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Tonic sensory pathways of the respiratory system

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ABSTRACT: Both respiratory centres and the preganglionic vagal motoneurones, which control respiratory (striated) and airway (smooth) muscles respectively, receive information on the lungs, the circulation and the skeletal and respiratory muscles. Each of these nervous pathways has two components: one is phasic, *i.e.* in phase with biological rhythms, and comes from mechanoreceptors connected to large myelinated fibres; the second has a tonic low frequency firing rate and corresponds to the spontaneous activity of polymodal receptors connected to thin sensory fibres, which act mostly as sensors of changes in extracellular fluid composition (O_2 and/or CO_2 partial pressure, pH, release of algesic agents etc...). Some of them also detect large mechanical disturbances or local temperature changes. The influence of tonic background sensory activity is well known in animals concerning the role played by arterial chemoreceptors in the control of ventilation and of thin vagal afferents from the lungs (bronchopulmonary C-fibres and irritant receptors) in reflex facilitation of the bronchoconstrictor vagal tone. Moreover, the stimulation of thin sensory fibres in particular circumstances is responsible for hyperventilation (arterial chemoreceptors and muscle afferents), increased airway tone (arterial chemoreceptors and mostly thin vagal afferent fibres) or bronchodilation (muscle afferents). These peripheral inputs project centrally on different structures and also on brain stem neurones, which integrate simultaneously chemosensory, vagal and muscle information. This results in complex interactions between the different sensory pathways.
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There are two closely connected motor pathways in the respiratory system. The first has its origin in the brain stem neurones which control breathing rhythm and tidal volume. The second arises in the preganglionic vagal motoneurones, located in the nucleus ambiguus and the dorsal motor nucleus of the vagus nerve, which control airway smooth muscle tone. Both motor pathways receive peripheral afferents, with information concerning the mechanical and chemical state of the lungs (afferent vagal fibres), the efficiency of circulation and respiratory gas exchanges (arterial baro and chemoreceptors) and the strength of contraction in respiratory and other skeletal muscles (muscle or somatic afferents) (fig. 1). This constitutes numerous and complex feed-back reflex loops, allowing the adaptation of ventilation and perhaps also of inspired air distribution to various physiological circumstances.

Each of the visceral and somatic sensory pathways has two components. One is modulated in phase with biological rhythms (tidal lung inflation, systolic blood pressure, isotonic muscular contraction). These phasic inputs have a high peak firing rate, 100 $c \cdot s^{-1}$ for muscle spindles as for vagal lung receptors [43] and are from mechanoreceptors connected to large myelinated (*i.e.* fast conducting) fibres (pulmonary stretch receptors, arterial baroreceptors, muscle proprioceptors as muscle spindles and Golgi tendon organs). They display a very slow adaptation in response to a sustained mechanical stimulation.

The second sensory component has a tonic low frequency firing rate (less than 4 or 5 $c \cdot s^{-1}$), sometimes inconsistently related to the respiratory cycle (lung receptors) or to rhythmic but strong or isometric muscular contractions (muscle afferents). This corresponds to the spontaneous activity of receptors which are free nerve endings connected to slow conducting fibres, *i.e* small myelinated (1–6 μm) and mostly unmyelinated fibres (0.2– 1.8 μm) (lung irritant receptors, bronchopulmonary vagal C-fibres, arterial chemoreceptors, group III and IV muscle fibres). Afferent unmyelinated fibres, which are called C-fibres in the vagus nerve and group IV fibres in somatic nerves, constitute 90% of sensory pulmonary vagal fibres [19] and at least 50% of carotid sinus or aortic chemosensory fibres [12] and skeletal or diaphragmatic afferents [10, 49]. As shown in figure 2, small myelinated sensory fibres, called B-fibres in the vagus nerve (lung irritant receptors) and group III fibres in muscle nerves, constitute a very small proportion of total afferent fibres in each nerve.

The present paper will be focused on the circumstances of the activation of thin afferent fibres, their role in control of ventilation and bronchomotor tone, and their central projections and interactions. Most of the data reported here concerns vagal and muscle

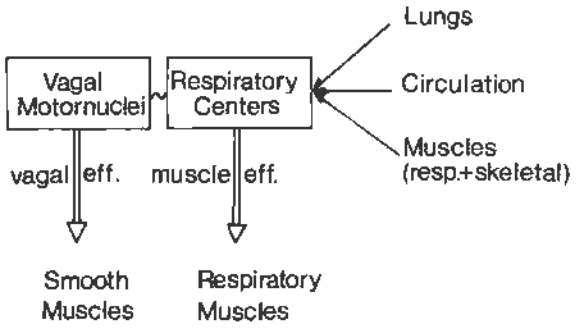

Fig. 1. Schematic representation of the respiratory control system, including both motor drives to respiratory muscles (muscle efferents) and smooth airway muscles (vagal efferents)) and the three main origins of sensory information.

