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Multimodal neuromonitoring

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The goal of the intensive care management of a neurosurgical patient is to preserve adequate cerebral perfusion, oxygenation and metabolism in order to prevent secondary neurological injury. In preventing secondary neurological insults interventions must be started early. For many years conventional methods of neuromonitoring have proved their efficacy. However, their sensitivity in detecting subtle metabolic derangements in the real time manner is low and the valuable time for the appropriate treatment is lost. In recent years, there are numerous study data suggesting that implying of advanced neuromonitoring techniques can improve outcomes. Moreover, it helps to guide goal-directed therapy. Although data on advanced neuromonitoring are preliminary and mostly observational, the number of modern neurosurgical centers applying it as a standard is high. With the increasing technical possibilities, the concept of multimodal neuromonitoring is of increasing popularity. Multimodal neuromonitoring allows continuous real time assessment of cerebral perfusion, oxygenation, metabolism and global function which makes it attractive and promising in clinical practice.

Key words: neuromonitoring, multimodal, traumatic brain injury, cerebral metabolism

CONCEPTION OF MULTIMODAL NEUROMONITORING

Traumatic brain injury (TBI), ischemic or hemorrhagic insults, and subarachnoid hemorrhage are leading pathologies in neurosurgical intensive care units (NeuroICU). Cerebral ischemia and hypoxia are the main causes of brain injury. Majority of patients admitted to the NeuroICU are unconscious, therefore clinical neurological evaluation is problematic. Neurological imaging methods are informative, but their sensitivity in detecting subtle derangements in the real time manner is low and the valuable time for the appropriate treatment is lost. Even normal values of conventional parameters reflecting systemic hemodynamics and oxygenation may not guarantee adequate cerebral perfusion and

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metabolism, therefore advanced neuromonitoring is necessary. Aiming for continuous real time assessment of cerebral perfusion, oxygenation and metabolism, the concept of multimodal neuromonitoring (MNM) was developed.

MNM methods include:

1) Clinical neurological evaluation;

2) Cerebral flow directed techniques (intracranial pressure (ICP)), cerebral perfusion pressure (CPP), laser Doppler flowmetry (LDF), thermal diffusion (TD) monitoring);

3) Cerebral oxygenation directed techniques (jugular venous bulb saturation ($SjvO_2$), brain tissue oxygen tension ($PbrO_2$), regional cerebral oximetry ($SprO_2$));

4) Reflecting cerebral metabolism (microdialysis);

5) Reflecting cerebral global function (electroencephalography (EEG));

6) Cerebral damage markers (S100B, NSE).

METHODS

Clinical neurological evaluation

In spite of broad technical diagnostic possibilities, clinical evaluation remains actual because of its availability, relative simplicity and quick dynamic assessment. Eye ball movement, pupil reaction to light, and motor responses to noxious stimuli, give a quick insight into the level of neurological damage and structural involvement. Continuous assessment of the mentioned clinical signs enables to detect progression of the primary insult. As the Glasgow Coma Scale (GCS) is routinely used in ICU setting and incorporates eye assessment and motor as well as verbal response, it is considered as "golden standard" for neurological dynamic evaluation. Periodic follow-up (e. g. every hour) is reasonable.

Cerebral flow-directed techniques

Cerebral autoregulation maintains cerebral blood flow at a constant level in spite of systemic hemodynamic changes. In neurological catastrophe autoregulation is dysfunctional and cerebral blood flow becomes increasingly dependent on systemic blood pressure (BP) and ICP, therefore continuous flow-directed monitoring and goal-directed therapy may help to preserve the adequate cerebral perfusion.

ICP monitoring. ICP ≤ 15 mmHg is considered as normal. Pathological intracranial hypertension is present at pressures ≥ 20 mmHg. In TBI, the indications for ICP monitoring are the following: unconscious patient (GCS ≤ 8) and radiological evidence of intracranial hematoma, contusions or cerebral edema (1). If CT imaging is normal, two additional criteria are needed: patient age >40 years, systolic blood pressure <90 mmHg and / or presence of abnormal motor posture (decorticate, decerebrate). Depending on the probe placement, there are four ICP monitoring techniques: intraventricular, intraparenchymal, subarachnoid, and epidural. Intraventricular technique is considered to be a "golden standard" because of its high accuracy and possibility to drain the ventricular system for therapeutic and diagnostic purposes. However, the method is associated with a relatively high infection (20%) and hemorrhage (2%) risk. Moreover, procedural difficulties should be considered when ventricles are compressed. In TBI, most guidelines recommend to initiate the intracranial hypertension treatment when ICP exceeds 20 mmHg.

