# Assessment of Pupillary Function during Sedoanalgesia for Mechanical Ventilation

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## INTRODUCTION

Critically ill patients receiving prolonged sedoanalgesia (SA) for adaptation to mechanical ventilation (MV) are at risk for developing neurological complications while at the same time their neurological evaluation is limited. We describe the pupillary function in patients without neurological or ocular injury that were exposed to MV for respiratory insufficiency and received either Remifentanil or Dexmedetomidine for SA.

## METHODS

A prospective cohort study was performed in a general ICU at a large academic medical center. We studied pupillary function in 27 adult patients using a pupillometer Neuroptics ® . A total of 6 measurements were performed in each individual: 4 while receiving sedoanalgesia (OnSA) and 2 after discontinuation of it (OffSA) with patients fully awake. During the OnSA measurements all patients had the same depth of sedation (as measured by the Richmond Agitation Sedation Scale - RASS). All measurements were performed under similar ambient light conditions (as measured by a light meter). Patients were hemodynamically stable and with minimal or no use of vasopressors (none of them received Epinephrine drip).

We excluded patients with known ocular and/or neurological injury and that were receiving drugs that could alter the pupillary function.

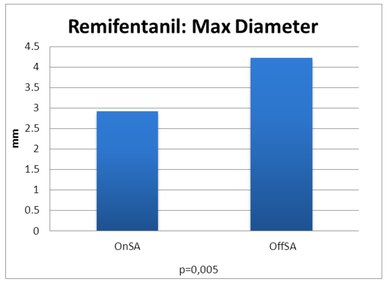
We used a T test for a sample with repeated measures. Each patient was compared to itself before and after the exposure. The test allowed us to compare the mean of the differences and its confidence interval. Given the small number of data we also used the Wilcoxon signed-rank test that further supported the statistical significance of our data.

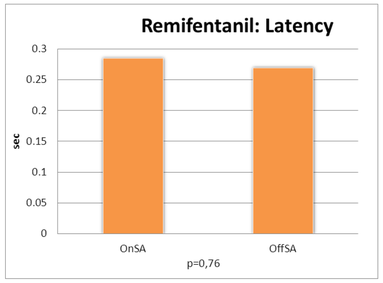
## RESULTS

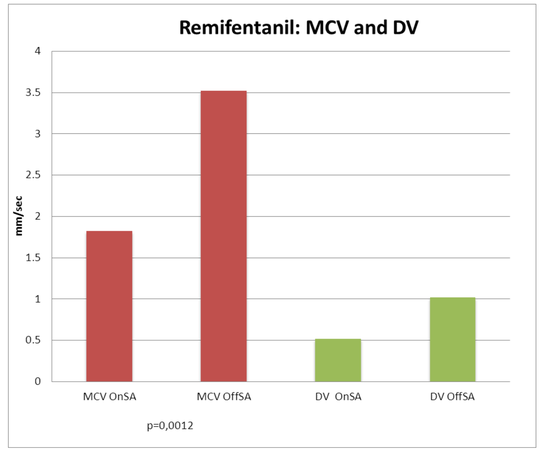
In the **Remifentanil** group (n: 11) the mean pupillary diameter was 2.91 mm (OnSA) and 4.22 mm (OffSA) (p=0.005). After exposure to light, the percentage of change of pupillary diameter was 12.07% (p=0.0009) and both the Maximum Constriction Velocity (MCV) and Dilatation Velocity (DV) changed significantly when comparing OnSA versus OffSA.

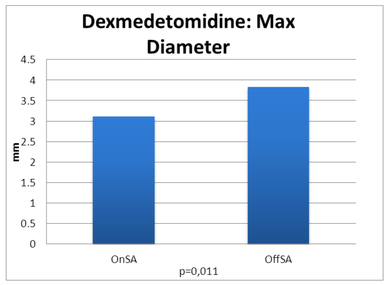
In the **Dexmedetomidine** group (n: 16) the mean pupillary diameter was 3.12 mm (OnSA) and 3.83 mm (OffSA) (p=0.011). After exposure to light, the percentage of change of pupillary diameter was 6.14% (p=0.001) and both the MCV and DV changed significantly when comparing OnSA versus OffSA. The Latency Period did not change in any group. (see Table and Graphs)

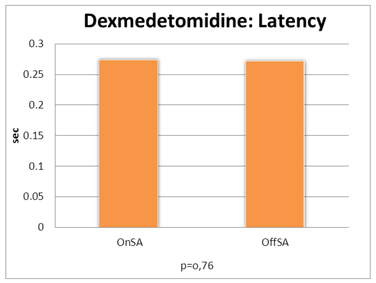
| | Max Diameter Difference | Percentage of Change | Latency | Maximum Constriction Velocity | Average Dilation Velocity |
|---|---|---|---|---|---|
| **Remifentanil** | 1.3 mm CI95% (0.49 a 2.11) **p= 0.005** | 12.07% CI95% (6.27 a 17.87) **p=0.0009** | -0.015 sec CI95% (-0.3 a 0.002) **p=0.76** | 1.69 mm/sec CI95% (0.84 a 2.54) **p=0.0012** | 0.5 mm/sec CI95% (0.3 a 0.7) **p=0.0002** |
| **Dexmedetomidine** | 0.72 mm CI95% (0.19 a 0.24) **p=0.011** | 6.14% CI95% (2.68 a 9.95) **p= 0.001** | -0.002 sec CI95% (-0.018 a 0.013) **p=0.76** | 0.82 mm/sec CI95% (0.24 a 1.4) **p=0.008** | 0.24 mm/sec CI95% (0.06 a 0.42) **p= 0.01** |

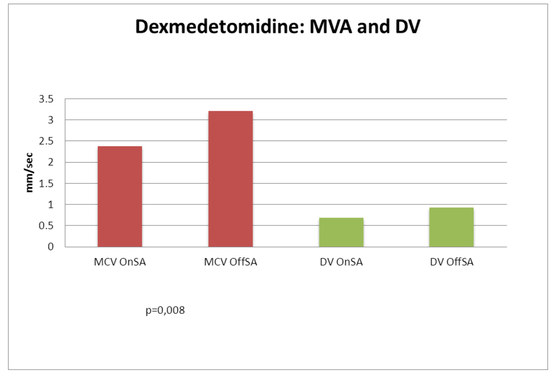



## CONCLUSIONS

Opiates such as Remifentanil are known to cause miosis. We now describe that Dexmedetomidine, at doses commonly used for intravenous sedation, alters pupillary physiology causing miosis and decreasing the constriction and dilatation velocities similar to the effects of Remifentanil infusion.

None of above mentioned drugs affects the Latency Period suggesting their lack of action on the afferent pathway of the light reflex.

Understanding the “expected normal” pupillary physiology in patients under SA with the studied drugs could help identify acute systemic or neurologic complications that may manifest as changes in the pupillary diameter.

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