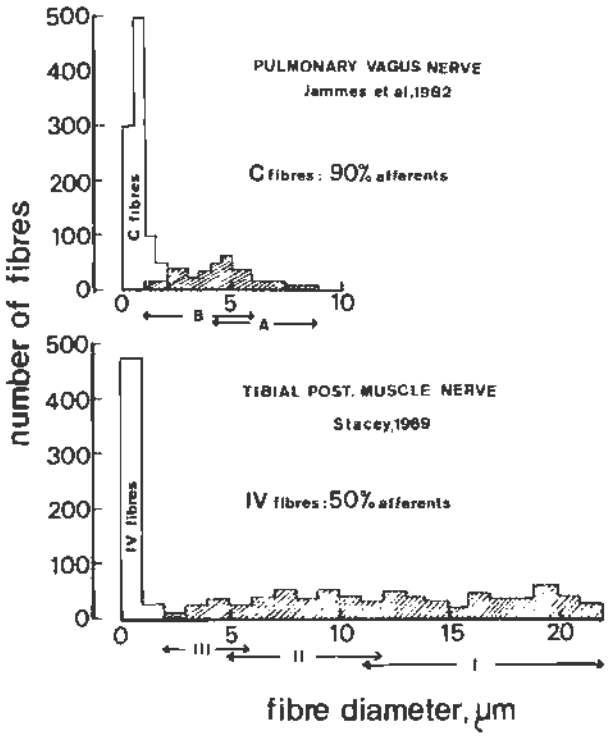

Fig. 2. Histograms of fibres obtained from electron micrographs in two sensory nerves (cats). Bronchial vagal branches are sampled after degeneration of vagal motor fibres and the tibial posterialis muscle is a sensory nerve. Open areas correspond to unmyelinated fibres (C-fibres for the vagus nerve and group IV fibres for somatic nerves) and dashed areas indicate myelinated fibres [redrawn from 17 and 49].

afferents, but we add some recent information on the role played by chemosensory inputs in the bronchomotor control and their interactions with vagal and somatosensory pathways.

Electrophysiological characteristics and stimuli of thin fibres

Tonic sensory pathways result from the spontaneous, low frequency discharge or the activation of rapidly adapting receptors. Most of them are polymodal receptors, which respond to mechanical, chemical and thermal stimuli. In general, normal tidal volume changes [4, 9] and isotonic contractions of skeletal [35] and respiratory muscles [24] are insufficient mechanical stimuli for these receptors. However, a subpopulation of small myelinated units are more sensitive to mechanical events than unmyelinated fibres. For example, lung irritant receptors increase markedly their activity when tidal volume or airflow rate or both increase and they are also excited by deflation of the lungs [4]. In addition, more than 40% of group III muscle fibres are low-threshold pressure-sensitive units, also activated during sustained tetanic contractions, compared to only 20% of group IV fibres [35]. Unmyelinated afferent fibres are particularly sensitive to chemical stimulation. Thus, vagal bronchopulmonary C-fibres are strongly stimulated by chemicals formed and released in the lungs in pulmonary anaphylaxis (histamine, serotonine, bradykinin, prostaglandins) [4].

Recent data also show that a large proportion of bronchopulmonary C-fibre afferents reflect the CO_2 content of mixed venous blood and expired gas [9, 42]. However, due to the very rapid adaptation of their discharge, only the peak firing frequency is proportional to the magnitude of CO_2 load [9] (fig. 3). This serves to distinguish them from arterial chemoreceptors which display a tonic and very slow adapting response to hypoxaemia or hypercapnia [31].

Group III and IV muscle afferents from skeletal muscles [35] and diaphragm [15, 24] are also sensitive to changes in the chemical composition of the extracellular space (osmolarity, pH, algesic agents) (fig. 4). Hypoxia has no effect on lung vagal sensory fibres [9] and its effect on group III and mostly group IV muscle fibres seems to result from consecutive acidosis [15, 29] (fig. 5).

Finally, many thin fibre afferents, especially unmyelinated fibres, are influenced by temperature. There are warm-sensitive vagal units in the lower trachea and intrapulmonary airways, activity of which is markedly reduced or abolished by cooling the inspired air from 35 to 30 °C [9] (fig. 6A). On the other hand, cold-sensitive units, with a threshold temperature of around 22 °C, have recently been identified in the superior laryngeal nerve [27] (fig. 6B). These latter behave like specific thermoreceptors because they are not activated by mechanical stimulation but only by cold and by injection of drugs. Similarly, both warm and cold-sensitive group IV units are present in afferents from skeletal muscles [35] and exhibit a response behaviour very similar to that of specific thermoreceptors in the skin.

