Statements accompanying the thesis

Cerebellar impact on thalamocortical networks in epilepsy

1. GSWDs are messaged to the cerebellum via inferior olive and pontine nuclei as evidenced by the in part sole occurrence of Simple and Complex spiking in Purkinje cells

2. On-demand or chronic enhancement of cerebellar nuclei output can immediately stop or dampen seizure occurrence in different mouse models of absence epilepsy

3. Optogenetic cerebellar nuclei activation can induce differential effects *in vitro* and *in vivo* in different thalamic nuclei hinting towards desynchronization

4. Despite the support for cerebellothalamic input being a 'driver', the prolonged *in vivo* effects of CN stimulation do not exclude (in)direct activation of mGluR group 1 and group 2 receptors

5. Electrical stimulation of thalamic and cerebellar nuclei is not capable of stopping generalized febrile seizures in a SCN1A-deficient Dravet mouse model

6. Specific cerebellar nuclei stimulation paradigms is key for structural network changes aiming for chronic stabilization of epilepsy in pediatric patients using plasticity and neurogenesis

7. Firm conclusive statements about the efficacy of hippocampal, centromedian thalamic, nucleus accumbens and cerebellar DBS are impossible with the current evidence (Sprengers et al. 2017, Cochrane Database of Systematic Reviews)

8. The emotional and psychomotor stamina required of a practicing neurosurgeon to maintain efficacy at odd hours when meeting the needs of their patients is not improved by an externally dictated duty hour restriction. It also did not improve patient safety and technical training. (as reviewed by Bina et al 2016, journal of Neurosurgery)

9. Goede zorg leveren voor een patient komt op veel meer neer dan p-waardes en gerandomiseerd onderzoek.

10. The client or patient is not always right – (Adapted from) Enzo Ferrari

11. Niemand weet hoe laat het is. Youp van 't Hek

Oscar Eelkman Rooda