IMPROVING CARDIO-PULMONARY FUNCTION FOR A SAFER ANESTHESIA OF WHITE RHINOCEROS (*Ceratotherium simum*): USE OF OPIATE COCKTAILS TO INFLUENCE RECEPTOR EFFECTS

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Abstract

Current field anesthesia protocols for white rhinoceros (*Ceratotherium simum*), using a potent mu opioid agonist (etorphine) and a tranquilizer (azaperone), cause respiratory depression and drug-induced muscle rigidity and tremors that further impair respiration, leading to a cascade of marked cardio-pulmonary alterations including hypoxia, hypercapnia, hypertension, tachycardia and acidosis.[1,2] This hypoxia was corrected in a field situation by nasal intratracheal intubation with oxygen supplementation (15 – 30 L/min), but the acidosis and elevated CO_2 were not corrected; probably due to impaired ventilation and ventilation/perfusion mismatching in the lungs of recumbent rhinoceros.

A study is underway to develop safer field anesthesia protocols with improved muscle relaxation and less cardio-pulmonary alterations using a dosage of 40 – 90 mg butorphanol (a mixed mu opioid antagonist and kappa agonist) and 25 – 50 mg midazolam in combination with 2.0 – 3.5 mg etorphine in adult animals. In preliminary studies, the mu antagonist effect of butorphanol greatly lessens the respiratory depression[3] and muscle rigidity and tremors caused by etorphine.[4] Animals were less hypoxic with an increased respiration rate and slower heart rate plus a lower end-tidal CO_2. The majority of the rhinoceros became standing immobile within 10 min, which facilitated minor manipulative procedures and allowed crating without partial opioid reversal. Once in the crate, either naltrexone and/or diprenorphine were given. Naltrexone reversed the effects of both the butorphanol and etorphine. Diprenorphine appeared to only reverse the etorphine, leaving the sedative effects of butorphanol intact. Respiration improved in these animals and excessive head-pressing in the crate was not seen, with its potential to impair respiration.

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