The immediate effects of deafferentation of the lungs on heart and breathing frequencies in ducks

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Three unilaterally vagotomised ducks were subjected to 'instantaneous' deafferentation of the remaining innervated lung by pulling snares through the vagal branches while recording heart and breathing frequencies. Deafferentation caused immediate cardiac and respiratory depression but the effect on tidal volume was inconsistent. Five minutes after deafferentation, breathing frequency had fallen from 25–30 to 6–8 breaths min^{-1}. Arterial pH, P_{O_2}, and P_{CO_2} were 7.49, 12, and 4.07 kPa before deafferentation and 7.05, 9.7, and 6.8 kPa, respectively, 10 min after deafferentation. These results confirm the idea that some form of feedback from the lungs is essential for generation of stable respiratory rhythms in birds and further indicate that artificial ventilation is advisable, for some hours, after deafferentation.

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Trois canards ont subi une vagotomie unilatérale pour être ensuite soumis à un arrêt subit de l'innervation (par étranglement des branches du nerf vague au moyen d'un lacet) dans le poumon encore innervé; les fréquences des battements cardiaques et des respirations ont été enregistrées tout au long de l'expérience. L'arrêt total de l'innervation entraîne immédiatement des ralentissements cardiaque et respiratoire, mais l'effet sur le volume de l'écoulement n'est pas toujours le même. Dans les 5 min qui suivent l'arrêt de l'innervation, la fréquence respiratoire passe de 25–30 à 6–8 respirations à la minute. Avant l'étranglement des branches du vague, le pH artériel (pH) est de 7.49 et les partielles pressions artérielles sont de 12 (P_{O_2}) et 4.07 (P_{CO_2}) kPa; 10 min après l'arrêt de l'innervation, ces valeurs deviennent respectivement 7.05, 9.7 et 6.8 kPa. Il semble donc qu'une certain rétroaction venant des poumons soit essentielle à la stabilité des rythmes respiratoires chez les oiseaux; en outre, il est préférable de procéder à une ventilation artificielle pour quelques heures après l'arrêt de l'innervation.

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Introduction

In a recent series of experiments we deafferentated the lungs of ducks by cutting the vagal branches to the lung in order to study the long-term effects of pulmonary deafferentation on the cardiovascular and respiratory responses to diving (Bamford and Jones 1976). We noticed that nerve section caused pronounced cardiovascular as well as respiratory effects in open-chest animals. How-

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ever, when the wound was closed and the animal resumed an upright posture, breathing at least appeared more normal except that animals breathed through their mouths. Since considerable research is now being performed on the role and nature of pulmonary vagal afferents in birds (see Respir. Physiol. 22 (1974)), we felt it would be worthwhile to elucidate the cardiovascular and respiratory effects of pulmonary nerve section in more or less intact ducks, thereby eliminating side effects due to working with open-chest animals.

Methods

The experiments were done on three ducks (*Anas platyrhynchos* var.) of about 5 weeks of age and an average weight of 750 g. The animals were anaesthetized with urethane (1 g/kg intramuscularly (i.m.)) and after right midcervical vagotomy the thorax was opened by a midline sternal incision and the branches of the pulmonary vagus to the left lung exposed. These were dissected free from surrounding connective tissue and a loop of thin stainless-steel wire secured around each nerve. At the same time several loops were placed around bundles of connective tissue coursing over the lung surface. The loops around the nerves were connected to white thread and those around connective tissue bundles to black thread. The animal was then closed with the threads protruding through the sternum. All white and all black threads were knotted together. After recovering to a light plane of anaesthesia, the animals were placed upright. The electrocardiogram (EKG) was recorded using bipolar copper wire electrodes, amplified conventionally, and heart rate was measured using an instantaneous rate meter. Arterial blood pressure was measured via a cannula of PE 160 polyethylene tubing in a femoral artery using a Hewlett–Packard 267 pressure transducer and Hewlett–Packard 350–1100 carrier preamplifier. Blood samples were withdrawan from the arterial cannula, over one or two breathing cycles, for analysis of blood gas tensions and arterial pH using a Radiometer BMS 3 blood gas analyser. Tidal volume was obtained by integrating the tracheal air flow measured by a pneumotachograph and Hewlett–Packard 270 differential pressure transducer. The integrator (Hewlett–Packard 350–3700) was calibrated by passing known volumes of gas through the pneumotachograph. Body temperature was monitored with a rectal thermistor probe and maintained at 41°C, with a maximum variation of ±1°C, by an infrared light situated over the animal.

