Toxic inhalation with hydrogen cyanide prompts a significant increase of mean pulmonal arterial pressure in an animal model

Beelitz J¹, Kill C¹, Feldmann C², Wulf H², Vogt N³, Veit F⁴, Dersch W²

¹ Department of Emergency Medicine, Department of Anaesthesiology and Critical Care, Philipps-University, Marburg, Germany; ² Department of Anaesthesiology and Critical Care, Philipps-University, Marburg, Germany; ³ Department of Neurosurgery, University of Giessen, Giessen, Germany; ⁴ Institute of Legal Medicine, Department of Forensic Toxicology, Justus-Liebig-University Giessen, Giessen, Germany

Objective

Toxic smoke inhalation contains mainly two major toxic gases carbonmonoxide (CO) and hydrogen cyanide (HCN) leading to hypoxia of cells [1,2]. We investigated the effect of prolonged hydrogen cyanide (HCN) exposure on pulmonal arterial blood pressure during intoxication in a smoke inhalation pig model.

Methods

With approval by local authorities an intoxication model for pigs (25-35kg) with anaesthesia, intubation and instrumentation including a pulmonal arterial catheter was used. Smoke intoxication was simulated by controlled ventilation in a closed circuit with: t=0min (baseline, no toxic gases), t=5min air with CO=1500ppm (parts per million) over 5 minutes, t=10min air with CO=750ppm and HCN=500ppm over 5minutes, t=15min air with HCN=1000ppm. Inhalation was continued until cardiac arrest occurred. The mean pulmonal arterial pressure (mPAP) was recorded from t=0min until t=30min. Analysis with Wilcoxon-Test compared to baseline. Results as median (25/75%percentiles).

Results

Cardiac arrest occurred in all pigs in a median time of 38min(25.5/48.5), so that 16 of 24 animals could be included until t=30min. t=0min 21mmHg(17/23), t=5min 21mmHg(19/30), p=0.1680; t=10min 37mmHg(33/43), p=<0.0001; t=15min 36mmHg(33/41), p=<0.0001; t=20min 31 mmHg(29/34), p=0.0003; t=25min 27mmHg(21/31), p=0.0259; t=30min 22mmHg(19/25), p=0.3745.

Mean pulmonal arterial pressure (mPAP) and mean arterial pressure (MAP) during inhalational cyanide intoxication

Conclusion

Hydrogen cyanide inhalation caused an early and significant increase of mPAP. During prolonged intoxication and shortly before cardiac arrest occurred mPAP decreased to baseline levels. Further investigations are needed to investigate the effects of hydrogen cyanide on pulmonary vascular tree in detail.

References

- [1] Postgrad Med. 1987;82(1):63-8
- [2] Crit Rev Toxicol. 2002;32(4):259-89.

Contact: Jost.Beelitz@gmx.de