(Reprinted from Nature, Vol. 261, No. 5558, pp. 327–328, May 27, 1976)

Are reptilian pulmonary receptors mechano- or chemosensitive?

BOTH birds and mammals possess pulmonary receptors which increase discharge in phase with each inspiration[1–5]. In mammals the adequate stimulus of these receptors seems to be the total transpulmonary pressure which varies with both inflation volume and rate and sign of the volume change[6]. The lungs of birds, however, consist of a complex system of air tubes[7,8] and unlike those of mammals are relatively inexpansible. Many avian pulmonary receptors have no mechanosensitivity, the adequate stimulus being the changes in airway CO_2 throughout the breathing cycle[9–12]. So rate and degree of inflation of the respiratory system is signalled by different sensory modalities in birds and mammals. The demonstration that mammalian pulmonary receptors are partially inhibited by high alveolar CO_2 (refs 13–15) and that some avian receptors may be mechanosensitive[16,17] suggests that in the more phylogenetically ancient vertebrate classes it might be possible to locate a pulmonary receptor which has distinct mechano- and chemosensitive properties, thereby allowing speculation on the evolution of the avian and mammalian receptor types.

We report here the responses of turtle (*Chrysemys picta*) pulmonary receptors to both mechanical and chemical stimuli. This animal was chosen because reptiles represent the evolutionary stem group for both birds and mammals. Although many reptiles or even the more phylogenetically primitive amphibia might have served, our choice was governed by two facts. First, because of their diving habits, turtles experience alveolar CO_2 levels in the mammalian range[18]; second, in possessing a chambered, saccular lung that is constrained in its expansion by the exoskeleton[19], turtles show a superficial resemblance to birds.

Turtles (600–1,200 g) were single-pithed and tidally ventilated with a constant volume, positive pressure, respiration pump. Single and multi-fibre nerve activity, in phase with artificial ventilation, was recorded in vagal slips using bipolar silver electrodes. Neural activity was amplified, visually displayed on an oscilloscope and audibly monitored. Tracheal air flow and intratracheal pressure (ITP), generated by the ventilator, were recorded with a pneumotachograph and pressure transducer respectively. All variables were stored on magnetic tape for later analysis on a Digital PDP Lab 8e mini-computer using conventional software.

Table 1 Reduction in discharge rate of pulmonary receptors following the introduction of 10% CO_2 to the ventilating gas

Fibre no.	Peak inflation pressure (cm H_2O)	% Reduction in discharge rate associated with		
		End inflation	End deflation	One breath
1	—	33	100	66
2	5.5	46	100	65
3	7.0	27	60	26
4	4.5	63	100	68
5	6.5	100	100	100
6	6.5	100	100	100
7	9.0	37	7	27
8	9.0	56	73	52
9	9.0	53	80	45
10	7.5	36	—*	47
11	7.5	50	—*	38
12	5.0	33	30	44
13	5.0	55	60	62
14	8.0	100	—*	100

The difference between the discharge rates, when ventilating with air and 10% CO_2, is expressed as a percentage of the discharge rate with air.
*Discharge rates fell to zero while animals were breathing room air.

Receptors with discharge modulated by artificial ventilation with air were of the slowly adapting low threshold type. Many units were active during the deflation phase when ITP approached that of the atmosphere (Fig. 1*b*), the average discharge frequency being 7.1 ± 1.4 impulses s^{-1} at end deflation. Discharge increased with lung inflation and reached its maximum at peak ITP but none of the receptors seemed to respond to the rate of change in pressure either on inflation or deflation. Discharge frequency increased with inflation by 2.09 ± 0.32 impulses per s per cm H_2O increase in ITP, and discharge was maintained when artificial ventilation was held at either end inflation or end deflation. In response to maintained inflation, only 3 of the 14 single fibres investigated showed marked adaptation which was still less than 25% when calculated from index 1 of Davis *et al.*[5]. After removal of the sternum, 6 of the 14 units were located by punctate stimulation to the major septa which divide the lung into 8–10 chambers (Fig. 1*a*) and receptor discharge patterns were shown to be unaffected by pulmonary artery occlusion.

