EDITORIAL VIEW

Postoperative cognitive dysfunction: the issues of diagnostics and possible prevention

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ABSTRACT

Its' long been known that the state of anesthesia has something to do with the cognitive functions of the brain, but it was thought that all impairment was purely reversible with no residual effects. Only recently it was theorized by some scientists that general as well as spinal anesthesia may leave behind some scars; the more complex the underlying disease process, and the more complicated the surgical process, the more likely that the patient will continue to exhibit impairment of some of his intellectual abilities. The precise attention to this problem is primarily associated with peri-operative anesthetic complications, their preventability, the banality of their causes and damaging power and the size of anesthesiological mistakes. Yasuhiro Morimoto in his article for the special issue of Apicare Journal entitled 'Post-operative delirium and cognition defects after cardiac surgeries; incidence, management and preventive strategies' has highlighted this problem after cardiac surgeries, and this editorial compliments it and discusses some more aspects linked to postoperative cognitive dysfunction from a neurologists point of view.

Key words: Cognition; Cognitive functions; Cognitive Defects; Cognitive Deficits; Cognitive impairments; Postoperative cognitive dysfunction; Delirium; Postoperative period

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One of the strategic points for the anesthesiologist is the brain, and one of the most important functions of the brain are cognitive functions. Cognitive functions are commonly understood as the most complex functions of the brain, by means of which the process of rational cognition of the world is happening.[1] Cognitive functions include memory, gnosis, speech, praxis and intelligence. Under intelligence we understand the ability to collate information, to find similarities and differences in it and make judgments and conclusions. Intellectual abilities are provided by integrated brain activity as a whole.[1,2]

Because cognitive function is associated with integrated brain activity as a whole, cognitive impairment develops naturally in a variety of focal and diffuse brain lesions. Chronic degenerative processes leading to the slow development of cognitive deficits, such as Alzheimer's disease, Parkinson's disease various types of dementia, are: in metabolic and mitochondrial diseases (MERRF, MELAS, lipidoses, dementia with Lewy bodies, etc) in its basics have slow mechanisms of lipid peroxidation, the outtake of free radicals, lactic acid and neuronal apoptosis.[3-5]

Brain damage after surgery and general anesthesia (GA) manifests itself as a separate neurological and neuropsychic problem. Therefore, precise attention to this problem is primarily associated with a high rate of anesthetic complications,[6-10] the controversy of the question of their preventability, the banality of their causes and damaging power and an increased number of lawsuits and the size of anesthesiological mistakes.[11-13]

A stay at the intensive care unit (ICU) is often stressful, even for a healthy person. For an operated patient under residual effects of anesthesia and potent pain killers staying at ICU usually turns out to be an unpleasant experience. An unfamiliar surroundings, continuous

monotonous noise from equipment operating,

permanent lighting and a disturbed sleep due to constant staff duties often lead to cognitive impairment. The presence of drains, catheters, monitoring of vital signs and continuous infusion therapy, etc. cause discomfort and exacerbates mental disorders.[14] Thus, postoperative period after GA may manifest itself by postoperative cognitive dysfunction (POCD), hypoxic encephalopathy, as well as a rare complication of the Posterior Reversible Encephalopathy Syndrome (PRES).[4] Acute cognitive impairment after GA can present with diffuse symptoms, e.g. delirium, convulsions, circadian cycle disorders, coordination disorders, choreoathetosis, etc. and focal symptoms including stroke, acute sensorineural hearing loss, spastic paraplegia, etc. Chiu-Ming Ho et al described the case of acute cognitive changes in a 33 year old primipara, who underwent cesarean delivery under spinal anesthesia and developed a posterior reversible encephalopathy syndrome (PRES) with vasospasm. The patient presented with severe headache over the occipital area, decreased awareness, confusion, visual disturbances and altered sensorium five days after an episode of typical postdural puncture headache (PDPH) after spinal anesthesia. MRI findings indicated that the lesion of PRES in this patient was associated with ischemia-induced cytotoxic edema.[4]

In the special issue of Apicare Journal the publication of Yasuhiro Morimoto is dedicated to one of the most frequent complications of GA, e.g. POCD. It is defined as a cognitive disorder that develops in the early, and continues in the late postoperative period. It clinically manifests itself with memory impairment, difficulty of concentration, attention and other cognitive impairments (thinking, speech, etc.) confirmed by neuropsychological data testing. Testing performance postoperatively declines not less than 10% compared to the preoperative level.[1,2] The etiology and pathogenesis of POCD can be determined by three groups of factors: 1) the influence of the residual components and GA, especially anesthetics and their biotransformation products, which are active against central nervous system, 2) the activation during surgery of antinociceptive protection of brain structures, insolvency of which leads to overexcitation and exhaustion of the energy balance of the neurons of the cerebral cortex and subcortical structures, and 3) the damaging effect of diffuse hypoxia (hypoxemia, severe anemia) and local hypoxia (failure of the cerebral blood flow, its redistribution) as a result of cerebral edema and increased intracranial pressure.[9]

