianopia occurs when the lower parts of the visual pathway are damaged in the temporal lobe). The involved visual field is opposite to the side of the affected artery and the focus of the stroke. Monocular blindness as a result of an acute decrease in vision in one eye on the side of the affected internal carotid artery (for example, with its extracranial occlusion). 5. Gaze paresis: restriction of arbitrary combined movement of the eyeballs in the direction opposite from the focus of the stroke. Cortical paresis of the eye is associated with damage to the posterior parts of the second frontal gyrus - the cortical branches of the middle cerebral artery.

5. Gaze paresis: restriction of arbitrary combined movement of the eyeballs in the direction opposite from the focus of the stroke. Cortical paresis of the eye is associated with damage to the posterior parts of the second frontal gyrus - the cortical branches of the middle cerebral artery.

6. Deviation of the tongue: occurs in the direction opposite to the focus of the stroke (the tongue is deflected towards the paralyzed limbs). At the same time, the whole tongue is rejected, and not the end , as with the defeat of the sublingual nerve. There are no atrophy and fasciculations in the muscles of the tongue. The deviation of the language is due to the fact that half the muscles of the tongue are innervated only by the opposite hemisphere of the brain (all corticonuclear fibers are crossed). 7. Smoothness of the nasolabial fold: angle the mouth is lowered, the tension of the lower facial muscles on the opposite side of the face from the focus of the in- soul is weakened. The patient is asked to smile or show his teeth, while there is a weakening of the movements of the lower facial muscles on the side of the paralyzed extremities. A patient with depression of consciousness may have a symptom — a "sail" cheek on the side opposite to the focus of the stroke.

7. Smoothness of the nasolabial fold: angle the mouth is lowered, the tension of the lower facial muscles on the opposite side of the face from the focus of the in- soul is weakened. The patient is asked to smile or show his teeth, while there is a weakening of the movements of the lower facial muscles on the side of the paralyzed extremities. A patient with depression of consciousness may have a symptom — the cheek "sails" on the side opposite to the focus of the stroke. II. Vertebral-basilar system of brain blood supply (arteries: vertebral, main/basilar, internal auditory, posterior cerebral).

II. Vertebral-basilar system of brain blood supply (arteries: vertebral, main/basilar, internal auditory, posterior cerebral).). 1. Dizziness: systemic dizziness in the form of a feeling of instability and tension, a feeling of movement of objects in a certain direction, a feeling of sinking of one's own body. Central vestibulopathy with pronounced persistent nystagmus and vestibular ataxia in combination with nausea, vomiting and pallor of the skin is more often noted. It should be remembered that isolated vertigo is a frequent symptom of a number of non-vascular diseases of the inner ear (Meniere's disease, benign

positional paroxysmal vertigo, vascular neuritis). The oculo- vestibular reflex is positive — the movement of the eyeballs or their convergence (look at the tip of your nose) leads to increased dizziness.

2. Cerebellar syndrome: cerebellar attack , violation of maintaining the vertical position of the body and walking with a deviation of the trunk towards the affected hemisphere moz- 9 Lectures by zhechka, discoordination in the limbs homolateral to the stroke side on one side of the body. Cerebellar symptoms that are noted on the side of stroke: statico-locomotor and dynamic ataxia, nystagmus, muscle hypotension in the extremities, muscle asinergia, adiadochokinesis. 3. Visual disturbances: various types of hemianopsia in the fields of vision opposite from the stroke, diplopia (double vision), visual agnosia, photopsia.

3. Visual disturbances: various types of hemianopsia in the fields of vision opposite from the stroke, diplopia (double vision), visual agnosia, photopsia.

4. Motor disorders: weakness — hemi-paresis /hemiplegia in the extremities on the opposite side from stroke (damage to the brain stem). If the main artery is affected , all four limbs may weaken (tetrapause). The face may be involved on the insulin side (weakness of the facial muscles), and weakness in the extremities develops on the opposite side. Alternating stem syndromes — cranial nerve damage on the side of the insula focus and conduction disorders (hemiparesis, hemianesthesia) in opposite extremities: 1) Weber syndrome (damage to the midbrain): on the stroke side — ptosis, mydriasis, divergent strabismus (oculomotor nerve) + on the opposite side — hemiparesis; 2) Miyar — Gubler syndrome (damage to the bridge of the brain): on the stroke side — weakness of the entire fault of the facial muscles (facial nerve) + hemiparesis on the opposite side; 3) Jackson syndrome (damage to the medulla oblongata): on the side of stroke — atrophy of half of the muscles of the tongue with a deviation of its tip in the opposite direction (hyoid nerve) + hemiparesis on the opposite side.