Functional role of thin fibre afferents

The functional role of tonic sensory pathways can be analysed by examining either the effect of spontaneous background activity or the effect of increased discharge of thin fibre afferents on the ventilatory and bronchomotor controls.

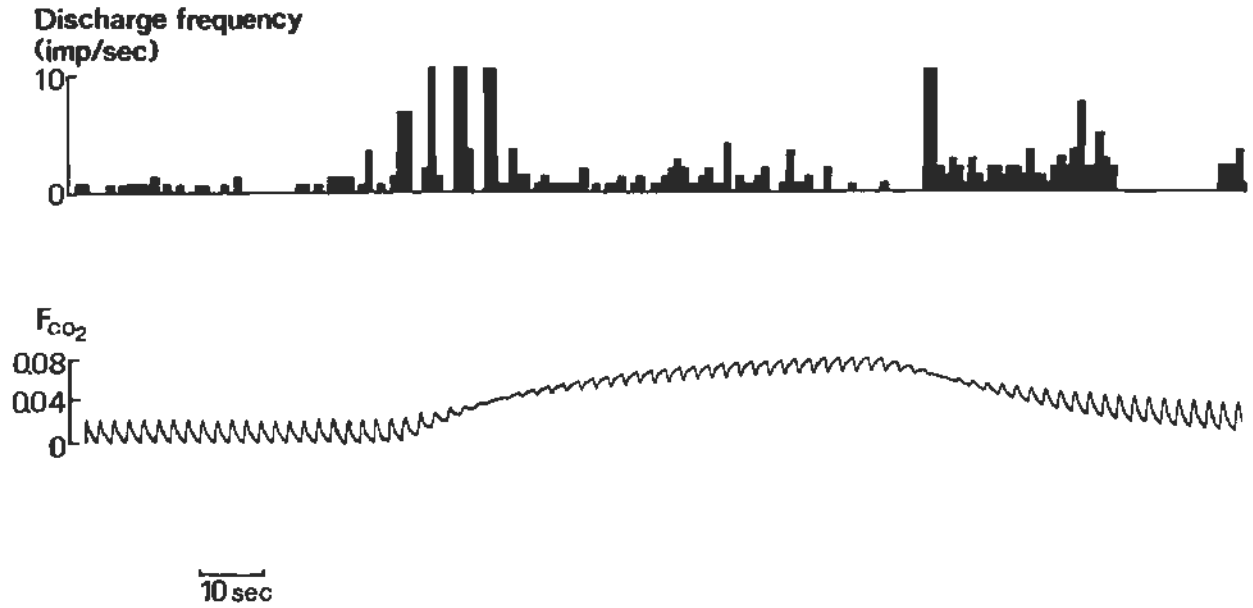

Fig. 3. Fast adapting response of a bronchial vagal C-fibre to a sustained increase in inspired CO_2 concentration (cats). From top to bottom are the spontaneous discharge frequency of a single vagal C-fibre and expired CO_2 concentration measured with a rapid analyser [9]. F_{CO_2}; fractional concentration of CO_2.

