Section of Laryngology

President H D Brown Kelly FRCS

Meeting March 7 1969

Papers

Nasal Reflexes

by Jennifer E Angell James MB
and Professor M de Burgh Daly MD SCD
(*Department of Physiology,
St Bartholomew's Hospital
Medical College, London*)

Stimulation of the nasal mucous membrane can be shown to produce striking reflex responses which affect not only respiration but the heart and peripheral blood vessels as well. The investigation of these reflexes, however, presents certain problems because they are modified, depressed or abolished by anæsthesia and furthermore the strength of the stimulus used determines to some extent the type of reflex response obtained. For instance, a chemical stimulus so weak that it might be termed an odour rather than an irritant causes an increase in breathing, whereas mild and strong irritants produce depression of respiration or even apnœa (Beyer 1901, Tomori 1965).

The purpose of this paper is to describe some of the effects resulting from stimulation of the nasal mucous membrane in man, to analyse more specifically some of the mechanisms by which the responses are brought about (here one has to resort to a large extent to the results obtained from experiments on laboratory animals) and, finally, to review the possible functions of these reflexes in the animal organism.

Nasal Reflexes in Man

The commonest type of respiratory reflex arising from the nose is the sneeze. This results from an abnormal stimulation of the trigeminal nerve endings by a combination of dilatation of the nasal blood vessels and a discharge of clear slightly viscous secretion (Brubaker 1919). These vasomotor and secretomotor effects may be caused by a number of factors, e.g. a reflex through excitation of cutaneous receptors by cold. Sneezing can also be triggered by afferent impulses from the eyes caused by bright light (Braus 1960). As a result of stimulation of the trigeminal nerve endings, a single slow inspiration or a series of inspiratory efforts occurs followed by an explosive expiratory effort. Sneezing in the cat and the dog is a common symptom in any nasal disease (Cook 1964) and can also be produced by electrical stimulation of the central cut ends of the parasympathetic nerves to the nasal mucous membrane (Malcomson 1959).

Little is known of the cardiovascular effects of sneezing, and this might be a profitable line of investigation. But they are probably dominated by the mechanical effects of changes of intrathoracic pressure which accompany the changes in respiration (Hamilton *et al.* 1944). The sneeze can be abolished by anæsthesia.

The respiratory and cardiovascular effects of mild irritants to the nasal mucous membrane have been studied by a number of workers. Inhalation of very low concentrations of sulphur dioxide causes rapid shallow breathing or deep slow breathing, tachycardia and variable changes in blood pressure (Amdur *et al.* 1953). But whether these responses were due to effects other than from the nose was not established. Other irritants such as ammonia vapour or cigarette smoke cause depression of breathing, bronchoconstriction, bradycardia and hypertension (Allen 1929, DuBois & Dautrebande 1958, Nadel & Comroe 1961). Irrigation of the nose with cold water or saline also results in apnœa and a rise of blood pressure (Ebbecke 1944). On the other hand, Harris (1939) found that the insufflation of ammonia vapour or cigarette smoke into the nose of unanæsthetized man in sufficient concentration to produce tears and pain had no effect on heart rate. However, electrical stimulation of the nose or aspiration of secretions from the nose of patients paralysed by disease and artificially ventilated caused an increase in heart rate and blood pressure (François-Franck 1889, Corbett *et al.* 1969). Russetzki (1925) applied a sinusoidal electrical current to the supraorbital, infraorbital and mental branches of the trigeminal nerve and observed in some normal subjects bradycardia and a fall in blood pressure. It is of interest that the responses he observed were much greater in patients with neuralgia or neuritis of the trigeminal nerve.

These results indicate that it is not in all normal subjects that reflex responses from the nose can be elicited, but on the other hand, it seems likely that exaggeration of the reflexes occurs in patients with certain pathological conditions of the nose and in particular in those with increased irritability of the trigeminal nerve (François-Franck 1889, Killian 1910, Russetzki 1925, Harris 1939).

