

20.7

SLEEP-DISORDERED BREATHING (SDB) EVENTS ARE RELATED TO PHASIC REM CHANGES IN RESPIRATORY MUSCLE ACTIVITY IN ENGLISH BULLDOGS. J.C.Hendricks, R.J.Kovalski, and L.R.Kline. Univ. of Pennsylvania, Philadelphia, PA 19104.

English bulldogs have SDB, as measured by decreased ribcage (RC) and/or abdominal (ABD) movements and $\geq 4\%$ drops in oxygen saturation (SaO₂). Since SDB is worst during REM sleep, we hypothesized that the changes in respiratory muscle control found during phasic REM might contribute to SDB. These include intermittent decrements in drive, pauses in inspiratory activity lasting 20 to 150 ms ("fractionations"), and asynchrony. These changes are seen in normal cats, people, and dogs. We instrumented 5 English bulldogs to record EEG, RC and ABD, airflow, SaO₂, and EMG of the diaphragm and sternohyoid (SH, a sample upper airway dilator). The mean rate of SDB was 61/REM hr. In all 5 dogs, SDB events were associated with an initial decrease in drive to both muscles ($p < 0.01$, paired t-test). The decrease was greater in the SH than in the diaphragm ($p < 0.01$, multivariate t-test). Other changes in EMG pattern, including fractionations and asynchrony, were also significantly associated with SDB ($p < 0.01$, paired t-test). We conclude from these data that REM-related changes in respiratory muscle activity compromise ventilation and, in the presence of an anatomically narrowed upper airway, cause decrements in saturation. This mechanism contrasts with the cyclic changes in automatic control of breathing thought to lead to SDB in non-REM sleep.

PULMONARY REFLEXES

21.1

MECHANISM OF BRADYPNEA INDUCED BY INHALATION OF GAS PHASE CIGARETTE SMOKE IN ANESTHETIZED RATS. L.-Y. Lee, R.F. Morton* and A.L. Wang* Univ. of Kentucky, Lexington, KY 40536

A possible involvement of oxygen radicals in evoking the acute effects of inhalation of gas phase (GP) cigarette smoke on breathing was examined in young Sprague-Dawley rats anesthetized with chloralose-urethane. Spontaneous inhalation of GP smoke (6 ml, 50% concentration) elicited a transient inhibitory effect on breathing, prolonging Te to a peak of $159 \pm 6\%$ of the average base-line Te; this response was only slightly lower than that triggered by inhaling the unfiltered cigarette smoke (peak Te = $177 \pm 12\%$; $p = 0.04$, $n = 20$). The bradypnea started within 1-4 breaths after the onset of GP smoke inhalation, lasted for 3-5 breaths and was completely abolished by bilateral vagotomy. This inhibitory effect of GP smoke on breathing was not affected ($p > 0.1$, $n = 11$) by a pretreatment with superoxide dismutase (30,000 units/kg, i.v.) plus catalase (480,000 units/kg, i.v.), enzymes metabolizing superoxide radical and hydrogen peroxide. However, the bradypnea was largely prevented (peak Te = $109 \pm 2\%$, $n = 12$) by a pretreatment with a hydroxyl radical scavenger, dimethylthiourea (500 mg/kg, i.v.). These results suggest that the gas phase is primarily responsible for eliciting the reflexogenic inhibitory effect of cigarette smoke on breathing in anesthetized rats and that hydroxyl radicals released endogenously in the lung may be involved. (Supported by grants NIH HL-40369 and UK/THRI 41066)

21.3

COUGH AND THE NASAL AIRWAY IN ANAESTHETIZED DOGS. James C.C. Wang* and Mary A. Lung. Department of Physiology, University of Hong Kong, Hong Kong.

The study was undertaken to investigate the action of coughing on the airflow resistance in the nasal airway. In pentobarbitone anaesthetized dogs with controlled vascular perfusion of the nasal mucosa, nasal arterial inflow, venous outflows, vascular resistance and nasal airflow resistance were monitored as described (J. Physiol. 391, 57-70, 1987). Pleural pressure was assessed by a catheter-tip pressure transducer which was inserted into the thoracic cavity via an oesophageal incision. Mechanical stimulation of the lower airways with a soft polyethylene thread induced coughing, an increase in nasal vascular resistance and venous outflows, and a decrease in nasal airflow resistance. Hence, stimulation of cough receptors causes constriction of both resistance and venous vessels resulting in a decrease in nasal airflow resistance.