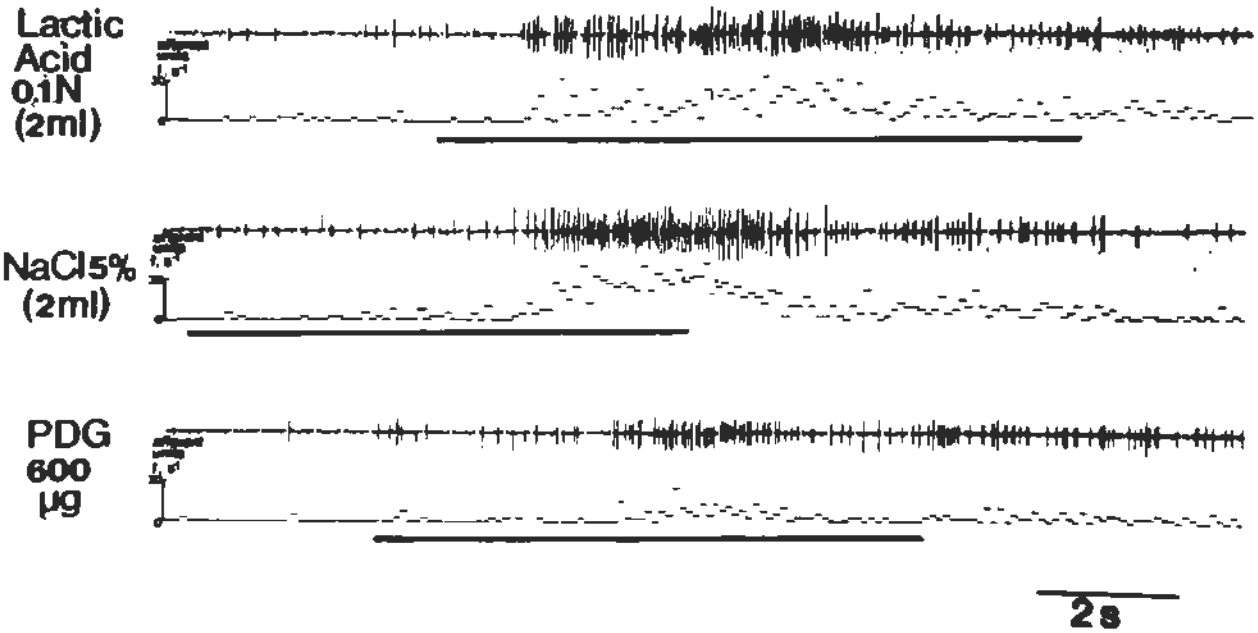

Fig. 4. Response of group IV phrenic sensory fibres to retrograde injection of lactic acid, hypertonic NaCl solution or phenyldiguanide (PGD) into the common carotid artery in cats. In each panel are shown: a raw multiunit recording of thin afferent fibres, identified from measurement of conduction velocity, and impulse rate of selected units. Horizontal bars indicate the duration of drug injection [simplified from 24].

Influence of tonic background sensory activity. The tonic background from arterial chemoreceptors certainly exerts a facilitatory influence on the respiratory centre activity [7, 50]. However, there are contradictory results concerning the ventilatory effects of background activity in lung irritant receptors and bronchopulmonary vagal C-fibres. All data have been obtained in studies using differential vagal cold block or local nerve anaesthesia during eupnoea. Some authors attribute the control of ventilatory timing to both phasic and tonic vagal activities [5, 40]. Others attribute the adjustment of spontaneous respiratory frequency only to phasic, volume-related vagal information [16, 23]. Then, the tonic vagal sensory background seems to exert only an inhibitory influence on the recruitment of inspiratory neurones during eupnoea, as revealed by an increase in integrated phrenic activity during selective pro-

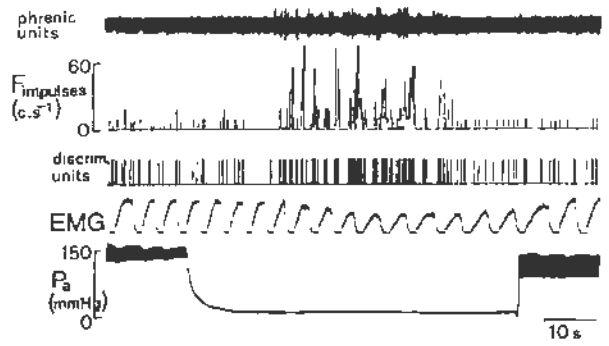

Fig. 5. Activation of group IV phrenic sensory fibres during diaphragmatic ischemia produced by aortic occlusion in cats. From top to bottom are shown: raw recording of phrenic sensory fibres; impulse rate of discriminated units; integrated diaphragmatic EMG and arterial blood pressure (P_a) recorded from a femoral artery [15].

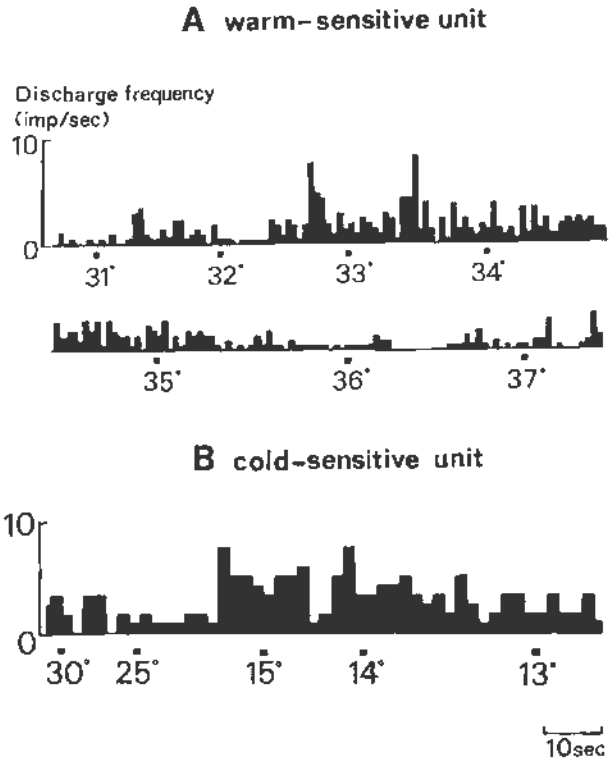

Fig. 6. Examples of warm and cold-sensitive sensory units recorded in the vagus nerve or in the superior laryngeal nerve in cats. Warm-sensitive units are identified as coming from the trachea and display an optimal spontaneous firing rate within the normal temperature range measured in the cervical trachea [9]. Cold-sensitive units are recorded in the superior laryngeal nerve, supplying the larynx and the upper part of the cervical trachea; their spontaneous discharge increases when the inspired temperature falls below 20–22 °C [redrawn from 27]. In each panel are shown the discharge frequency of single units, recorded using glass microelectrodes from the nodose vagal gauglion, and the temperature of inspired gas.

caine block of conduction in thin vagal fibres (fig. 7) [23].

