Lund Concept

Lund concept are based on physiological mechanisms for regulation of brain volume and brain perfusion and to reduce transcapillary plasma leakage and the need for plasma volume expanders, involving a “volume-targeted” strategy for intracranial pressure control.

It is based on the premise that the blood brain barrier is disrupted after traumatic brain injury and cerebral autoregulation is impaired; hence, the transcapillary water exchange is determined by the differences in hydrostatic and colloid osmotic pressure between the intracapillary and extracapillary compartments. The Lund concept argues that the only way of inducing transcapillary reabsorption of interstitial fluid is to control the transcapillary osmotic and hydrostatic differences and utilizes a complex combination pharmacotherapy involving β1-antagonist metoprolol, α2-agonist clonidine, low-dose thiopental, dihydroergotamine, and maintenance of colloid osmotic pressure by red blood cell transfusion and albumin administration [1].

Originated in the University of Lund, Sweden, more than 20 years ago the concept has remained controversial ever since.

The Lund concept, directed at bedside real-time monitoring of brain biochemistry by CM showed better results compared to the Rosner concept CPP-targeted therapy in the treatment of comatose patients sustaining SBI after aneurysmal SAH and severe TBI [2] [3].

There have been nine non-randomized and two randomized outcome studies with the Lund concept or modified versions of the concept. The non-randomized studies indicated that the Lund concept is beneficial for outcome. The two randomized studies were small but showed better outcome in the groups of patients treated according to the modified principles of the Lund concept than in the groups given a more conventional treatment [4].

Goals

The therapy has two main goals:

(1) to reduce or prevent an increase in ICP (ICP-targeted goal)

(2) to improve perfusion and oxygenation around contusions (perfusion-targeted goal). The Lund therapy considers the consequences of a disrupted blood-brain barrier for development of brain oedema and the specific consequences of a rigid dura/cranium for general cerebral haemodynamics. It calls attention to the importance of improving perfusion and oxygenation of the injured areas of the brain. This is achieved by normal blood oxygenation, by maintaining normovolaemia with normal haematocrit and plasma protein concentrations, and by antagonizing vasoconstriction through reduction of catecholamine concentration in plasma and sympathetic discharge (minimizing stress and by refraining from vasoconstrictors and active cooling). The therapeutic measures mean normalization of all essential haemodynamic parameters (blood pressure, plasma oncotic pressure, plasma and erythrocyte volumes, PaO(2), PaCO(2)) the use of enteral nutrition, and avoidance of over nutrition [5].

Management

Prevention of brain edema formation to reduce fluid shift from capillaries into brain parenchyma, by preserving capillary colloid osmotic pressure and reducing capillary hydrostatic pressure The improvement of the cerebral microcirculation by the avoidance of arterial vasoconstrictors

Preserve osmotic pressure

albumin (considered a contra-indication by some based on SAFE trial subgroup analysis)

blood products

diuretics

Reduce hydrostatic pressure

metoprolol

clonidine

thiopentone

dihydroergotamine (precapillary vasoconstriction)

CPP target

if ICP normal aim for 60-70mmHg however, if ICP elevated a CPP of 50mmHg is accepted

Vasoactive use

avoid dobutamine (cerebral vasodilatation)

avoid noradrenaline (cerebral vasoconstriction)

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