# OUTLINE: THE SYMPATHETIC INNERVATION OF THE HEART

1. Introduction
   1. Purpose
      1. SNA is important to heart – mediates normal reflexes and pathophysiology
      2. VT/VF/SCD is example of SNA to understand its consequences
   2. Objectives
      1. understand relevant anatomy/physiology including CV reflexes (examples including thoracic injury, heart transplant)
      2. Understand major example of VT/VF as how SNA is clinically important
         1. Ventricular substrate
         2. Coronary blood flow leading to VT/VF events
         3. MI/scar as triggers for VT/VF, as well as increased resting SNA
      3. Treatment paradigms including neurohormonal blockage (from stellate block to medicine)
2. Neurocardiac axis
   1. Example of importance of extra-cardiac features (e.g. Jonnesco and sympathectomy)
   2. Description of anatomy with figure
   3. Thoracic spinal cord injury and its effect on reflexes
      1. Bainbridge reflex – can mention cirrhosis example c- NE and bradycardia
      2. Bezold-Jarish reflex
      3. Baroreflex
3. Ventricular substrate
   1. History behind concept of ventricular substrate (John MacWilliam), including model of VT/VF as a result of a trigger and autonomic modulation
   2. Ventricular substrate and ventricular fibrillation overview
   3. Neuropsychological phenomenon that can change the VF threshold (review of Bernard Lown)
   4. Psychological stress and relationship to ectopy
4. Coronary blood flow
   1. Importance of CBF regulation in VT/VF events
   2. Coronary anatomy (mainly innervation)
      1. Normal reflexes and responses to stress
      2. Pathophysiological changes in reflexes due to SNA
   3. Acute myocardial ischemia
      1. Changes in VF threshold
      2. Response to beta blockade and importance of beta blockers in prevention
5. Myocardial ischemia/infarction and scars
   1. Chronic infarction/ischemia as cause for VT/VF
   2. Remodeling of heart after infarction and chronic ischemia
      1. Normal myocardial innervation patterns by sympathetic nerves
      2. Changes after infarction leading to nidus for VT/VF
   3. Chronic changes to resting SNA due to infarction
6. Catecholamine excess
   1. How do catecholaminergic states lead to increased VT/VF events?
   2. Important local and systemic neurotransmitters (nor epinephrine, epinephrine, galanin, neuropeptide Y, acetylcholine)
   3. Effect of neurohormonal regulation systemically
      1. Example of ICM c- remodeling of heart due to neurohormonal changes
      2. Effect of ACEI and stopping cardiac remodeling
   4. Effect of neurohormonal regulation locally
7. Conclusion
   1. Review central purpose of paper (SNA activity leads to normal cardiac control, but also pathological conditions)
   2. Treatment paradigms of the future (alpha-methyldopa, reserpine, ablation of cardiac ganglia, carotid sinus stimulation, vagal nerve stimulation)
   3. Future directions