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| **The role of the inner ear in arousal. findings under light anesthesia and natural sleep conditions: Potential implication for the Sudden Infant Death Syndrome** |
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| **Introduction** **INTRODUCTION**: Infants that succumb to the Sudden Infant Death Syndrome (SIDS) have been found to have inner ear dysfunction at birth and on autopsy: A suppressed newborn hearing profile and bleeding with inflammation has been identified on post mortem analysis of SIDS cases 1,2. We previously investigated in an animal model whether inner ear dysfunction could play a mechanistic role in SIDS. We discovered that animals with inner ear dysfunction displayed significant suppression of the movement arousal response to a hypoxic-hypercarbic gas mixture while asleep under light anesthesia (3,4). In the current study we set out to investigate the individual role of each of the gases in this response and also to investigate the arousal response to hypercarbia during natural sleep without anesthesia.  **Material and Methods** **MATERIALS AND METHODS:** Wild type CD-1 mice at 17 days of age received intra-tympanic gentamicin (IT-Gent) injections bilaterally or unilaterally to precipitate inner ear dysfunction. Controls included wild type mice with no intervention and mice that received intra-tympanic saline. The movement response to hypercarbia or hypoxia was tested under light anesthesia, four days after injections. The arousal movement to each gas was compared to that under the combined hypoxic-hypercarbic gas mixture. A significant movement arousal response was defined as a head movement greater than 10 cm from its original position.  **Results** **RESULTS:** Hypercarbia did not stimulate any vigorous movement responses in test or control animals under light anesthesia or under natural sleep conditions. Hypoxia triggered vigorous arousal movements in control animals (p<0.05) and a decreased yetadequate movement response in unilateral IT-Gent animals under light anesthesia (Figure 1). This contrasted with the responses to combined hypoxia-hypercarbia, in which unilateral IT-Gent animals displaced significantly suppressed movements compared to control animals (p<0.05). Bilateral IT-Gent animals did not make any significant movements in response to any of the experimental conditions (p<0.05). We identified small spontaneous stirring movements (‘”mini-arousals”) in all animals during the natural sleep state that were not seen under light anesthesia. There was no difference in the number of mini arousals seen during natural sleep between IT-Gent or Control animals during exposure to hypercarbia versus air (p<0.05).  **Conclusions** **CONCLUSION:** Our findingsportray that a degree of intact inner ear function is necessary for instigating a movement arousal response. Additionally, hypoxia is the trigger for the movement arousal response and carbon dioxide *suppresses* this response. The finding that carbon dioxide also did not stimulate movement arousal in the natural sleep state is a novel and important finding (Figure 1). These findings, which focus on the movement component of the arousal response, contrast with other studies that have identified hypercarbia as an arousal stimulus. Additional studies of the arousal response under the natural sleep state to gas mixtures other than hypercarbia are warranted. Further studies are warranted to evaluate the precise role of the inner ear in the arousal response and a potential association with SIDS. The early detection and treatment of inner ear dysfunction in SIDS predisposed cases might be a novel and important instrument for alerting and preventing a fatal event during sleep. |
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