It has been recognized for a long time that diseases of the nasal passages, such as sinusitis, hypertrophic rhinitis and polypi, are of importance in the etiology of bronchial asthma (McBride 1887, Adam 1900, Sluder 1919) and there are a number of reports of successful treatment of the bronchial condition after eradication of the nasal abnormality (Adam 1900, Francis 1902).

Undoubtedly the most dramatic effects of nasal stimulation are those resulting from changes in the cardiovascular system, and these may result in unconsciousness and even death. Grove (1922) has reviewed the recorded cases of 29 patients who became unconscious and of 16 who died suddenly as a result of puncture and irrigation of the maxillary sinus. Although not excluding a trigeminal-vagal reflex, he considered that the most likely cause of these mishaps was air embolism. Similar accidents occurring during minor procedures in the nose and sinuses have been reported by Dabney (1915) and Loeb (1923). In man the mucosa covering the approaches to the paranasal sinuses are the most sensitive parts of the nasal mucous membrane, at least to pain (McAuliffe *et al.* 1945).

Sudden submersion in water in such a way that water enters the nose may also lead to loss of consciousness and death. In this connexion the murder trial known as the case of 'The brides in the bath' may be cited. George Joseph Smith was thought to have caused the death of each of his three victims by submerging her head in water while taking a bath (Watson 1922, Crew 1933). This, it was believed, was accomplished by slipping his hand and arm under the knees of the victim, placing the other hand on the head and pushing the head down and the legs up, so that a rush of water entered the nose. As the case was unique in forensic medicine, one of the detectives engaged on the case persuaded a young lady of his acquaintance, a practised swimmer, to sit in a bath filled with water. Although fully aware she was to be submerged, she was unable to get her head above water after it was once forcibly submerged. The inspector said: 'She was unconscious. For nearly half an hour my detectives and I worked away at her with artificial respiration and restoratives. The lady explained afterwards that immediately she fell back with her legs held in the air, the water rushed into her mouth and up her nostrils, making her unconscious' (Crew 1933). Sudden reflex cardiac arrest occurring during water immersion due to impingement of water on the upper respiratory tract is now a cause of death well recognized by forensic experts (Gardner 1942, Camps 1968).

In view of the fact that the more potent of these reflexes are not well-recognized and that patients may clinically present signs and symptoms not unlike those of the common 'faint' or vasovagal attack, the reflexes will be analysed now in more detail.

Experimental Evidence for Reflexes from the Nose

These reflexes arising from the nasal mucous membrane and affecting respiration and the cardiovascular system are amongst the most potent in the experimental physiologist's armoury and their existence was first demonstrated by Kratschmer (1870). He showed in the cat and the rabbit that chemical irritation of the nasal mucous membrane, particularly by tobacco smoke, caused apnœa, closure of the larynx and bronchoconstriction; on the cardiovascular system these stimulants caused slowing of the heart rate and variable changes in blood pressure. Subsequently his results were confirmed by François-Franck (1889), Brodie & Russell (1900), Dixon & Brodie (1903), Ellis (1936), Rall *et al.* (1945) and by many others. Amongst the irritants which have been shown to produce these effects are ether, cigarette smoke, chloroform, benzol and ammonia vapour, and sulphur dioxide. Similar responses can be evoked by irrigation of the nose with water, or by mechanical or electrical stimulation of the nasal mucous membrane. Dixon & Brodie (1903) describe the most effective spot in the nose for bringing about these responses as a small area well back on the nasal septum (for comparative findings in man *see* McAuliffe *et al.* 1945).

In experiments in which the stimulus is applied to the nasal mucous membrane, it can be shown that all the above-mentioned responses are abolished by the application of a local anæsthetic to the nose, or by division of the trigeminal nerves which therefore form the afferent pathway for the reflex (Kratschmer 1870, François-Franck 1889, Allen 1929, Ebbecke & Knüchel 1943). This finding is supported by the fact that electrical stimulation of the trigeminal nerve also causes apnœa, bradycardia and hypotension (François-Franck 1876).