More convergent results are obtained on the effect of background tonic vagal activity on the broncho-

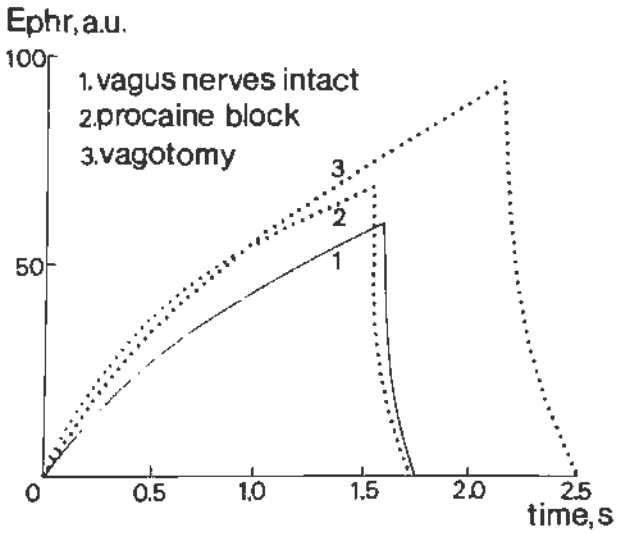

Fig. 7. Integrated motor phrenic activity (Ephr) measured during spontaneous breathing: 1) in intact cats; 2) after procaine block of conduction in thin vagal fibres; 3) after bivagotomy. This allows successive suppression of background tonic sensory vagal pathway (curve 2), then phasic volume-related vagal information (curve 3). Each curve is an average of 100 breaths sampled in the same animal [23].

motor tone. Thus, the differential cold block of vagal conduction in large myelinated vagal fibres at 7–8 °C unmasks excitatory effects on airway smooth muscle, exerted by thin vagal afferents [41]. On the other hand, procaine block of conduction in thin vagal fibres abolishes the bronchoconstrictor vagal tone in cats (fig. 8) [17]. In addition, selective sensory vagotomy at the level of nodose ganglion lowers the value of total lung resistance (fig. 8) [17] and the section of pulmonary vagal branches reduces or abolishes the tonic discharge of preganglionic vagal motoneurones [3]. It seems likely that vagal bronchoconstrictor tone depends on facilitatory influences carried by afferent vagal C-fibres. As Coleridge [4] said 'the afferent vagal C-fibres supplying the lower airways can no longer be regarded simply as a high-threshold afferent system whose influence is exerted only in situations that threaten well-being'.

The results are less obvious concerning the ventilatory influence of background tonic sensory activity in somatic and mostly muscle nerves. All studies have been performed in anaesthetized animals, thus excluding the possible influence of skeletal muscle afferents activated during the postural muscle tone. Controversial data have been reported concerning changes in spontaneous breathing pattern following spinal cord section or selective thoracic dorsal rhizotomy, which suppresses chest wall but not phrenic afferents [14, 25, 48]. Moreover, these effects of background somatosensory pathway depend on the type of anaesthesia and mostly on the integrity of vagal afferent information [25].

The importance of such interactions between visceral and somatic inputs will be discussed below. In fact, we may suppose that group III and IV muscle