Diagnostics of POCD currently varies depending on the country and the proposed criteria. There are three problems of POCD identifying and determining the degree of cognitive impairment after GA. The first problem correlates with the lack of neuropsychological tests in the preoperative period and is often explained due to the absence of such recommendations in clinical protocols or alleged lack of need. In this case POCD will be hardly differentiated from the worsening of cognitive deficit, already existing in the patient prior to GA. The second problem is the inconsistency of neuropsychological tests use, some of which are even used incorrectly in the diagnosis of POCD. Hanning in his article about POCD[15] emphasizes the imperfections of some commonly used tests to detect cognitive dysfunction, e.g. the test MMSE, which was designed as a screening tool in the clinical examination of patients with dementia. Most normal adult subjects of any age score, at or very close to the maximum, with ease. Thus, after appearance of a minimal cognitive deficit after GA these impairments will not be detected with the use of MMSE. A more informative and sensitive test for the detection of cognitive disorders in a patient "tied to bed" is the Frontal Assessment Battery (FAB). The FAB is a brief battery of six neuropsychological tasks that was specifically designed to assess frontal lobe function at bedside.[16] The FAB consists of the following six tasks: similarities (conceptualization), lexical verbal fluency (mental flexibility), motor series (programming), conflicting instructions (sensitivity to interference), go – no go (inhibitory control) and prehension behavior (environmental autonomy). This test is easy to administer, requires less than ten minutes to complete and is well accepted by patients. As a practicing neurologist I strongly recommend to administer the FAB as a neuropsychological screening for all patients who will undergo general anesthesia and operations on heart. The third problem is the fact that many studies about POCD aimed to identify the cognitive deficits in patients immediately after surgery, or within 1-2 days after the event. In this period of time, a patient is usually examined by a neurologist or a neuropsychologist in the ICU. However, there was a group of patients who initially did not show POCD symptoms and was dropped out of the surveillance; hence, cognitive pathology that appeared later was not diagnosed at the stage of post-surgical recovery (at ICU). Cognitive disorders,, which are delayed after the GA but still are connected to it, could be further classified as age-related cognitive disorders in this group of patients, or could be related to

other causes, e.g. trauma, alcohol intoxication, degenerative diseases.

The most common cardiac surgical procedure performed in the United States and Western Europe is coronary artery bypass grafting (CABG).[3] The greatest incidence of POCD and the greatest number of studies is in this group of patients. The potential for brain injury in these patients, including hypoperfusion and micro-emboli, is self-evident and POCD is thus not surprising.[15] In his paper, Yasuhiro Morimoto points out that the highest incidence of POCD occurs in patients undergoing cardiac surgery. He underlines that the incidence of POCD after cardiac surgery in general is higher than that after other non-cardiac surgeries. McKhann et al. in their paper gave proof that the incidence of encephalopathy and stroke connected with CABG appears to be increasing, and this increased incidence is because more 'high risk' patients currently undergo surgery at our institutions than 10 years ago.[5] The presence of stroke in the postoperative period may be signaled by the inability of the patient to emerge from anesthesia (follow commands, move all extremities) in the first 6 hours after the operation. This is a valuable symptom which should attract sighting attention of ICU specialists and is a reason for an urgent neurological examination. Patients who are unable to emerge from anesthesia, who develop post-GA delirium, hallucinations, seizures, psychomotor excitation and focal disturbances (paralysis, sensory deficit, hyperkinesis) should undergo brain MRI, specifically diffusion-weighted imaging (DWI), which is the most sensitive and accurate neuroimaging technique in this patient population. The mechanisms leading to postoperative strokes, encephalopathy and COPD include preexisting cerebrovascular diseases, microemboli, hypoperfusion and atrial fibrillation. McKhann et al. in their paper distinguished neuroprotective strategies to be used in cardiac surgery to prevent cognitive and focal neurological complications. They offer to examine patient before surgery on the presence of carotid artery stenosis, preexisting cerebrovascular disease and ischemic lesions identified on MRI (DWI), as well as searching for an atrial fibrillation. During surgery doctors should pay attention on possible development of aortic atheroma, systemic hypoperfusion, brain hyperthermia, hyperglycemia and microemboli. And to prevent POCD after surgery doctors should treat atrial fibrillation, to reveal timely ischemic brain lesions and brain perfusion mismatch.[5] I would like to draw the attention of cardiac surgeons and anesthesiologists towards condition of brain vasculature before the operation and detection of previous underlying cerebrovascular disease. The algorithm may be the following: using neuropsychological tests, such as the FAB, and then in revealing the cognitive impairment to make the patient undergo the brain MRI to see collaterals and the probability to compensate possible hypoxia. Neurologists and neuropsychologists should join colleagues in cardiology and cardiac surgery to identify those at risk for adverse neurological outcomes.

Thus, due to multifactorial genesis of POCD and neurological focal disorders as complications of GA in recent years, there appeared a trend towards a multidisciplinary approach to the problem with the involvement of specialists of various disciplines, including not only anesthesiologists and neurologists, but also clinical neurophysiologists, pathophysiologists and clinical psychologists. Reducing the damaging effect of GA on the central nervous system is a precondition for its high quality and has a special significance for the young working age patients. The only way to prevent CNS disturbances after surgical interventions in the conditions of long-term general anesthesia is neuroprotection by possible prevention, timely diagnosis and pathogenetically substantiated therapy.

Early tracheal extubation, or 'fast track anaesthesia', in operations on the 'open' heart is now spreading more in adult and in pediatric cardiac anesthesiology.[16-18] This happens due to several reasons: optimal physiology of the systemic and pulmonary circulation in the conditions of spontaneous breathing, a shorter duration of stay on mechanical ventilation and at ICU (and following, respectively lower cost of treatment), better cooperation of the patient with the medical staff and the possibility of an adequate assessment of the severity of pain in the early postoperative period in a patient who is unconscious.[16,19]

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