DESFLURANE PHARMACOLOGY

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Desflurane’s structure is similar to isoflurane except for the substitution of a fluorine atom for isoflurane’s chlorine atom. The vapor pressure of desflurane is 681, therefore desflurane requires a special vaporizer. The low solubility of desflurane in the blood and body cause a very rapid uptake and wash-out of anesthetic. Hence, the alveolar concentration of desflurane will tend to approach the inspired concentration much more rapidly than other volatile anesthetics. Wake-up times are about half as long as those observed following isoflurane. This is primarily attributed to the low blood / gas (.45) partition coefficient.

Increasing the dose is associated with a decline in systemic vascular resistance leading to a fall in arterial blood pressure. Cardiac output remains relatively unchanged or slightly depressed at 1-2 MAC. There is a moderate rise in heart rate, central venous pressure, and pulmonary artery pressure that does not become as significant at low doses. Rapid increases of desflurane lead to transient elevations in heart rate, blood pressure and catecholamine levels.

Desflurane causes a decrease in tidal volume and an increase in respiratory rate. There is an overall decrease in alveolar ventilation that causes a rise in PaCO2. Desflurane also depresses the ventilatory response to PaCO2. Pungency and airway irritation during desflurane induction can be manifested by salivation, coughing, and laryngospasm. This makes desflurane a less than ideal choice for inhalation induction.

Desflurane vasodilates the cerebral vascular structures, increasing cerebral blood flow and intracranial pressure at normotension and normocapnia. Use of the agent also decreases cerebral metabolic rate of oxygen which tends to cause cerebral vasoconstriction and moderate any increase in cerebral blood flow. The cerebral vasculature remains responsive to PACO2, therefore, ICP can be lowered by hyperventilation.

Reference:


Ibid. Andrews, J. Inhaled Anesthesia Delivery Systems, pg. 184