Rhinology, 24, 271-276, 1986

Nasal vasomotor responses in man to breath holding and hyperventilation recorded by means of intranasal balloons

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SUMMARY

Nasal vasomotor responses were recorded in conscious human subjects by means of water filled balloons. Hyperventilation caused an increase in intranasal balloon pressure associated with vasodilatation whereas breath holding caused a decrease associated with vasoconstriction. The amplitude of the nasal vasomotor response was influenced by the nasal cycle with the greatest response always observed on the congested or low airflow side of the nose. The results suggest that an elevated arterial level of carbon dioxide causes a pronounced vasoconstruction of the nasal blood vessels and that this response may be clinically relevant in controlling nasal bleeding.

Malm (1973, 1974) recorded vasomotor responses from the nasal passages of anaesthetized cats using water filled balloons to detect changes in the state of engorgement of the venous erectile tissue of the nasal mucosa. This simple but effective technique of recording from the nose has not previously been applied to man. In the present report we demonstrate that nasal balloons may be used in man and that the nasal vasomotor responses to hyperventilation and breath holding may be studied by means of this technique.

METHODS

Experiments were performed on 2 male subjects whilst resting supine on a couch. Water filled balloons connected to pressure transducers (Ormed type UP2) were inserted into both nasal passages and pressure responses from the balloons were displayed on a pen recorder (Ormed type M19). The latex rubber balloons were made from the distal 4 cm of the teat of a condom tied over the end of a plastic cannula 45 cm in length with an internal diameter of 0.15 cm. The portion of the cannula in the balloon had 6-8 holes punched along the last 2.5 cm of its length in order to allow free water movement between the balloon and cannula (Figure 1). The deflated balloon was covered with a local anaesthetic gel (2% lignocaine

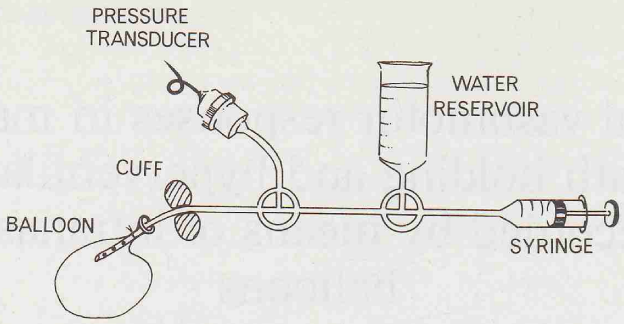

Figure 1. Nasal balloon connected to pressure transducer and water reservoir via three way taps.

BPC) and gently inserted into the nasal cavity to rest in the inferior meatus. The balloon was inflated with water by adjusting the three way taps so that water ran from the reservoir whilst the reservoir was topped up with water to maintain a final pressure of approximately 10 cm H_2O. The three way taps were then adjusted so that the balloon was connected to the pressure transducer in a closed system and any small pressure changes in the balloon were transmitted to the pressure transducer. The pressure transducers were calibrated by means of a Sloping Manometer (Airflow Developments Ltd. Type 504). Respiratory movements were recorded using a mercury in rubber strain gauge wrapped around the chest with a trace displayed on the pen recorder.

RESULTS

The technique proved to be relatively simple and safe. The only problems encountered were occasional sneezing on insertion of the balloons and a tendency for the balloon to move towards the nasopharynx. Sneezing was controlled by liberal application of local anaesthetic gel to the balloons and movement of the balloons towards the nasopharynx was prevented by small cuffs on the cannula which rested against the entrance to the nostril (Figure 1).

Before insertion of the balloons the subject was asked to assess the patency of each nasal passage in order to determine any asymmetry of airflow related to the nasal cycle. In general one nasal passage could readily be identified as having the dominant airflow and in a decongested state whilst the other had the lower airflow and was relatively more congested.

With the subjects resting supine with the balloons inserted in the nasal passages, control recordings were made for a period of approximately 20 min on 14 occasions in two subjects. On all occasions the nasal pressure traces were different for the two nasal passages as the congested nasal passage always exhibited small fluctuations in pressure (2–4 mm H_2O amplitude) with the same frequency as the

heart rate whereas the decongested nasal passage showed no such activity or much smaller pressure fluctuations. The asymmetry of the nasal pressure recordings is apparent in Figures 2 and 3 which illustrate fluctuations in pressure related to heart rate on the trace from the right nasal passage.