5. Sensitivity disorders: numbness, decreased pain and other types of sensitivity, paresthesia in opposite extremities, which can be combined with facial numbness on the stroke side (alternating hemihypesthesia).

6. Speech disorder: blurred and indistinct speech, poor articulation and pronunciation (dysuria). It can be combined with a violation of the voice (dysphonia) and a violation of swallowing (dysphagia) - bulbar syndrome with damage to the medulla oblongata.

Clinical characteristics characteristic of ischemic stroke: 1) TIA or transient monocular blindness occurred 1-6 months before the stroke; 2) previously identified angina pectoris or symptoms of ischemia of the lower extremities; 3) pathology of the heart with a high risk of cardioembolism (cardiac arrhythmias, most often in the form of atrial fibrillation, the presence of artificial heart valves, rheumatism, infectious endocarditis, acute myocardial infarction, mitral valve prolapse, etc.); 4) often in old age develops during sleep, after taking a hot bath, physical fatigue, as well as during an attack of atrial fibrillation, including against the background of a decrease in blood pressure, acute myocardial infarction, colitis, hypovolemia; 5) gradual development of neurological symptoms (several hours), in some cases its flickering, that is, an increase, decrease and again an increase in the severity of clinical symptoms; 6) age over 50 years; 7) prevalence of neurological focal symptoms over general cerebral symptoms ; depression of consciousness and intense headache are rare.

During an objective examination of a patient with a suspected stroke, it is necessary: 1) to assess the general condition and the state of vital functions: the level of consciousness, the passage of the respiratory tract, respiration and general circulation; 2) carefully examine and palpate the soft tissues of the head (identification of possible traumatic brain injury), examine the external auditory and nasal passages (to detect cerebrospinal fluid and hematorrhea); 3) measure the pulse rate, heart rate, blood pressure on both hands, respiratory rate; 4) perform auscultation of the

heart and lungs, measure body temperature; 5) conduct a clinical neurological examination: qualitative assessment of focal, general cerebral and meningeal symptoms with fixation in medical documentation; 6) perform electrocardiography; 7) conduct a laboratory screening test of blood glucose levels

Treatment at the pre-hospital stage Treatment of patients with strokes at the prehospital stage (for a family doctor - before the arrival of an ambulance) includes two main directions — basic and specific therapy. The main directions of basic therapy: 1. General first aid measures : – lay the patient on a horizontal surface with his head raised by approximately 30° (the pillow is placed from the level of the shoulder girdle, and not just under the head to avoid flexion in the cervical spine); – provide access to fresh air, unbutton clothes that make breathing difficult, free the oral cavity from removable dentures (if any);

in case of depression of consciousness and vomiting, it is necessary to turn the patient's head and torso to the side to prevent tongue entrapment (asphyxia) and aspiration, sanitize the oral cavity from mucus and vomit. 2. Antihypertensive therapy. A gradual decrease in blood pressure in ischemic or undifferentiated stroke is carried out if Blood pressure exceeds 200/110 mm Hg. In case of suspicion of the hemorrhagic nature of ONMC (when meningeal symptoms are detected along with the ONMC clinic during examination), blood pressure should be reduced if it exceeds 170/100 mm Hg. The target level of drug hypotension is blood pressure exceeding the usual for the patient by 15-20 If the usual blood pressure is unknown, it is not recommended to lower blood pressure below 160/85 mm Hg. Any sharp drop in blood pressure should be avoided, and therefore the use of nifedipine (corin- far) and intravenous bolus administration of hypotensive drugs is undesirable. Preference should be given to antihypertensive drugs from groups of ACE inhibitors — captopril (capoten 12.5–25 mg under the tongue with blood pressure control every 15 minutes). 3. Relief of convulsive syndrome. If the debut of the clinical picture of ONMC is accompanied by an epileptic and convulsive seizure (generalized or partial), diazepam (seduxen, relanium) 10 mg 2 ml intravenously jet slowly per 10 ml of 0.9% NaCl solution is used for its relief (or 10 mg 2 ml intramuscularly). It is possible to repeat after 10-20 minutes another 2 ml of 10 mg or intramuscularly 2 ml of 10 mg. The maximum daily outpatient dose of diazepam is 40 mg. Neuroprotective therapy. Early use of some neuroprotectors is possible already at the prehospital stage, before the nature of ONMC is clarified. According to the leading international clinical guidelines for the treatment of stroke and the results of most large randomized studies on the effectiveness of cerebroprotective drugs, there is no neuroprotective program yet.