Cerebral perfusion pressure (CPP). ICP monitoring allows for CPP assessment, which is a surrogate measure of cerebral blood flow (CBF). CPP is expressed as a difference between the mean arterial pressure (MAP) and ICP. Normally, autoregulation maintains CBF at a constant level within a wide range of CPP (50–100 mmHg) (2, 3). In neurological disaster autoregulation is dysfunctional and CBF becomes increasingly dependent on CPP changes. When CPP is lower than 50 mmHg, autoregulation cannot maintain the adequate CBF and a focal or global cerebral ischemia may occur. When CPP exceeds 120 mmHg, autoregulation becomes inadequate leading to excessive CBF and cerebral hyperemia, edema and hypertensive encephalopathy (4). Low CPP is associated with poor neurological outcome in TBI. Aggressive hemodynamic augmentation targeting for CPP >70 mmHg may improve survival and neurological outcomes (5). However, recent studies question such a strategy because of high incidence of extracerebral complications (5). In TBI, the 2007 American Association of Neurosurgeons Guidelines recommend the CPP target of 60 mmHg (values lower than 50 and higher than 70 should be avoided) (6).

Thermal diffusion (TD). TD is an invasive quantitative CBF assessment technique based on

tissue ability to dissipate heat. Parenchymal CBF is monitored continuously in the real time manner and is expressed in ml/100 g/min. TD CBF monitoring may be beneficial in patients with TBI (7), SAH (8) and during neurosurgical procedures. CBF ≤ 15 ml/100 g/min is predictive of a secondary vasospasm following SAH (8). TD may also guide a triple H therapy in SAH (9). Limitations of the technique include the invasive and focal (reflecting perfusion in the sensor area only) nature.

Laser Doppler flowmetry (LDF). LDF is an invasive qualitative CBF assessment technique based on the Doppler's principle. LDF enables continuous real time monitoring of CBF at the bedside. Dynamic changes are assessed. LDF reflects autoregulation state and is predictive of its derangement or recovery (10). Disadvantages of the technique include invasive, focal and qualitative nature.

Cerebral oxygenation-directed techniques

As brain tissue has restricted oxygen accumulation ability, cerebral oxygenation status gives an insight into the balance between delivery and consumption.

Jugular bulb oxygen saturation ($SjvO_2$). Venous blood oxygen saturation reflects the oxygen extraction intensity by the corresponding tissue. Thus, $SjvO_2$ reflects cerebral flow / metabolism relationship. The technique enables to monitor ipsilateral hemispheric oxygenation in the real time fashion. Normally $SjvO_2$ is 55–69%. Low $SjvO_2$ is indicative for high cerebral metabolic rate or cerebral hypoperfusion. In TBI, low $SjvO_2$ is reported in as many as 39% cases and is associated with a poor outcome (11). The technique has a potential in neurosurgical setting, as venous desaturation episodes are reported in up to 50% of cases and are associated with a poor postoperative cognitive function (12). However, sensitivity in detection of focal oxygenation disturbances is low, as normal values may be seen even if a focal ischemia exists (13).

Regional cerebral oximetry ($SprO_2$). $SprO_2$ is based on near-infrared spectrum (wave length 700–1 000 nm) ability to penetrate human body tissues. Depending on the oxygenation status, light absorption properties of hemoglobin change and differences in the reflected signal are registered.

Normally, cerebral oxyhemoglobin values variate from 60% to 80%. The technique is widely applied in major vascular surgery to guide cross-clamping and circulatory arrest. In NeuroICU $SprO_2$ monitoring is attractive for its low invasiveness, easy interpretation and continuous real time cerebral oxygenation assessment (14). As with $SjvO_2$, the weak point is poor sensitivity in detection of focal ischemia.