Addition of 10% CO_2 to the ventilating gas caused a rise in threshold and a decrease in sensitivity of the receptors for any given pressure change compared with air inflation (Table 1). The increase in discharge rate on inflation was

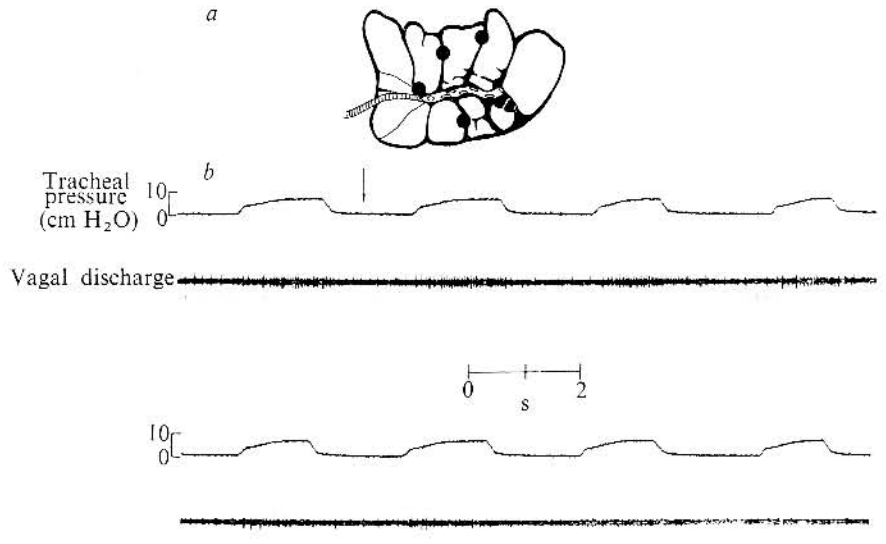

Fig. 1 *a*, Schematic diagram of the right lung of *Chrysemys picta* showing approximate site (●) of receptors located by punctate stimulation. *b*, Effect of 10% CO_2 on the discharge of a turtle's pulmonary receptors; continuous recording of intratracheal pressure (upper) and pulmonary receptor discharge (lower), 10% CO_2 introduced into the ventilating gas mixture at the arrow.

reduced to almost half (56%) the air inflation value and 3 units remained silent throughout inflation. For 5 units discharge ceased during lung deflation whereas, on the average, discharge rate during deflation was reduced by 74%. The decrease in activity began during the first inflation following a step change in CO_2 content of the ventilating gas and continued to fall over several cycles of ventilation (Fig. 1*b*) (using tidal ventilation, 2–3 breaths were required before the expired gas composition equalled that of the inspired gas). High CO_2 did not seem to affect the adaptation rate to maintained inflation. The effects of ventilating with 5% CO_2 in air were about half as pronounced as the effects of 10% CO_2. Since some units were completely silenced by ventilation with air containing 10% CO_2, any changes in lung compliance induced by the presence of CO_2 cannot explain this reduction in receptor discharge. Lung compliance, however, was decreased in four turtles whereas three showed no change in compliance when 10% CO_2 was added to the ventilating gas. In only one case did the fall in compliance (35%) match the fall in the maximum discharge frequency, at peak ITP, of the receptor being recorded.

This work has shown that although all pulmonary receptors we have monitored are mechanosensitive, some are extremely sensitive to CO_2, being completely inhibited by pulmonary concentrations of 10%. This sensitivity to CO_2 resembles that shown by avian pulmonary chemoreceptors. On the other hand, those receptors exhibiting only slight sensitivity to CO_2 have responses similar to those of mammalian pulmonary mechanoreceptors. For these reasons, the reptilian receptor type may be viewed as the functional precursor of those in higher vertebrates. It also suggests that the reflex respiratory responses to intrapulmonary CO_2 shown by reptiles[20], birds[21] and mammals[22] are phylogenetically ancient.

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Received February 2; accepted March 30, 1976.

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Printed in Great Britain by Henry Ling Ltd., at the Dorset Press, Dorchester, Dorset

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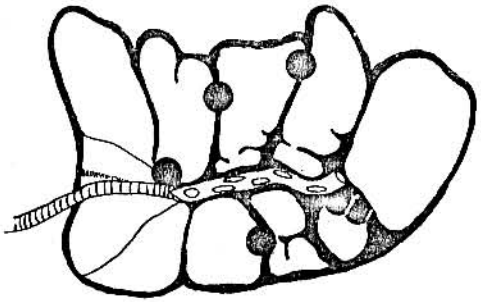

b.