Voluntary breath holding or hyperventilation caused marked pressure changes in the nasal balloons. The effects of breath holding were studied on 31 occasions in two subjects and a typical trace of pressure recordings is shown in Figure 2. During the period of breath holding there was a gradual increase in balloon pressure of up to 2.0 cm H_2O followed by a fall in pressure of up to 5.0 cm H_2O. The pressure changes recorded from the two nasal passages in response to breath holding were always asymmetrical with the congested nasal passage exhibiting the greatest change in pressure and a similar but much smaller response from the decongested nasal passage.

The effects of hyperventilation were studied on 29 occasions in two subjects and a typical trace of pressure recordings is shown in Figure 3. After 10–15 sec of hyperventilation there was a gradual increase in pressure which continued for 10–15 sec after cessation of hyperventilation, up to a maximum increase of around 5 cm H_2O. The responses recorded from the two nasal passages were asymmetrical with a much larger increase in pressure in the congested nasal passage. At the peak of the pressure change there was an increase in the amplitude of the heart rate related oscillations in pressure apparent on the congested side of the nose, and similar oscillations, but of smaller amplitude, became apparent on the decongested side of the nose. This is in contrast to the pressure response to breath holding where any heart rate related oscillations were suppressed or abolished at the peak of the response.

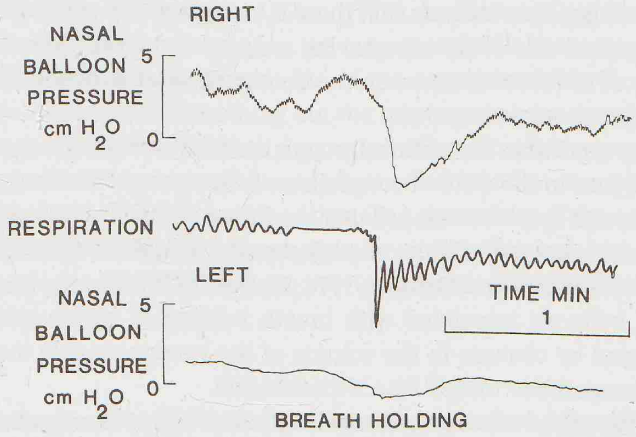

Figure 2. Changes in nasal balloon pressure caused by a period of breath holding. Note the presence of vasomotor oscillations on the right nasal trace and the pronounced fall in nasal balloon pressure caused by breath holding.

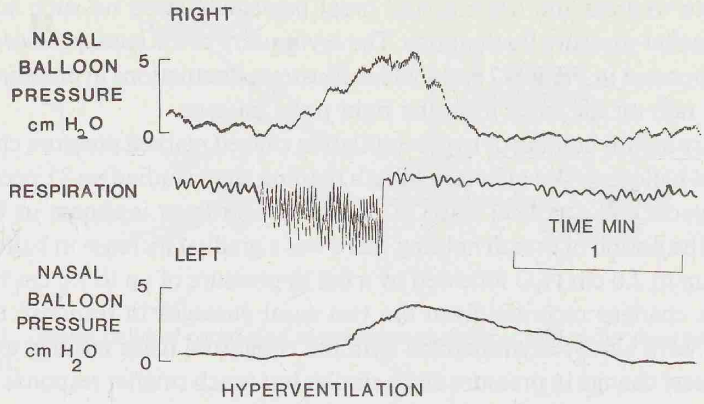

Figure 3. Changes in nasal balloon pressure caused by a period of hyperventilation. Note the vasomotor oscillations apparent on the right side and the rise in balloon pressure caused by hyperventilation.

DISCUSSION

The results demonstrate that the technique of using nasal balloons to record vasomotor activity in anaesthetized animals may also be used in conscious human subjects (Malm, 1973, 1974). The balloons were placed in direct contact with the nasal mucosa and changes in the volume of the nasal blood vessels would cause pressure changes in the balloons. Under resting control conditions the nasal balloon pressure was relatively constant and a consistent finding was that the congested side of the nose exhibited small oscillations in pressure at the same frequency as heart rate. These pressure oscillations are probably due to arterial pulsations and they may indicate that there is an asymmetry of nasal blood flow with the congested side of the nose having an increased blood flow during a recovery phase of the nasal cycle when the dominant nasal airflow is directed towards the other nasal passage.