In accordance with the standard of emergency medical care for undifferentiated stroke, approved by Order of the Ministry of Health of the Russian Federation No. 1282n in 2012, a general practitioner at the prehospital stage (before the ambulance arrives) of providing care to a patient with a stroke can use a combination of two neuroprotective preparations (evidence level C): magnesium sulfate (magnesia), aminoacetic acid (glycine). Magnesium sulfate is administered intravenously drip slowly (10 ml of 25% solution in dilution per 100 ml of 0.9% NaCl solution) for 30 minutes. Glycine (for patients who are conscious) is used sublingually or transbuccally once 1 g (or 10 tablets of 100 mg each) in tablets or in powder form after grinding the tablet [6].

Drugs contraindicated at the prehospital stage of treatment of undifferentiated stroke: calcium chloride, ethamzylate, aminocaproic acid, nifedipine (not recommended to use), acetylsalicylic acid and other antiplatelet agents, warfarin and other anticoagulants, furosemide or dexamethasone (sometimes mistakenly prescribed to reduce brain edema), vasoactive agents (pentoxifylline, eufillin, vinpocetine, nicergoline, etc.), nootropic activating agents brain drugs (piracetam, nootropil, gliatilin, etc.) [4].

Drug therapy in the recovery period:

The basis of rehabilitation therapy for stroke patients is: multidisciplinary

rehabilitation, prevention and control of complications, drug prevention of recurrent stroke, proper care and restoration of self-service skills. Various groups of drugs (with a low level of evidence) that improve cerebral circulation and neurotrophic brain functions, symptomatic therapy, and treatment of post-stroke conditions can be used as a supplement. It should be noted that from the point of view of evidence-based medicine, there is currently no convincing confirmation of the high effectiveness of cerebroprotective, vasoactive and nootropic drugs in the treatment of stroke patients [4]. At the same time, in most domestic publications and in the standards of the Ministry of Health , these drugs are recommended to improve the effectiveness of rehabilitation treatment of stroke [7, 8]

1. Neuroprotective (cerebroprotective) agents (especially indicated after extensive strokes, speech disorders and cognitive disorders, reduced mental and motor activity, level of evidence C): 1.1. Neurotrophic neuropeptides: – a complex of peptides from the pig brain (cerebrolysin) intravenously drip 5-10 ml per 200 ml of 0.9% NaCl solution, a course of 10 days, 2-3 courses during the first year; – deproteinized hemoderivate of calves' blood (actovegin) intravenously drip 5-10 ml for 200 ml of 0.9% NaCl solution, a course of 10 days, 2-3 courses during the first year; – a complex of peptides from the brain of calves (cortexin) intramuscularly 10 mg (1 bottle), a course of 10 days, 2-3 courses during the first year. 1.2. Antioxidants: – ethylmethylhydroxypyridine succinate (mexidol). Intravenously drip 4-5 ml (50 mg in 1 ml) per 200 ml of 0.9% NaCl solution or intramuscularly 2-5 ml, a course of 10 days, then 1 tab. 125 mg 3 times a day for 1 month;

– succinic acid + riboxin + nicotine- mfa + riboflavin (cytoflavin). Intravenously drip 5-10 ml per 200 ml of 0.9 % solution NaCl, the course is 10 days, then 2 tab. 2 times a day 1 month; – thioctic acid (thiogamma). Intravenous drip 600 mg per 200 ml of 0.9 % solution NaCl, the course is 10 days, then 1 tab. 600 mg 1 time a day for 1 month. 2. Nootropics (in cognitive disorders , evidence level C): – choline alfoscerate (gliatilin). Intravenous drip of 1000 mg (4 ml) per 200 ml of 0.9% NaCl solution, a course of 10 days, then 1 capsule. 400 mg × 3 times a day for 1 month; – citicoline (ceraxone). Intravenously, 1000 mg (4 ml) per 200 ml of 0.9% solution NaCl, a course of 10 days, then 5-10 ml inside 2 times a day for 1 month. 3. Vasoactive agents (after a minor ischemic stroke or TIA, evidence level D): – vinpocetine (cavinton). Intravenously drip 20-50 mg (5 mg in 1 ml), 4-10 ml per 200 ml of 0.9% NaCl solution, a course of 10 days, then 1 tab. 5-10 mg 3 times a day for 1 month; – pentoxifylline (trental) intravenously drip 100-200 mg (20 mg in 1 ml), 5-10 ml for 200 ml of 0.9% NaCl solution, the course is 10 days, then 1 tab. 100 mg 3 times a day for 1 month. Vasoactive agents are contraindicated in the following cases: arterial hypotension, unstable blood pressure for 6 months after myocardial infarction, cardiac arrhythmia, heart failure with the risk of decompensation. 4. Other means affecting the central nervous system (level of evidence C): - ginkgo biloba extract standardized (EGB 761, tanakan, memoplant). According to 1 tab. 40 mg 3 times a day or 80 mg 2 times a day, the course is 1 month; – vitamins of group B (thiamine + pyridoxine + cyanocobalamin, milgamma) intravenously 2 ml 1 time a day, a course of 10 days. Neuromultivitis 1 tab. 3 times a day, course 1 month;

