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Nasal vasodilation induced by electrical stimulation of the vagus nerve

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SUMMARY

Nasal obstruction has been previously linked with cardiac and pulmonary disorders as a causative factor. The present study demonstrates that nasal vasodilation may be induced by electrical stimulation of the cut central end of the vagus. This suggests that cardiac and pulmonary disorders may cause nasal obstruction via a vagal reflex.

INTRODUCTION

Nasal obstruction has been previously linked with cardiac symptoms (Cvetnić et al., 1980) and there is some evidence that nasal obstruction may produce cardiopulmonary changes (Jackson, 1976; Ogura et al., 1971). In these studies it was suggested that nasal obstruction was a causative factor in cardiopulmonary disease either inducing hypoxia or initiating a trigeminal-vagal, nasal reflex. One factor which has not been previously considered is that cardiopulmonary disorders may induce nasal obstruction by a vagal reflex. The purpose of the present study is to demonstrate that nasal obstruction may be induced by a vagal reflex.

METHODS

Experiments were performed on 13 healthy large white pigs (12 female, 1 male; body weights between 30–70 kg). The pigs were anaestetised with "Halothane" through a face mask followed by a slow intravenous injection of sodium pentobarbitone (25 mg/kg). The trachea was cannulated and the pigs were ventilated artificially. Arterial blood pressure was recorded by means of a cannula inserted into the femoral artery and connected to a pressure transducer. The pigs were covered with an electric blanket and the body temperature was maintained at 39°C. Nasal vasomotor response were recorded from the sealed nasal cavity by means of a plethysmographic technique which mainly records changes in the volume of the venous erectile tissue in the nasal mucosa (Eccles et al., 1974, 1981). The vagus nerves were exposed by an incision in the neck and carefully separated from the cervical sympathetic nerves. The intact or the cut central end of the vagus was mounted on silver electrodes for electrical stimulation with the parameters set at a supramaximal voltage, pulse duration 1 m sec and at a frequency between 1–50 Hz for 10 sec trains.

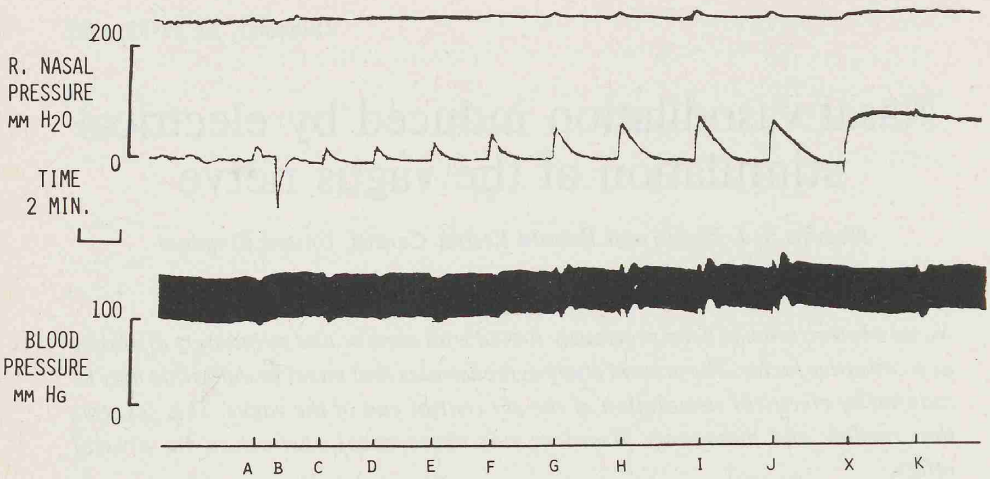

Figure 1. Nasal vasomotor responses induced by electrical stimulation of the vagus nerve, and cervical sympathetic nerve (parameters, 10 sec. train, 1m sec. pulse, supramaximal voltage). Vagal stimulation A.C.D.E. 1Hz, F. 5Hz, G. 10Hz, H. 15Hz, I. 25Hz, J. 50Hz. Sympathetic stimulation B. 1Hz. The cervical sympathetic nerve was cut at X and subsequent vagal stimulation at K. 15Hz, did not induce vasodilation.

RESULTS

In nine out of thirteen pigs electrical stimulation of the intact or cut central end of the vagus nerve caused nasal vasodilation. Bilateral nasal vasodilation was normally induced by unilateral vagal stimulation with the largest response ipsilateral to the stimulation. Electrical stimulation of the cut peripheral end of the vagus caused changes in heart rate and blood pressure but had no effect on the nose. Nasal vasodilation was induced by vagal stimulation at frequencies as low as 1 Hz and reached a maximum at around 10 Hz as shown in Figure 1A, G.

Section of the cervical sympathetic nerve ipsilateral to the vagus in nine pigs caused nasal vasodilation (Figure 1.X) and abolished the vagally induced vasodilator response as shown in Figure 1.K.

