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MODERATE HYPOTHERMIA AFTER A SHORT PERIOD OF HYPOXIA IMPROVES ELECTROPHYSIOLOGICAL RECOVERY IN THE RAT HIPPOCAMPAL SLICE

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Introduction: Hypoxic brain injury is one of the most devastating complications in the perioperative period. Hypothermia has been shown to be protective when applied, in animal models, before or during hypoxia. It is unclear whether hypothermia is protective when applied after hypoxia. This study was designed to determine if hypothermia, applied immediately after hypoxia, improves electrophysiological recovery in the rat hippocampus.

Methods: The study was approved by the Institutional Animal Care and Use Committee. Hippocampal slices were prepared from adult male S-D rats by cutting the hippocampus transverse to its long axis to preserve the synaptic connection between the Schaffer collateral pathway and the CA1 pyramidal cells. The slices were superfused with artificial cerebrospinal fluid (aCSF) aerated with $95\%O_2$-$5\%CO_2$ at 37°C. A postsynaptic population spike was evoked in the CA1 pyramidal cells by stimulating the Schaffer collaterals. The evoked response in the CA1 cells represents a summation of the electrical activity of these cells and its amplitude correlates with the number of life, functioning cells. The amplitude of the postsynaptic population spike in the CA1 cells was used to evaluate the effect of hypothermia on the hypoxic injury. Hypoxia was achieved by superfusing the slices with aSCF aerated with $95\%N_2$-$5\%CO_2$ for 3, 3.5 or 4 minutes. After hypoxia the temperature of the aCSF was kept at 37°C in the normothermic group and was reduced to 30°C in the hypothermic group for a period of 30 minutes, after which the temperature was returned to 37°C for the remainder of the recovery period. Recovery from hypoxia was calculated by dividing the amplitude of the population spike obtained 2 hours after the termination of hypoxia by the base line amplitude obtained immediately before the institution of hypoxia. An unpaired t test was used and $p<0.05$ was considered significant.

Results: During hypoxia there was a loss of the postsynaptic population spike in the CA1 pyramidal cells. After 3 min of hypoxia, the recovery of the population spike in the normothermic group (n=12) was 24±11% (mean±SEM) and in the hypothermic group (n=8) 66±9% (p=0.01). After 3.5 min. of hypoxia, the recovery in the normothermic group (n=6) was 21±9% and in the hypothermic group (n=10) 44±19% (p=0.41). After 4 min. of hypoxia, the recovery in the normothermic group (n=7) was 23±17% and in the hypothermic group (n=8) 6±6% (p=0.32).

Conclusion: Hypoxia causes damage to the electrophysiologic response of the hippocampus. Moderate hypothermia applied immediately after hypoxia improves recovery of the CA1 population spike after 3 min but not after 3.5 min or 4 min of hypoxia.

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TITLE: CHEST RADIOGRAPH INTERPRETATION SKILLS AMONG ANESTHESIOLOGISTS

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ABSTRACT

Background: The objective of the study was to assess the skills of anesthesiologists in the interpretation of chest radiographs.
Methods : An evaluation of chest radiograph diagnostic skills was conducted at the Postgraduate Assembly of the New York State Society of Anesthesiologists in 1999. After completing a demographic survey participants were asked to review a series of ten chest radiographs. A brief clinical scenario accompanied each radiograph. No time limit was set for these interpretations.
Results : A total of 61 anesthesiologists (48 attendings; 13 residents) volunteered. The demographic characteristics of the participants included university faculty (46%), private group practitioners (41%), independent practitioners (11%), and one with an unspecified type of practice. Additional training among the participants included internal medicine (31%), surgery (19%), and pediatrics (3%). Thirty four percent did not specify any additional training. Ninety two percent of the participants were involved in cases requiring general anesthesia. Ninety six percent manage patients in the recovery room, and 34% in the intensive care unit. Eighty seven percent of participants usually order chest radiographs, but only 42% interpret the films themselves.The rates of misdiagnosed radiographs included pneumothorax (11%), free air under the diaphragm (41%), bronchial perforation from a nasogastric tube (28%), right mainstem intubation (20%), superior vena cava perforation from a central venous catheter (31%), normal film (75%), negative pressure pulmonary edema (16%), left lower lobe collapse (80%), pulmonary infarction from a pulmonary artery catheter (29%), and tension pneumothorax (41%). Overall scores of the attendings were not significantly different from that of residents ($p > 0.05$).
Conclusions : Anesthesiologists lack skills in interpretation of chest radiographs. The skill level of university-based physicians is not greater than those in private practice, and does not improve with level of training or experience. Most anesthesiologists rely on radiologists for interpretative results. Further training during the residency years may help improve their diagnostic skills.
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INTERPRETATION OF CHEST RADIOGRAPHS

RADIOGRAPH DESCRIPTION	CORRECT% (N=61)	INCORRECT % (N=61)
Pneumothorax	89 (54/61)	11 (7/61)
Free Air Under Diaphragm	59 (36/61)	41 (25/61)
Bronchial Perforation/Pneumothrax from Nasogastric Tube	72 (44/61)	28 (17/61)
Right Mainstem Intubation	80 (49/61)	20 (12/61)
Superior Vena Cava Perforation from central venous line	69 (42/61)	31 (19/61)
Normal (skin fold)	25 (15/61)	75 (46/61)
Negative Pressure Pulmonary Edema	84 (51/61)	16 (10/61)
Total Left Lower Lobe Collapse	20 (12/61)	80 (49/61)
Pulmonary Infarction from overwedged PA catheter	71 (43/61)	29 (18/61)
Tension Pneumothorax	59 (36/61)	41 (25/61)