From a pharmacology perspective, neuroanesthesia can be thought of as the practice of applied cerebrovascular physiology using anesthetics, particularly as it relates to the manipulation of cerebral blood volume. The “neuro-pharmacology” profile of an anesthetic has traditionally been characterized in terms of the drug’s effects on cerebral blood flow (CBF), cerebral metabolic rate (CMRO₂), intracranial pressure (ICP) and cerebrospinal fluid production and absorption. The effects of anesthetics on these parameters are important because untoward changes can lead to cerebral ischemia (a physiologic problem) and even cerebral herniation (an anatomic problem).

Cerebral ischemia occurs when cerebral perfusion pressure (CPP) is insufficient to deliver adequate energy substrate and oxygen to neuronal tissue. CPP is determined by the complex interplay of CMRO₂, CBF and ICP (and other parameters such as mean arterial pressure). Decreases in CMRO₂ lead to decreases in CBF which in turn lead to decreases in ICP. Known as “flow-metabolism-coupling,” this phenomenon is the scientific foundation for anesthetic mediated manipulation of cerebral hemodynamics. Intraoperatively, expert control of this phenomenon (along with other interventions) can prevent stroke and brain tissue herniation through the craniotomy site.

In the early days of modern neuroanesthesia practice, conventional wisdom dictated that neuroanesthetists select drugs that had an overall favorable “neuroanesthesia” profile in terms of a drug’s effects on CMRO₂, CBF, ICP, etc.¹ In other words, drugs were selected in large part on the basis of pharmacology studies considered in isolation despite recognition that outcome studies to support superiority of one drug recipe over another were lacking.²,³ Nowadays, a much more practical approach is applied wherein the neuroanesthesia drug regimen is selected with the surgical priorities in mind (integrated with knowledge about the drug’s effects on cerebral hemodynamics).⁴,⁵