methadone(1), ketamine(1) and gabapentin(6). Statistically significant results in pain reduction compared to placebo or standard pain medication were found in the studies concerning pregabalin \( p = 0.003 \), nortriptyline \( p = 0.04 \), methadone \( p = 0.03 \), ketamine \( p = 0.012 \) and in 2 out of 6 gabapentin studies \( p < 0.004 \). Two of the studies had no comparison arm.

**Conclusions** Interventions including pregabalin, nortriptyline, methadone, ketamine and gabapentin, were found to provide pain relief. While there is a plethora of pharmacological interventions available, only a few studies have been conducted regarding the pharmacological management of therapy-related-NP in HNC patients, including a small range of interventions. More studies should be conducted regarding the pharmacological approaches in HNC therapy-related-NP and the specific treatment algorithms.

### B159

**THE EFFECT OF PROPOFOL ON ROPIVACAINE-INDUCED CENTRAL NERVOUS SYSTEM TOXICITY IN PIGS**

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**Background and Aims** Ropivacaine is a long-acting local anesthetic, widely used in regional anesthesia. Although less toxic than bupivacaine, ropivacaine has been implicated in the occurrence of central nervous system toxicity. The primary objective of this study was to determine whether propofol at subanesthetic doses protects against ropivacaine induced central nervous system toxicity in pigs.

**Methods** A preliminary study to determine the dose, rate of administration and the plasma concentration of ropivacaine, which induces paroxysmal electroencephalographic activity (PEA) without causing cardiotoxicity, was performed in five pigs. Thereafter 20 pigs were divided in 4 groups of 5 receiving intravenously either ropivacaine alone, ropivacaine+propofol, ropivacaine+intralipid or propofol alone. Electroencephalogram (EEG) was recorded continuously (Nr of Ethic commission approval: APAFIS 28480–2020120112486566 v2III)

**Results** For similar blood levels in 4 out of 5 animals in the ropivacaine and in all in the ropivacaine+intralipid group PEA were observed and recorded. Bursts of PEA occurred similarly in the ropivacaine+intralipid and ropivacaine group. EEG in the ropivacaine+propofol group showed slow delta wave, but no PEA. In the propofol group stage 2 sleep-like activity was observed without PEA.

**Conclusions** Propofol in subanesthetic doses prevents in this model the occurrence of PEA induced by intravenous ropivacaine. A dose-response relationship of propofol on the occurrence of ropivacaine-induced paroxysmal electroencephalographic activity is likely. In patients receiving regional anesthesia, administration of a subanesthetic propofol dose could protect from ropivacaine-induced central nervous system toxicity.