The effects of pulling all the black threads served as a control for the effects of instantaneous lung deafferentation achieved by pulling the white threads. To confirm that the cardiac vagal innervation was intact the left vagus was sectioned in the neck, under local anaesthesia, and the heart-rate response to stimulation of the cut peripheral end of the left vagus (2.5–5 V, 50 Hz, 10-ms duration) was recorded.

Results

Pulling the black cotton, connected to snares around 5 or 6 strands of connective tissue, served as a 'control' for the physical disturbance of the instantaneous deafferentation procedure. This 'control' procedure caused no change in heart or breathing frequency although mean blood pressure fell, on average, by 40% after 5 s. In one animal tidal volume increased by 40% some 10 s after this procedure. In all cases these variables had returned to their initial values within 1 min of the 'control' procedure. However, instantaneous deafferentation caused marked cardiac and respiratory depression; both heart rate and breathing frequency on average fell to 50% of their initial values some 5 s after deafferentation (Fig. 1*a*). On average tidal volume increased by 25% in this period although in one duck tidal volume did not change. In all animals the breathing pattern changed; the inspiratory period was reduced greatly and the expiratory phase prolonged (Fig. 1). As was the case with the 'control,' mean blood pressure fell, on average, by 40% but, in spite of a depressed heart rate in two of the three ducks, returned to the initial level within 1 min of deafferentation. In the period from 1 to 5 min after deafferentation breathing frequency continued to decline falling to a range of 6–8 breaths min^{-1} (initial range was 25–30 breaths min^{-1}) (Fig. 1*b*). On the other hand heart rate increased in all animals and was above the initial rate 5 min after deafferentation, although, despite the loss of pulmonary afferents, prominent tachycardia accompanied each breathing movement (Fig. 1b and 1c). Unfortunately one animal died 5.5 min after deafferentation but arterial blood samples were obtained from the survivors 10 min after deafferentation for comparison with samples taken before. The mean initial values for arterial pH, P_{O_2}, and P_{CO_2} were 7.49, 12, and 4.07 kPa respectively whereas 10 min post deafferentation the corresponding values were 7.05, 9.7, and 6.8 kPa. At this time tidal volume had increased about 50% but was obviously insufficient to maintain normal blood gas tensions in the face of a 60% fall in breathing frequency. The tachycardia and hypopnoea persisted throughout the rest of the period of observation (Fig. 1*c*). One hour after deafferentation, the intact vagus, which had been previously exposed in the neck, was sectioned and the distal cut end stimulated (2.5–5 V, 50 Hz, 10-ms duration). Since during stimulation both heart rate and blood pressure fell, the cardiac branches of the vagus were obviously unaffected by the deafferentation procedure.

Discussion

The immediate effects of instantaneous lung deafferentation in ducks are bradycardia and respiratory depression. The effects of instantaneous deafferentation are similar to those observed when nerves were sectioned singly (Bamford and Jones 1976), and so are unlikely to be an artefact due to massive centripetal nervous activity on deafferentation. The effects on breathing, namely a reduction in frequency with long expiratory pauses, are similar to those caused by bilateral cervical vagotomy, and as has been already shown for the