Under resting conditions the airflow through the nasal passages is asymmetrical due to differences in the state of congestion of the venous erectile tissue in the nasal mucosa, and there is evidence that the filling of this tissue is regulated by the sympathetic innervation of the nose (Stoksted, 1953; Bojsen-Moller and Fahrenkrug, 1971; Änggård and Densert, 1974; Eccles, 1978). The pressure changes in the nasal balloons associated with breath holding or hyperventilation are probably caused by changes in the volume of the venous erectile tissue due to changes in sympathetic tone.

Breath holding caused a decrease in nasal balloon pressure due to nasal vasoconstriction and hyperventilation caused an increase in pressure due to nasal vasodilation. Nasal vasoconstriction in the dog caused by asphyxia has been shown to be abolished on section of the cervical sympathetic nerve (Tatum, 1923), and

similarly the reduction in human resistance caused by breath holding is abolished by stellate ganglion blockade (Hasegawa et al., 1976). These observations support the explanation given in the present study that rebreathing caused a vasoconstriction and a reduction in balloon pressure due to an increase in sympathetic tone. The increase in sympathetic tone is probably caused by an increase in the partial pressure of carbon dioxide in blood leading to stimulation of carotid and aortic chemoreceptors. The breath holding manoeuvre could be useful in some conditions of mild epistaxis as the induced vasoconstriction would promote haemostasis and control bleeding. Conversely a decrease in blood carbon dioxide caused by hyperventilation may bring about a reduction in sympathetic tone and cause an increase in balloon pressure due to increase filling of the venous erectile tissue. However, Tatum in 1923 reported that the nasal vasodilatation caused by hyperventilation in the anaesthetized dog was unaffected by section of the cervical sympathetic nerve. Therefore the nasal response to hyperventilation may not be simply due to a reduction in sympathetic tone. A possible direct effect of carbon dioxide on nasal blood vessels or changes in parasympathetic tone may be involved in the nasal response to hyperventilation. Any hyperventilation during nasal bleeding would tend to increase nasal blood flow and promote bleeding. In nasal surgery the ventilation of the anaesthetised patient may be of importance in determining nasal blood flow with hyperventilation causing nasal congestion and promoting nasal blood flow.

It is interesting to note that the congested nasal passage always gave the largest change in balloon pressure for both breath holding and hyperventilation. Similar results have been obtained in human studies where changes in nasal resistance to airflow were obtained in response to rebreathing or hyperventilation (Dallimore and Eccles, 1977; Hasegawa and Kern, 1978). In these airflow studies the congested side of the nose always gave the largest response to respiratory changes. One might expect the congested side of the nose to exhibit the largest vasoconstrictor response to rebreathing but the response to hyperventilation is paradoxical as it represents a further congestion of an already congested passage.

The results of the present study provide further information about the nasal vasomotor responses to changes in respiration. The asymmetry of the nasal responses may be due to an asymmetrical sympathetic tone to the nasal blood vessels and further studies on this problem may provide a better understanding of the nasal cycle in man.

ZUSAMMENFASSUNG

Mit Hilfe von wassergefüllten Ballons wurden am wachen Menschen vasomotorische Reaktionen in der Nase aufgezeichnet. Hyperventilation führte zu einem mit einer Vasodilatation einhergehende Anstieg des intranasalen Ballondruckes, während ein Anhalten des Atems einen Druckabfall und eine Vasokonstriktion

hervorrief. Die Amplitude dieser vasomotorischen Reaktionen in der Nase wurde von dem Nasenzyklus beeinflußt, wobei die stärkste Reaktion immer auf der Nasenseite auftrat, auf der eine Kongestion oder eine niedrigere Luftströmung bestand. Diese Ergebnisse lassen vermuten, daß ein erhöhter Kohlendioxydspiegel in den Arterien zu einer erheblichen Vasokonstriktion der Blutgefäße in der Nase führt. Bezüglich einer Kontrolle des Nasenblutens könnte dieses von klinischer Bedeutung sein.

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