PERCUTANEOUS RECORDING OF MUSCLE NERVE SYMPATHETIC ACTIVITY DURING PROPOFOL, NITROUS OXIDE AND ISOFLURANE ANESTHESIA IN MAN

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Introduction. Sympathetic nervous activity during anesthesia in man is often monitored by indirect parameters such as arterial blood pressure, heart rate, blood flow, p-cathecholamines etc. In this study we have applied a microneurographic percutaneous technique (1,2) for registration of muscle nerve sympathetic activity (MSA) during anesthesia with propofol, nitrous oxide (N_2O) and isoflurane.

Methods. Six patients (38-70 years of age) admitted for ENT-surgery entered the study after informed consent and approval by the local Ethics Committee. Following diazepam premedication a tungsten electrode (tip diameter a few μ) was introduced into a peroneal muscle nerve fascicle and located close to sympathetic nerve fibers. Measurements of intraarterial blood pressure and ECG were made. All signals were stored on magnetic tape.

Anesthesia was induced with propofol and norcuron was used as muscle relaxant. Before surgery the patients were ventilated artificially with O_2/N_2O or O_2/air with or without isoflurane as follows:

- 10 minutes with N_2O/O_2
- 10 min. with N_2O/O_2 + 0.3% isoflurane (=0.5 MAC)
- 10 min. with N_2O/O_2 + 0.6% isoflurane (=1.0 MAC)
- 10 min. with O_2/air + 0.6% isoflurane (=0.5 MAC)
- 10 min. with O_2/air + 1.2% isoflurane (=1.0 MAC)

The F_iO_2 was kept constant (0.30) as well as the end-tidal pCO_2 during the anesthesia.

The different variables were measured during 5 minutes before anesthesia, during one minute immediately before and after intubation and during the last 3 minutes of each 10 minutes-period. Both the number of pulse synchronic sympathetic bursts and their amplitude in a mean voltage neurogram were measured. The strength of activity was expressed as the product of bursts/minute and mean burst amplitude.

Results. (Figure 1) After induction with propofol MSA was reduced to 28% and mean arterial pressure (MAP) to 70% of control value. The intubation was associated with rapid increases in MSA and MAP to levels above control. Increasing doses of isoflurane were associated with a dose dependent decrease in MSA. The effect of isoflurane on MSA was significant ($p<0.05$) both with and without N_2O. When administration of N_2O was discontinued blood pressure increased and MSA decreased considerably ($p<0.01$).

Discussion. The present study provides the first direct measurements of MSA during anesthesia. The parallel reduction of MSA and blood pressure after induction suggests that propofol causes a central sympathetic inhibition which is effectively counteracted by the exitatory effect of intubation. As expected we see a similar inhibition during increasing doses of isoflurane. It is notable that discontinuation of N_2O with decrease in anesthetic depth (1.0 to 0.5 MAC) show a decrease in MSA. This may be due to several factors such as activation of the baroreceptor reflex or a central sympathetic activation of N_2O.

References.

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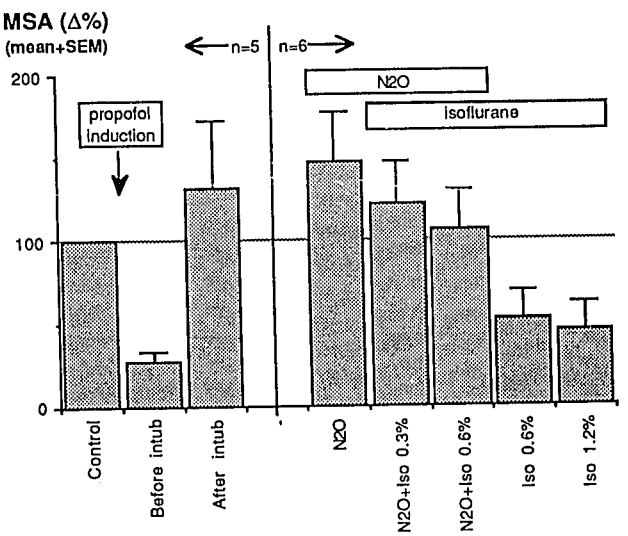

Figure 1. Relative changes of MSA compared to the control period.