N urogenic puhnonary edema (NPE) is an acu te life-threatening complication of the c ntral nervous system (CNS) injury. Anesthetics can either promote or inhibit the NPE development. We examined the role of different concentrations of isoflurane anesthesia (1.5 - 3%) on the developmen t of NPE in rats with balloon compressed spinal cord. The development of NPE was examined in vivo and on histological sections of lung tissue. Neurological recovery in animals anesthetized with 1.5% or 3% isoflurane was monitored using BBB and pian tar tests for 7 weeks post-injury. The grade of the spinal gray and white matter sparing was evaluated using morphornetry. The r le of gradually developed spinal cord lesíon and spínal cord transection in the developmen t of NPE were evaluated also. NPE developed in all animals anesthetized with 1.5-2% isofluran . Almost 42% of animals died due to massive pulmonary bleeding and suffocation; X-ray imaging, pulmonary index and histological sections showed massive NPE. More than 71 % of animals anesthetized with 2.5-3%
i oflu rane had no signs of NPE. Blood pressure rose more rapidly in animals from 1.5% group than in 3% group; this hypertensive reaction was caused by the sympathetic hyperactivi ty. Animals from 3% group recovered their motor and sensory func tions more rapidly than animals from 1.5% group; morphometry and MRl did not showed significant difference. Gradual or incomplete spinal cord compr ssion prevented the NPE development. NPE did not dev lop in the mod 1 of spin al cord transection. Low concentrations of isoflu rane promote the NPE in rats with spinal cord injury and significantly comp1icate the recovery of neurological functions. The most 1ikely mechanism of NPE development is the severe sympathetic ischarge on the basis of increased intracranial p ressure in an enclosed intracranial space above the spinal cord lesion site. This sympathetic hyperactivity might be prevented by a
deeper anesthesia level.