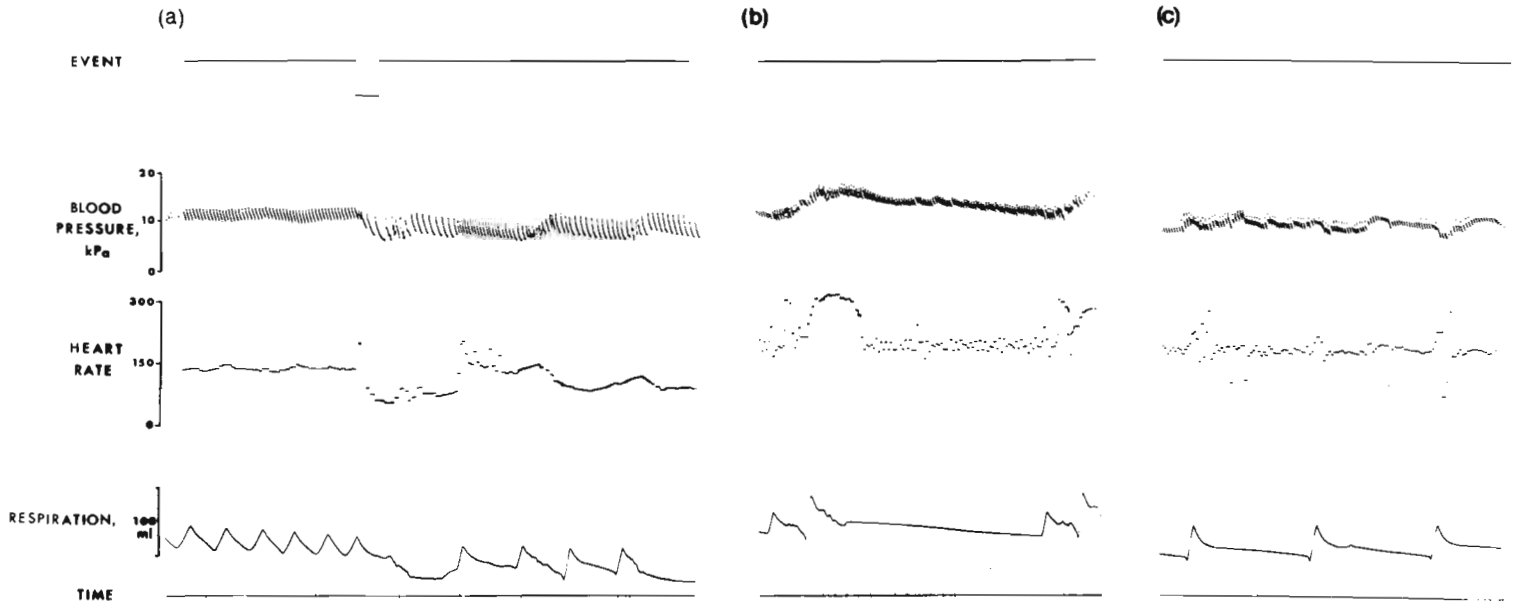

FIG. 1. (*a*) The effect on arterial blood pressure, heart rate, and breathing of instantaneous lung deafferentation performed at the deflection on the event channel. (*b*) Five minutes after deafferentation. (*c*) Thirty minutes after deafferentation. Traces from top to bottom: 1, event marker; 2, arterial blood pressure (kilopascals); 3, heart rate (beats per minute); 4, breathing, tidal volume (millilitres); 5, time (seconds).

chicken (Fedde *et al.* 1963), pulmonary deafferentation rather than deafferentation of other reflexogenic zones is the cause of the respiratory response to vagotomy. After denervation the duck became asphyxic, so the increase in tidal volume was insufficient to compensate for the reduction in breathing frequency and indicated the necessity for either artificial ventilation or stimulation of breathing rate (i.e. by electrical foot shock) to maintain the animal immediately postdeafferentation.

The importance of pulmonary afferent information in maintenance of heart rate was apparently confirmed by the bradycardia which accompanied deafferentation. That the bradycardia was not caused by the increased output of carotid chemoreceptors in response to the advancing asphyxia was shown by the fact that it was not maintained. Fluctuations in heart rate and blood pressure still accompanied the breathing movements after denervation. The integrity of the arterial barostatic reflex was indicated by the fact that mean arterial pressure returned to predenervation levels within 1 min after denervation. Stroke volume of ducks seems to be little affected by numerous manipulations (Folkow *et al.* 1967; Jones and Holeton 1972) and if this was also the case after pulmonary deafferentation then a substantial increase in total peripheral resistance (50 to 100%) is indicated, 1 min after deafferentation.

Deafferentation caused both a change in the pattern of breathing and a marked decline in breathing rate, which confirms the suggestion of Kunz and Miller (1974) that some form of feedback from the lungs is normally involved in generation of stable respiratory patterns in birds and indicates that input from carotid body chemoreceptors alone is unable to maintain normal respiratory rhythmicity when pulmonary feedback is suddenly abolished. However, the long-term effects of pulmonary deafferentation on breathing are much less drastic and so the respiratory centre is clearly able to compensate for this loss of input (Bamford and Jones 1976).

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