

## CARTAS AL EDITOR

## Reflex Apnea and Bronchodilation in the Cat

The selective stimulation of the laryngeal mucosa evoked a reflex response consistent of apnea, normally expiratory, followed by a glottic closure (3, 6, 7, 10). This study has aimed to assess the effects of bronchodilation on the different components of the laryngeal reflex.

Experiments were performed in 10 cats ( $3.4 \pm 0.4$  kg, b.w.) anaesthetized with a mixture of Ketamine and Xylazine (15 mg/kg and 1 mg/kg, i.m., respectively). The *in situ* isolated glottis technique (1, 7) allowed to obtain the values of respiratory flows and subglottic pressure independently. Pleural and blood pressures were also recorded. Total lung resistance was displayed on an oscilloscope screen from transpulmonary pressure and respiratory flow values. The laryngeal reflex was induced by mechanical stimulation of the infraglottic mucosa with a nylon fibre introduced through the tracheal cannula. The stimulation was made in both control and bronchodilation situations. The bronchodilation was induced by administration of Fenoterol (10  $\mu$ g/kg, i.v.) and two hours later, by administration of Isoprenaline (0.1 mg/kg, i.v.). A decrease of total lung resistance was evoked with both Fenoterol ( $p < 0.01$ ) and Isoprenaline ( $p < 0.01$ ). The adrenergic agonists also produced a decrease of subglottic pressure ( $p < 0.01$ , both drugs), as previously indicated (4, 5).

In relation to control value, the laryngeal reflex response to the subglottic stimulation, presented two components: an expiratory apnea and an increase in the sub-

glottic pressure, accompanied by glottic closures. During the induced pharmacological bronchodilation, there was a significant decrease in the duration of the apnea while the increase in subglottic pressure persisted (table I). This reduction in apnea duration, that has not been previously described, could be determined by the pharmacological modification of the bronchomotor tone, activating reflex mechanisms, although some central effects cannot be discarded. It has been also described a decrease of the cough reflex during bronchodilation by adrenergic agonists in both humans and animal experimentation (8, 9, 11) although other reports refer no changes (2, 12). The decrease of the apnea duration and the persistence of the increase of subglottic pressure during

Table I. Reflex response to subglottic stimulation during bronchodilation

	Apnea duration (s)	Subglot. press. (cm H <sub>2</sub> O)
<i>Control</i>		
before stim.	—	$1.15 \pm 0.25$
during stim.	$6.42 \pm 1.71$	$4.08 \pm 0.71$
<i>Fenoterol</i>		
before stim.	—	$1.04 \pm 0.24$
during stim.	$4.28 \pm 1.70^*$	$3.71 \pm 1.20$
<i>Isoprenaline</i>		
before stim.	—	$0.80 \pm 0.23$
during stim.	$4.14 \pm 2.03^*$	$3.42 \pm 1.30$

\*  $p < 0.05$  (Student's *t* and variance tests; the statistical significances are referred to the variations of the laryngeal reflex during bronchodilation related to the laryngeal reflex during control). Number of cats, 10.

pharmacological bronchodilation suggest that the two components of the laryngeal reflex could be centrally integrated through different mechanisms.

**Key words:** Bronchodilation, Apnea, Laryngeal reflex.

**Palabras clave:** Broncodilatación, Apnea, Reflejo laríngeo.

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