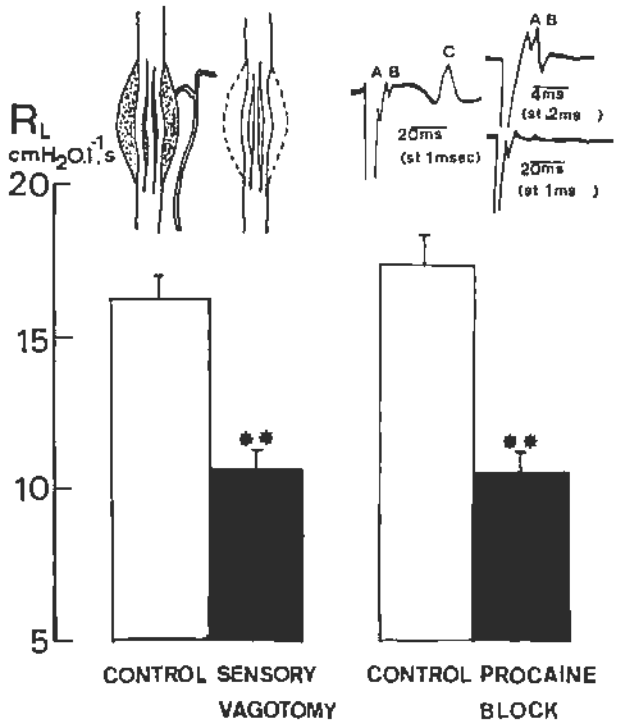

Fig. 8. Decrease in total lung resistance (R_L) measured in cats breathing spontaneously after selective sensory vagotomy at the level of vagal nodose ganglion (left part) or procaine block of conduction in thin vagal fibres (right part). The selectivity of blockade is assessed by the suppression of the compound C wave (unmyelinated fibres) with persistency of the A and B (myelinated fibres) in the vagal evoked potentials [redrawn from 17].

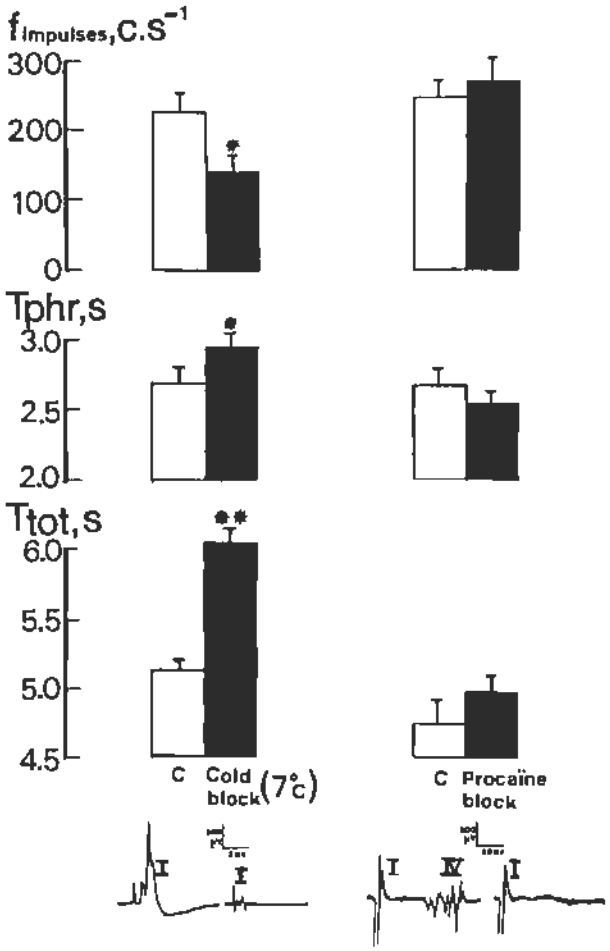

Fig. 9. Effects of selective block of conduction in group I (large myelinated) phrenic fibres (cold block at 7 °C) or in group IV (thin unmyelinated) phrenic fibres (procaine block) on the contralateral motor phrenic discharge (cats). Blockade of thin sensory phrenic fibres has no effect on the impulse rate of phrenic motoneurones (f impulses), the duration of phrenic discharge (Tphr) and total breath duration (Ttot) but the blockade of large myelinated phrenic afferents produces significant changes in breathing pattern [redrawn from 24].

afferents are not active during isotonic spontaneous contractions of respiratory muscles. This is confirmed by the observation that procaine block of thin afferent phrenic fibres does not alter the breathing pattern but cold block of large phrenic fibres (*i.e.* mostly Golgi tendon organ afferents) lowers the respiratory frequency and the firing rate of phrenic motoneurones (fig. 9) [24].

Reflex effects of enhanced tonic sensory activity. Reflex ventilatory and bronchomotor effects of stimulation of thin sensory fibres are well documented in particular circumstances. Stimulation of arterial chemoreceptors by hypoxaemia or hypercapnia increases both tidal volume and mean inspiratory flow [7, 37] and also the airway smooth muscle tone [22], due to reflex activation of vagal motor fibres (fig. 10) [3]. Stimulation of thin vagal afferents, particularly bronchopulmonary C-fibres, increases the airway tone [4, 22] as well as the secretion by tracheal submucosal glands [45]. These effects participate in the airway defence reaction but are also found when alveolar CO_2 concentration increases in cats and dogs [8, 22].

