Subarachnoid hemorrhage caused by intracranial pressure

Intracranial aneurysms are acquired or congenital vascular malformation featuring as cerebral arterial enlargement. It’s often described as a “ballooning” of the blood vessel. As arterial lumen enlarges, vessel wall becomes thinner and frail and can eventually break causing blood extravasation into the skull. This is a dramatically neurological event called Subarachnoid Hemorrhage (SAH) from the name – the subarachnoid space – of the particular space enveloping the brain in which the major cerebral vessels lie and blood can collect. After such a neurological acute injury patients can suffer a broad spectrum of neurological impairment, from a thunderclap headache to a deep coma. Doctors called to initially manage a patient with SAH have to face several life-threatening clinical problems: form the need of an urgent aneurismal repair (through a neurosurgical or endovascular approach) to the medical consequences of neurological deficit (in term of respiration and circulation), to the prevention of further neurological damage after bleeding.

When a cerebral vessel breaks blood flows out and collects into the skull. As the skull is a rigid unexpandable case and the volume of blood adds to the volume of brain, pressure inside the skull increases and consequently brain is compressed. This scenario is called “intracranial hypertension” and represents a real neurological emergency that requires an aggressive treatment. A number of medical and surgical options are actually available to treat this condition, from mannitol infusion (a osmotic diuretic that promotes water outlet from brain tissue) till, in refractory cases, the removal of part of the skull. Obviously, the diagnosis, and treatment of high intracranial pressure (ICP), needs a feasible and reliable pressure measurement that can be obtained though a thin probe inserted into the skull.

Unlike the widely accepted in other kind of brain injury (namely traumatic brain injury) and although high ICP is common after aneurismal rupture (80% of patients in the first week as reported from a previous work), only a limited number of studies have assessed the time course of ICP and its effect on outcome in this condition. Furthermore, despite widespread use of ICP monitoring the precise threshold at which a patient requires a treatment and the impact of the duration of intracranial hypertension in unclear.

From a theoretical point of view, a physiological insult to a delicate structure, like the brain, can be described by two dimension: intensity and duration. This is well known by physics: a little force applied to a body for a long time can cause the same consequences of a great and brief push.

Technological improvement at bedside, like the possibility to record and store high definition (i.e. one data for second or minute) physiological variables paved the road to such an approach in quantify the impact that a certain physiological insult has on clinical outcome.

Using the mathematical tool of Pressure-time dose (PTD), that incorporates both duration and intensity of an insult above a determined threshold, we studied the influence of high ICP burden in a population of SAH patient admitted to neuro-intensive care unit during the first seven days after
aneurismal rupture. We collect one data for minute, instead of the hourly based ICP value classically reported on the medical record, and tried to quantify the dose of High-ICP experienced by the patients using four different threshold (15, 20 – the widely accepted threshold for ICP treatment – 25 and 30 mmHg). Clinical outcome was evaluated at hospital discharge and at six months, using standard methods. We observed an association between mortality at hospital discharge and higher level of ICP dose in term of PTD using 20, 25 and 30 mmHg and between exposure to a moderate dose of ICP (using 30 mmHg threshold) and unfavourable long-term outcome.

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**Publication**

[High-Resolution Intracranial Pressure Burden and Outcome in Subarachnoid Hemorrhage](https://doi.org/10.1161/STROKEAHA.115.011502).

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*Stroke*. 2015 Sep