On the efferent side of the reflex arc, the respiratory effects are mediated by the nerves to the respiratory muscles, whereas the bradycardia and bronchoconstriction are almost entirely dependent on the integrity of the vagus nerves (Dixon & Brodie 1903). Since vagal effects are mediated by a cholinergic mechanism, they can be largely counteracted by atropine. The small residual effects persisting after blocking the parasympathetic nerve endings may be due to a reduction in the impulse discharge in sympathetic nerves (Rall *et al.* 1945).

Effects of Stimulation of the Nasal Mucous Membrane in the Dog

So far as we are aware there have been no previous reports of the changes in vascular resistance in different parts of the body accompanying stimulation of the nasal mucous membrane. A study of the reflex changes in distribution of systemic blood flow during stimulation of the nasal mucous membrane was therefore made in anæsthetized dogs, the alterations in respiration, heart rate and arterial blood pressure being recorded simultaneously.

Methods

The method by which the responses from the nose were evoked is shown in Fig 1. A cuffed tube was inserted into the posterior nasal fossa and air was slowly drawn through the nose in the direction of the arrows. In this way, vapours or water could be administered through the air-tight mask surrounding the nose. A free airway was maintained by a tracheostomy tube and this was connected to a closed-circuit respiratory system for recording respiration quantitatively by means of a balanced spirometer. We used water or saline (0·9%) at measured temperatures which varied from 4 to 37°C in different tests to stimulate the nasal mucous membrane in most of our experiments because this was found to give more reproducible

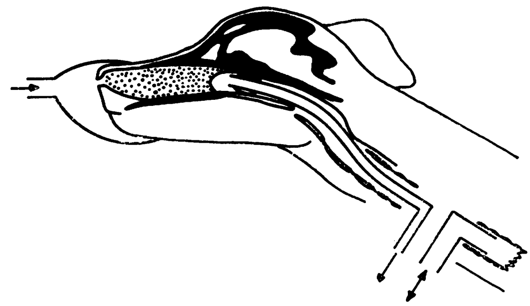

Fig 1 *Diagram showing the method for passing liquids and vapours over the nasal mucous membrane (stippled area) in the direction of the two arrows. The balloon on the end of a cuffed tube is inflated in the nasopharynx and an airtight mask surrounds the muzzle. A tracheostomy tube is connected to a closed circuit respiratory system for recording tidal volume (double-headed arrow)*

results than chemical stimulants, such as ammonia, ether and tobacco smoke, or electrical stimuli. Measurements of arterial blood pressure were made using a Statham transducer (model P23Gb). The amplitude distortion of the catheter-manometer system was less than 5% up to a frequency of 40 Hz. The mean blood pressure, obtained electrically by a simple resistance-capacitor network with a time constant of 1 sec, was also recorded. Simultaneous measurements were made of mean blood flow (Nycotron electromagnetic flow meter) in different arteries, of heart rate and of the movements of the spirometer. The electrical signals, after suitable amplification, were displayed on a direct-writing ultraviolet light recorder (S.E. Laboratories Ltd).

Vascular resistance was calculated according to the formula:

$$\text{Vascular resistance} = \frac{\text{mean arterial pressure} - \text{mean venous pressure (mm Hg)}}{\text{mean blood flow (ml/min)}}$$

The vascular resistance was expressed in convenient units (peripheral resistance units) representing the pressure necessary to force blood at 1 ml/min through the vascular bed under test. The venous pressure was not measured in all experiments, but it was found to vary by not more than 5 mmHg (Fig 5). The errors introduced by ignoring this figure in the calculation of vascular resistance are therefore negligible.