The results concerning the associated ventilatory response are somewhat contradictory. Thus, there is controversy whether lung irritant receptors, which constitute only 4% of vagal lung sensory fibres, participate in the rapid shallow breathing response

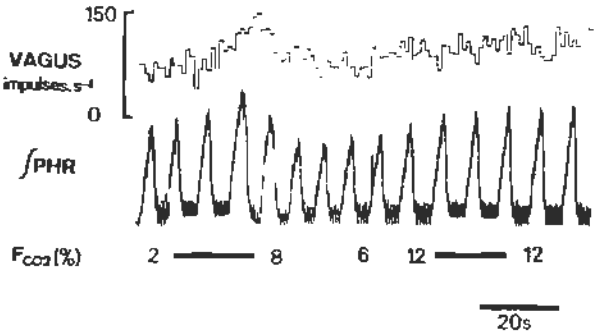

Fig. 10. Reflex increase in activity of vagal motoneurones recorded near the pulmonary hilum during inhalation of hypercapnic gas mixture (cats under artificial ventilation). Two short periods of asphyxia are also produced by stopping the ventilatory pump (horizontal bars). Lung denervation is performed by sectioning all pulmonary vagal branches, thus the observed effects result only from the stimulation of arterial and/or central chemoreceptors. From top to bottom: impulse rate of motor vagal fibres (multiunits recording); integrated motor phrenic activity and CO_2 concentration in expired gas (F_{CO_2}) (unpublished observation).

after administration of histamine or antigen [36], because this response survives selective cold blockade of all myelinated vagal fibres and then becomes even more pronounced [13]. By contrast, the stimulation of vagal C-fibres by chemicals such as phenyldiguanide or capsaicine, or by substances released during the inflammatory reaction, produces an initial apnoea followed by rapid shallow breathing [4]. The stimulation of lung vagal C-fibres during inhalation of CO_2-enriched gas mixture may explain the ventilatory response described in birds [2] and mammals [1] in experimental circumstances of cardiopulmonary bypass. This response is abolished by bivagotomy and has been attributed to the stimulation of vagal C-fibres [42].

Recent observations also show that the stimulation of thin vagal afferent fibres by deflation of the lungs, acetylcholine- or histamine-induced bronchospasm, or injection of phenyldiguanide produces tonic contraction of inspiratory muscles and tonic phrenic discharge [26]. Stimulation of group III and IV fibres from hindlimb muscles [34] or diaphragm [24] also induces tachypnoea with an inconstant increase in tidal volume. However, the activation of thin muscle fibres reflexly decreases total lung resistance to airway smooth muscle [30, 33], the opposite effect of activation of thin vagal fibres and arterial chemoreceptors.

Central projections and interactions

Vagal and arterial chemoreceptor afferents project directly onto the nucleus tractus solitarius (NTS), which is closely connected to dorsal respiratory group neurones and preganglionic vagal motoneurones [44]. However, thin afferent fibres from skeletal and respiratory muscles ascend via the lateral funiculus of the spinal cord and project onto the medulla [47], the cerebellar cortex [11, 39] and also the sensorimotor cortex [6]. Recent data also show that the central integration of muscle afferents during static muscular contraction needs the integrity of the subthalamic locomotor region [51]. In the medulla muscle fibre afferents seem to project onto the same respiratory neurones which integrate vagal and chemoreceptor inputs [47]. This is also supported by the observation that the ventilatory effects of spinal cord section depend on the integrity of vagal afferents [25] and that the ventilatory response to vagal or chemostimulation is reduced or even abolished when respiratory muscle afferents are strongly stimulated (fig. 11) [20, 21]. Such interactions between somatic or visceral information may involve thin muscle fibre afferents, which are stimulated in these experimental conditions, producing quasi-isometric contractions of respiratory muscles against high external loads. Moreover, the selective activation of muscle proprioceptors including muscle spindles does not modify the ventilatory response to hypercapnia [18].

A viscero-somatic reflex loop also exists between pulmonary vagal C-fibres and the alpha and fusimotor drives to the skeletal muscles [38, 46]. The functional significance of this complex reflex is not clear as it needs the integrity of the central structures lying just below the cerebral cortex [28]. PAINTAL [38] has proposed that the stimulation of pulmonary C-fibres by increased pulmonary arterial pressure and/or CO_2 flux during supramaximal exercise exerts