Brain tissue oxygen tension ($PbrO_2$). Advanced poliarographic or optical luminescence technique enables direct measurement of cerebral tissue oxygen tension. Cerebral oxygen content reflects cerebral perfusion and a local oxygen extraction fraction (15). Normal $PbrO_2$ value is approximately 40 mmHg. $PbrO_2$ value of 22 mmHg is generally accepted as the critical ischemic threshold of cerebral perfusion (18 ml/100 g/min) (16). In TBI, low $PbrO_2$ is indicative for cerebral perfusion derangement (17). $PbrO_2$ >36 mmHg is associated with a good outcome, $PbrO_2$ 26–35 mmHg with a significant neurological deficit, and $PbrO_2$ < 25 mmHg with a poor outcome (18). There are study data suggesting that $PbrO_2$ is more accurate in detecting compromised perfusion: CPP > 70 mmHg may not always ensure the adequate oxygenation of the affected cerebral region, as reflected by low $PbrO_2$ (19). $PbrO_2$ is a powerful neurological outcome predictor (20). Compared to $SjvO_2$, $PbrO_2$ is more accurate in detecting cerebral ischemia during cerebral vascular surgery (21). There are reports that in TBI and SAH $PbrO_2$ directed therapy demonstrates better clinical outcome and decreased mortality as compared to ICP / CCP protocol (22). However, the technique is invasive and associated with a small hemorrhage and infection risk. Moreover, sensitivity is highly dependent upon surgeon skills as a probe must be placed precisely in the relevant area.

Methods reflecting cerebral metabolism

Intracerebral microdialysis. This method allows for biochemical assessment of the cerebral extracellular space. A small capillary-like dialysis probe filled with artificial cerebral fluid is placed in the brain tissue. Biochemical compounds move across the semipermeable membrane according to the concentration gradient until the dynamic equilibrium is achieved. The brain tissue is highly dependent on constant oxygen supply for aerobic glicolysis (producing pyruvate) with a high ATP output. In hypoxemia anaerobic metabolism occurs (produc-

ing lactate) leading to insufficient energetic supply. Therefore the lactate / pyruvate ratio (LPR) is a reliable marker of cerebral ischemia, hypoxia and mitochondrial dysfunction (23, 24). As a result, cerebral microdialysis can be useful in early diagnosis of metabolic derangements of the injured brain (23). LPR exceeding 35–40 is associated with a poor neurological outcome. LPR is an independent mortality predictor in TBI (25). Detected metabolic derangements are helpful in the diagnosis of neurological deterioration, progression of intracranial hypertension, vasospasm following SAH, intraoperative ischemia during cerebral aneurysm surgery (26, 27). There are preliminary data suggesting a possibility to monitor cerebral drug concentrations (28). Although promising, the technique is invasive, allows only focal monitoring and results are delayed due to the duration (approximately 1 hour) of dialysis.

Methods reflecting cerebral global function

Continuous EEG. EEG is a noninvasive technique, which allows continuous real time monitoring of cerebral bioelectrical activity. Data acquired from EEG may be presented as raw EEG which requires an involvement of well-trained specialist, and quantitative EEG (qEEG) which has simplified interpretation but is often influenced by artifacts. Invasive cortical EEG may also be used. EEG as a part of MNM is helpful in detection of seizure activity, cerebral ischemia, vasospasm following SAH and has a potential to guide neurosurgery.

Nonconvulsive seizures are a common problem in NeuroICU (8–48% incidence in comatose patients) (29). Undiagnosed seizures waste energetic substrates of primarily injured brain and are associated with an increased risk of mortality and neuronal injury (30), therefore an early diagnosis and appropriate treatment are necessary. Continuous EEG has higher sensitivity in the diagnosis of seizure activity in comatose patients as compared with the standard EEG which makes it a possible source for better outcome (29).

