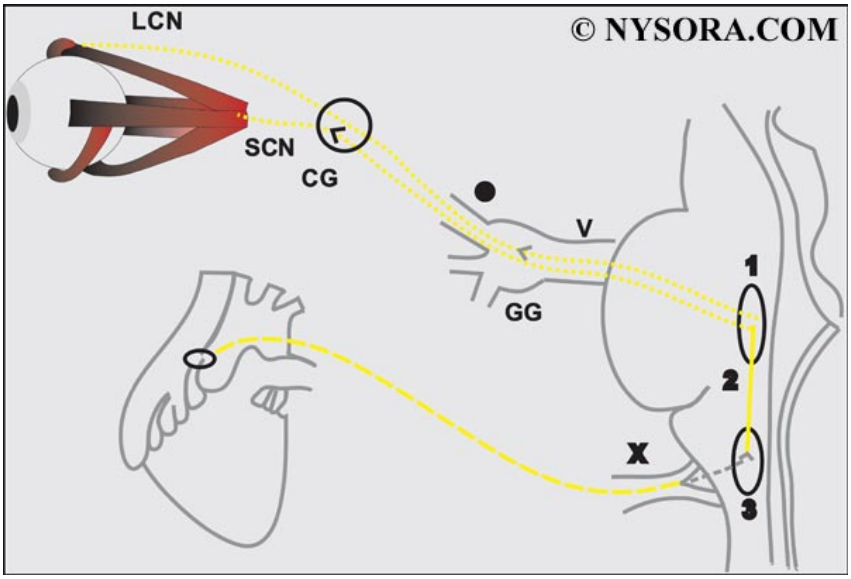
# Oculocardiac Reflex

**Anesthetic Pearls:** Anesthetic Causes and Implications of the OculoCardiac Reflex (OCR)

**Etiology**:

A. Pressure on the eyeball
B. Traction on the extraocular muscles
C. Orbital hematoma
D. Ocular trauma
E. Ocular pain




**Pathophysiology**:

1. Trigeminovagal reflex manifesting as bradycardia, nodal rhythms, ectopic beats, ventricular fibrillation, or asystole
2. **Afferent pathway**: long / short ciliary nerves ➔ Ciliary ganglion (CG) ➔ Gasserian ganglion (GG) along the ophthalmic division of Trigeminal nerve (V-2) ➔ termination in the main sensory nucleus in the floor of the 4th ventricle.
3. **Efferent pathway**: impulses start in the muscles ➔ Vagal (X) cardiac depressor nerves causing negative inotropic and conduction effects
4. Pathway is Trigeminal (V-2) to Vagal (X)

**Incidence**:

- Strabismus surgery (varies from 32-90%).
- Transient cardiac arrest as frequent as 1 in 2200.
- Influencing factors include force and type of stimulus (more acute the onset and more sustained the traction leads to a more likely OCR).
- Medial rectus muscle implicated as the most sensitive in eliciting a response.
- Hypoventilation and increased $PaCO_2$ increase the incidence of bradycardia during strabismus surgery.

**Treatment**:

**Stop the stimulus!** Fatigue of the reflex makes recurrence less likely and gentle handling of the muscles by the surgeons reduces the overall incidence of OCR. Atropine administration is controversial (pretreatment does not guarantee ablation of the reflex) and incidence of tachyarrhythmias may be higher. Lidocaine may be injected into the eye muscles to diminish sensitivity for the reflex. Normocarbia should be maintained throughout the anesthetic. Again, vigilance is key with all anesthetics!