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21.2

CHEMICAL DESTRUCTION OF CELLS IN THE CAUDAL VENTROLATERAL MEDULLA (CVL) ATTENUATES BRONCHODILATOR REFLEXES, AS WELL AS BRONCHODILATION EVOKED FROM THE POSTERIOR MEDIAL HYPOTHALAMUS (PMH) IN DOGS. P.A. Padrid, J.R. Haselton, M.P. Kaulman. Div. of Cardiovascular Medicine, University of California, Davis, CA 95616

The CVL in the area around the lateral reticular nucleus (LRN) is believed to be involved in integration of afferent impulses resulting in the exercise pressor reflex. This may be due in part to tonic inhibition of areas within the rostral ventrolateral medulla (RVL) which exerts a sympathetic excitatory influence on the cardiovascular response to exercise. The role of the CVL in integration of afferent impulses resulting in exercise induced reflex bronchodilation, as well as the bronchodilator responses to electrical stimulation of the PMH and sciatic nerve (SN), has not been addressed. Bronchomotor responses to the above stimuli, as measured by changes in total lung resistance (TLR), were recorded before and one hour after bilateral injection of ibotenic acid (IA) (to destroy cell bodies) into the CVL in the area around the LRN. Before placement of IA, static contraction of both hindlimbs (SC) decreased TLR ($\bar{x} \pm SE$) from 8.1 ± 0.6 to 7.2 ± 0.6 ($P < 0.05$; $n = 8$), electrical stimulation of SN decreased TLR from 9.2 ± 0.8 to 7.8 ± 0.7 ($P < 0.05$; $n = 8$) and electrical stimulation of the PMH decreased TLR from 9.5 ± 0.7 to 8.0 ± 0.7 cm H₂O/L/s ($P < 0.05$; $n = 8$). One hour after injection of IA, SC resulted in an increase in TLR from 8.5 ± 0.7 to 9.2 ± 1.0 , stimulation of SN increased TLR from 9.2 ± 0.8 to 9.4 ± 1.1 , and stimulation of the PMH increased TLR from 9.0 ± 0.9 to 9.1 ± 1.5 cm H₂O/L/s. In 3 dogs, bilateral injection of IA dorsomedial to the LRN had no effect on the bronchomotor response to SC, SN or PMH stimulation. In one dog, bilateral injection of cobalt chloride into the CVL reversibly attenuated the above responses. We conclude that the CVL may be a site of integration of afferent impulses arising from stimulation of the PMH and SN afferents, as well as the exercise induced bronchodilator reflex.

21.4

EFFECT OF INHALATION OF THE ERUCTED GASES ON THE BREATHING PATTERN IN CATTLE.

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It is well known that most of the eructed gases (40-60% CO₂) are inhaled in ruminants. The aim of this study was to evaluate the effect of inhaled eructed gases on the breathing pattern in cattle.

The physiological phenomenon of belching was simulated and its effect on the peripheral chemoreceptors was evaluated in 4 Friesian and 2 Belgian White and Blue calves (age : 220 ± 25 days; weight : 179.2 ± 22.0 kg) using a single tidal-breath of approximately 50% CO₂ in equal parts of O₂ and N₂. The experimental apparatus consisted of a tight-fitting mask, a Fleisch pneumotachograph Nr 3, a giant Hans-Rudolph valve, a manual bidirectional valve and a bag which contained the test gas. Respiratory gases were continuously analysed on a breath-by-breath basis, by a mass spectrometer. After a period of breathing in normal conditions, the calves were exposed to the test gas by turning the bidirectional valve during the expiratory phase of the previous respiratory cycle. On the other hand, the effects of physiological eructations were also studied.

Minute volume (V_e) and mean inspiratory flow (V_I = V_I/T_I) were significantly increased from the second or third breath after the simulated eructation. The peak increase was 80% and 62% in V_e and V_I/T_I respectively. The rise in V_e was exclusively due to an increase in V_T (Tidal volume) while the respiratory rate remained constant. The same phenomena were observed after physiological eructations.

These data show that eructation significantly influences the pattern of breathing in cattle and that the ventilation in basal condition in this species is characterized by a cyclic variability coupled to belching.