As pyramidal neurons are highly sensitive to hypoxia, even minor changes of the cerebral blood flow are reflected on EEG in several seconds. With CBF of 25–35 ml/100 g/min, rapid EEG waves begin to disappear; when CBF decreases to 17–18 ml/100 g/min, a progressive slow wave activity reflecting the ischemic threshold is observed, but changes are reversible. CBF less than 10–12 ml/100 g/min leads to neuronal death and EEG waves are no longer seen. EEG may also be applied in the diagnosis of vasospasm following SAH. Even before apparent vasospasm, characteristic changes in raw EEG are often observed. Quantitative EEG is increasingly used in early diagnosis of ischemia due to vasospasm following SAH (31). However, data on the use of qEEG are preliminary.

Intraoperative EEG is of certain potential (e. g. diagnosis of evolving ischemia in carotid artery endartercetomy), however, it is significantly influenced by general anesthesia complicating its interpretation. On the other hand, somatosensory evoked potentials (SEP) (visual, auditory, sensory) altering baseline EEG may be involved in central nervous system integrity assessment as they are relatively resistant to the impact of anesthetic drugs (32).

Cerebral damage markers

MNM requires huge personal, technical and financial resources. Early clinical and image evaluation lacks sensitivity in predicting neurological outcomes, so the idea of a simple, quick and reliable prognostic tool seems reasonable (33). There are two cerebral damage markers available at the moment – S100B and NSE. S100B is a cerebral protein found in peripheral blood. Concentration above 0.5 μg/L is considered as abnormal and reflects astrocite death (34). This marker could be used as an early and reliable poor neurological outcome predictor (33). The American Academy of Neurology Guidelines state that S100B reflects the severity of cerebral ischemic / hypoxic injury following cardiac arrest (35). As protein has short half-life in peripheral blood (97 min), repeated samples can detect ongoing cerebral damage. NSE is a specific cerebral enzyme also found in peripheral blood. The half-life is significantly higher as compared to S100B (48 hours), making continuous monitoring unreasonable. On the other hand, the subsequent peak may reflect a secondary cerebral damage (34). The American Academy of Neurology claims that NSE >33 μg/L is associated with a poor neurological outcome in coma patients (35). There is a strong correlation between the NSE concentration, CT findings and GCS after three months following injury (36).

CONCLUSIONS

The success of neurologically directed treatment is highly dependent on prevention and management of secondary neuronal damage. MNM as a complex approach gives an insight into cerebral blood flow, oxygenation and metabolic status in continuous real time manner; therefore it is perspective in early detection and directed management of secondary neurological insults. Although data on MNM methods is preliminary and sometimes controversial, most studies suggest their beneficial impact on neurological outcome. Modern neuromonitoring techniques are of increasing popularity among leading neurosurgical centers and MNM seems the only way to follow.

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MULTIMODALINIS NERVŲ SISTEMOS MONITORAVIMAS

Santrauka

Neurochirurgijos intensyviosios terapijos tikslas yra užtikrinti adekvačią galvos smegenų perfuziją, oksigenaciją ir metabolizmą, kad būtų išvengta antrinio neurologinio pažeidimo. Antrinio pažeidimo prevencijai būtinas ankstyvas ir tikslinis gydymas. Jau įrodytas standartinių nervų sistemos monitoravimo metodų efektyvumas, tačiau šie metodai nesuteikia galimybės nepertraukiamai vertinti smegenų būklę, todėl tikslinė terapija dažnai yra pavėluota. Pastaruoju metu literatūroje atsiranda vis daugiau duomenų apie multimodalinio nervų sistemos monitoravimo metodikas bei teigiamą jų poveikį pacientams. Be to, tokia monitoravimo taktika suteikia galimybę taikyti tikslinę terapiją. Nors įrodymai apie multimodalinio nervų sistemos monitoravimo naudą yra preliminarūs ir daugiausia pagrįsti stebimosiomis studijomis, daug modernių pasaulio neurochirurgijos centrų šiuos metodus jau įtraukė į standartinį monitoravimą. Gerėjant techninėms galimybėms multimodalinio nervų sistemos monitoravimo koncepcija tampa vis populiaresnė. Šis monitoravimas leidžia nuolat stebėti smegenų perfuziją, oksigenaciją bei bendrą funkciją, todėl yra labai patrauklus ir perspektyvus klinikinėje praktikoje.

Raktažodžiai: nervų sistemos monitoravimas, multimodalinis, trauminis smegenų pažeidimas, smegenų metabolizmas