DISCUSSION

The nasal vasodilation caused by electrical stimulation of the vagus nerve is probably caused by stimulation of sensory fibres which travel in the vagus towards the brain from structures in the thorax or abdomen. Nasal vasodilation was not observed on stimulation of the cut peripheral end of the vagus although changes in heart rate and arterial blood pressure were observed. It is proposed that electrical stimulation of the vagus causes a reflex inhibition in sympathetic tone to the blood vessels of the nose and thus causes vasodilation. The vasodilation probably occurs mainly in the venous erectile tissue of the nasal mucosa as the plethysmo-

graphic technique of recording nasal vasomotor responses is most sensitive to changes in volume in this vascular bed (Eccles, 1981).

Section of the cervical sympathetic nerve caused a persistent vasodilation explicable on the basis of an interruption of sympathetic vasoconstrictor tone. After sympathetic nerve section, the vagal reflex nasal vasodilation could not be elicited due to interruption of the efferent loop of the reflex arc.

The proposed vagal reflex is illustrated in Figure 2. Electrical stimulation of the cut central end of the vagus at (A) caused nasal vasodilation whereas stimulation of the cut peripheral end of the vagus at (B) only caused changes in heart rate and blood pressure with no effect on the nose. The nasal vasodilation elicited by stimulation of the cut central end of the vagus at (A) was abolished after section of the cervical sympathetic nerve at (C). Since the cervical sympathetic nerve conveys only vasoconstrictor fibres to the nasal mucosa the results indicate that there is a continuous sympathetic tone on the nasal blood vessels and this activity may be inhibited by afferent activity in the vagus nerve.

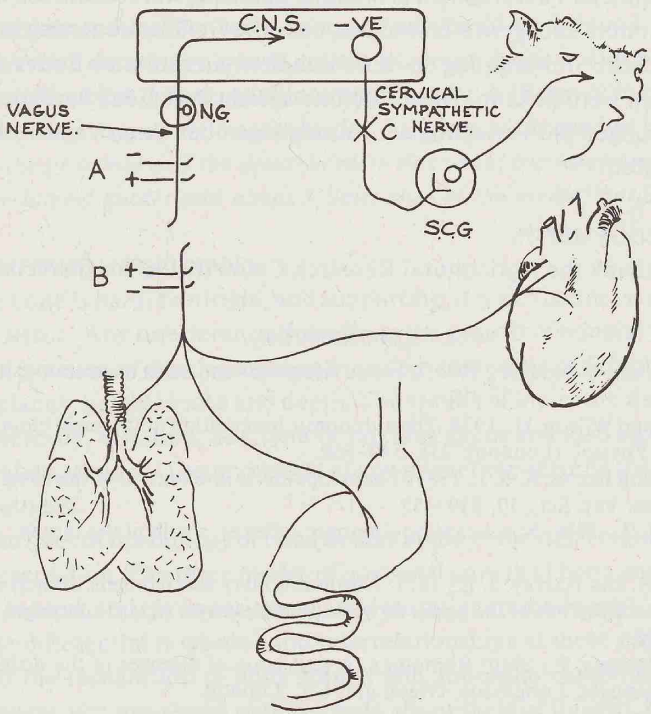

Figure 2. Proposed pathway for vagal reflex inducing nasal vasodilation. Electrical stimulation of the cut central end of the vagus nerve at (A) caused vasodilation by inhibition of sympathetic vasoconstrictor tone. Section of the cervical sympathetic nerve at (C) interrupted the reflex pathway to the nose. See text for further explanation, (N.G.) Nodose ganglion, (S.C.G.) Superior cervical ganglion.

The vagus provides sensory fibres to the heart, lungs and digestive tract and disorders in all of these areas have been previously linked with nasal obstruction, Cvetnić et al. (1980) stated that there was no reference in the cardiological literature to any connection between the nose and the heart and the authors speculated on the existence of a vagal pathway linking the two areas. Watson Williams (1910) stated that a chronic rhinitis may be caused by constipation or gastric disorders thus indicating a vagal link between the digestive tract and the nose.
In the present study it is not possible to determine which vagal sensory pathway is responsible for the nasal vasodilation and further studies are required to clarify the reflex pathway. The many clinical observations linking nasal obstruction with areas of the body innervated by the vagus nerve provide some evidence for a vagal-nasal reflex and the results of the present study support this proposal.

ZUSAMMENFASSUNG

Eine Verlegung der Nasengänge ist früher mit kardialen und pulmonalen Störungen als ursächliche Faktoren in Verbindung gebracht worden. In der vorliegenden Arbeit konnte nachgewiesen werden, daß eine Gefäßerweiterung in der Nase durch elektrische Stimulierung des durchschnittenen zentralen Endes des Vagus hervorgerufen werden kann. Dieser Befund spricht dafür, daß kardiale und pulmonale Störungen die Verlegung der Nasengänge über einen Vagusreflex verursachen könnten.

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