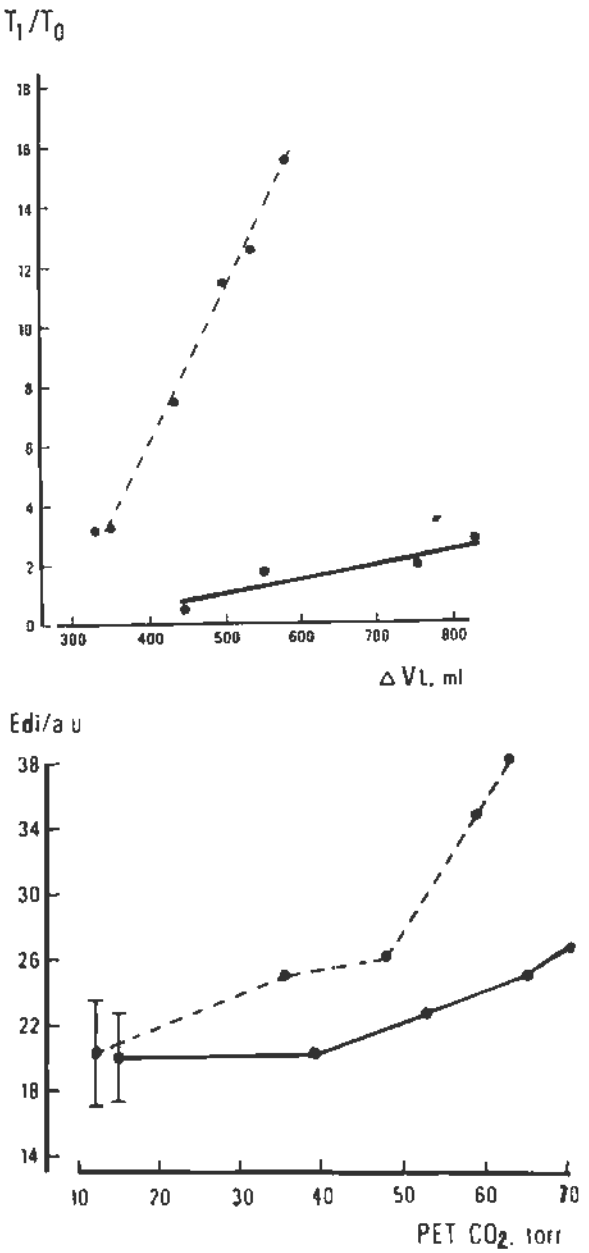

Fig. 11. Interactions between somatic afferents from respiratory muscles and vagal or chemosensitive afferents in dogs under cardiopulmonary bypass breathing against high expiratory threshold loads (ETL). In the upper diagram, the apnoeic response to lung hyperinflation, expressed by plotting the inhibitory ratio ($T_I/T0$) against changes in lung volume (ΔVt) is abolished after 10 min of ETL breathing from [20]. In the lower diagram, increase in integrated diaphragmatic EMG (Edi) due to CO_2 rebreathing disappeared during ETL breathing [21] (Dashed line: control; Solid line: ETL breathing)

an inhibitory influence on the cortico-spinal tracts which control the strength of muscular contraction.

The question remains, whether the respiratory centres differentiate phasic or tonic inputs from visceral and somatic afferents. One hypothesis is that central neurones integrate particular patterns of afferent discharge, *i.e.* tonic low frequency discharge of rapidly adapting receptors or phasic high frequency firing rate of slowly adapting mechanoreceptors. Another hypothesis is that central structures may detect different neuromediators released by sensory fibres. Evidence for the latter is that substance P is released by unmyelinated sensory fibres but not by large myelinated fibres [32].

In conclusion, both ventilatory control of respiratory muscles and vagal motor drive to the lungs depends on the central integration of both visceral and somatic inputs. This results from the summation of phasic and tonic sensory pathways and some of these peripheral inputs provide antagonistic influences.

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RÉSUMÉ: Tant les centres respiratoires que les motoneurones vagaux préganglionnaires qui contrôlent respectivement les muscles respiratoires (striés) et les muscles des voies aériennes (lisses) reçoivent des informations sur les poumons, la circulation et les muscles squelettiques et respiratoires. Chacune de ces voies nerveuses a deux composantes; l'une est phasique, liée aux rythmes biologiques et provient des mécanorécepteurs connectés aux grosses fibres myélinisées; l'autre est tonique avec des décharges à basse fréquence, correspondant à l'activité spontanée des récepteurs polymodaux connectés aux petites fibres sensitives, qui agissent principalement comme senseurs des changements dans la composition des liquides extracellulaires (pression partielle d'O_2 et/ou de CO_2, pH, libération d'agents algésiques, etc...). Certains d'entre eux détectent également des changements mécaniques de grande amplitude ou de température locale. L'influence de l'activité tonique sensitive basale est bien connue chez l'animal en ce qui concerne le rôle joué par les chémorécepteurs artériels dans le contrôle de la ventilation et par les petites fibres vagales afférentes d'origine pulmonaire (fibre C et récepteurs à l'irritation) dans la facilitation d'origine réflexe du tonus vagal bronchoconstricteur. De plus la stimulation de ces petites fibres sensitives dans certaines circonstances est responsable d'une hyperventilation (chémorécepteurs artériels et afférences musculaires), d'un augmentation du tonus des voies aériennes (chémorécepteurs et principalement fines fibres vagales afférentes) ou d'une bronchodilatation (afférences musculaires). Ces influx périphériques projettent sur diverses structures centrales ainsi que sur les neurones du tronc cérébral qui intègrent simultanément l'information provenant des chémorécepteurs, du nerf vague et des muscles. Il en résulte des interactions complexes entre les différentes voies sensitives.