References:

1. Неврология: национальное руководство / Под ред. Е.И. Гусева, А.Н. Коновалова, А.Б. Гехт. — М.: ГЭОТАР-Медиа, 2014.— 688 с. [Nevrologiya: natsional’noe rukovodstvo. Ed by E.I. Guseva, A.N. Konovalova, A.B. Gekht. Moscow: GEOTAR-Media; 2014. 688 p. (In Russ).]
2. Скоромец А.А., Щербук Ю.А., Алиев К.Т., и др. Догоспитальная помощь больным с мозговыми инсультами в Санкт-Петербурге. Материалы Всероссийской науч.-практ. конференции «Сосудистые заболевания нервной системы».— СПб., 2011.— С. 5–18. [Skoromets AA, Shcherbuk YA, Aliev KT, et al.

Dogospital'naya pomoshch' bol'nym s mozgovymi insul'tami v Sankt-Peterburge. Materialy Vserossiyskoy nauch.-prakt. konferentsii "Sosudistye zabolevaniya nervnoy sistemy". (conference proceedings). — Saint Petersburg; 2011:5-18. (In Russ).]
3. Кандыба Д.В. Профилактика инсульта: учебное пособие. — СПб.: Издательство СЗГМУ
4. им. И.И. Мечникова, 2013. — 84 с. [Kandyba DV. Profilaktika insul'ta. Saint Petersburg: Izdatel'stvo
5. SZGMU im. I.I. Mechnikova; 2013. 84 p. (In Russ).] 4. Кандыба Д.В. Ошибки в амбулаторной неврологической практике. Часть II // Российский семейный врач. — 2015. — Т. 19. — № 1. — С. 13–20. [Kandyba DV. Mistakes in outpatient neurological practice.Part II. *Russian family doctor.* 2015;19(1):13-20. (In Russ).] doi: 10.17816/RFD2015113-20.
6. Guidelines for Management of Ischaemic Stroke and Transient Ischaemic Attack. The European
7. Stroke Organization (ESO), 2008. 120 p. http://www.eso-stroke.org.
8. Стандарт скорой медицинской помощи при инсульте. Утвержден приказом Министерства здравоохранения Российской Федерации от 20.12.2012 № 1282н. 4 с. http://www.rosminzdrav.ru/documents. [Standart skoroy meditsinskoy pomoshchi pri insul'te. Utverzhden prikazom Ministerstva zdravookhraneniya Rossiyskoy Federatsii ot 20.12.2012 No 1282n. 4 p. (In Russ).]
9. Стандарт специализированной медицинской помощи при инфаркте мозга. Утвержден приказом
10. Министерства здравоохранения Российской Федерации от 29.12.2012 № 1740н. — 14 с. http://www.
11. rosminzdrav.ru/documents. [Standart spetsializirovannoy meditsinskoy pomoshchi pri infarkte mozga.
12. Utverzhden prikazom Ministerstva zdravookhraneniya Rossiyskoy Federatsii ot 29.12.2012 No1740n. — 14 p. (In Russ).]
13. Стандарт специализированной медицинской помощи при внутримозговом кровоизлиянии (консервативное лечение). Утвержден приказом Министерства здравоохранения Российской Федерации от 29.12.2012 № 1692н. 18 с. http://www.rosminzdrav.ru/documents. [Standart spetsializirovannoy meditsinskoy pomoshchi pri vnutrimozgovom krovoizliyanii (konservativnoe lechenie). Utverzhden prikazom Ministerstva
14. zdravookhraneniya Rossiyskoy Federatsii ot 29.12.2012 No 1692n. 18 p. (In Russ).]
15. Goldstein LB, Bushnell ChD, Adams RJ, et al. Guidelines for the Primary Prevention of Stroke: A Guideline for Healthcare Professionals From the American Heart Association. American Stroke Association. *Stroke*. 2011;42(2):517-584. doi: 10.1161/str.0b013e3181fcb238.