Respiratory and Cardiac Responses

In dogs under sodium pentobarbitone (Nembutal) or chloralose-urethane anæsthesia, stimulation of the nasal mucous membrane caused a reduction or inhibition of respiration. The typical responses

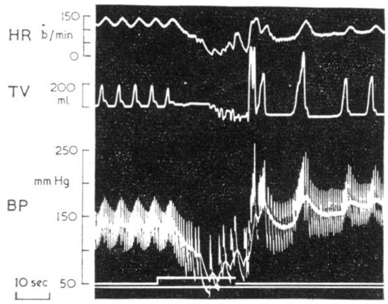

Fig 2 *Effects of stimulation of the nose by water at 21° C. Dog, female, 13·3 kg.* HR, *heart rate;* TV, *tidal volume (inspiration upwards);* BP, *arterial blood pressure and mean blood pressure. Time calibration, 10 sec*

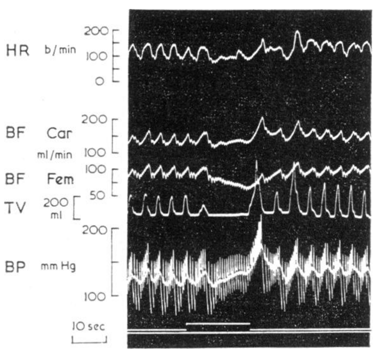

Fig 3 *Effects of blowing tobacco smoke through the nose on carotid and femoral blood flows. Dog, female, 21·0 kg.* HR, *heart rate;* BF, *blood flow;* Car, *carotid artery;* Fem, *femoral artery;* TV, *tidal volume (inspiration upwards);* BP, *arterial blood pressure and mean blood pressure. Time calibration, 10 sec*

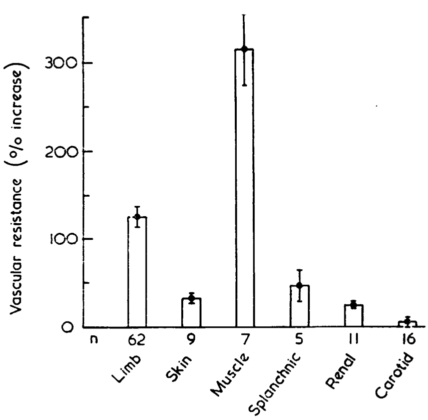

Fig 4 *The effects of stimulation of the nasal mucous membrane on the vascular resistance (expressed as a percentage of the control value) in different vascular territories. Each rectangle represents the mean ± S.E.M., and* n *is the number of observations*

are shown in Figs 2, 3 and 4. Breathing was invariably inhibited in the expiratory position, a finding to which Kratschmer (1870) drew attention. In 86 tests in 13 experiments the heart rate slowed, the mean reduction in rate being 27·3% (S.E.M. ±1·9). The arterial blood pressure responses were variable, but when marked reductions of heart rate occurred, a fall in pressure always occurred (Figs 2 and 3). Varying the temperature of the nasal perfusate between 4 and 37°C appeared to make little difference. Similar responses were evoked by tobacco smoke and these are shown in Fig 3.

Vasomotor Responses

Nearly all workers who have studied the effects of stimulation of the nasal mucous membrane have observed variable changes in blood pressure. This is hardly surprising because the blood pressure is dependent on the product of cardiac output and the total peripheral vascular resistance, and if these parameters were to alter in opposite directions during stimulation of the nose, the magnitude of their respective changes would determine whether the blood pressure increased, decreased or remained unchanged.

Stimulation of the nasal mucous membrane of the dog invariably caused an increase in vascular resistance in skin, muscle, splanchnic vascular bed and kidney, but not in the carotid circulation. These increases in vascular resistance indicate vasoconstriction. In Fig 3, for instance, the arterial blood pressure showed little change initially but increased slightly during the latter part of the period of the stimulus which in this case was tobacco smoke. During this time there was a progressive reduction in femoral blood flow from 90 to 65 ml/min so that the calculated femoral vascular resistance increased.

The results of all experiments are shown in Fig 4 in which the changes in vascular resistance in various vascular territories have been expressed as a percentage change from the control value. The increase in resistance in the femoral artery of the normal limb was 125·7±11·9%, and both skin and muscle contributed to this response, the vascular resistance increasing by 31·9±5·7% and 314·0±50·0% respectively. Vasoconstriction also occurred in the splanchnic vascular bed, as indicated by an increase in resistance in the vascular bed of the superior mesenteric artery of 46·6±18·1%, and also in the kidney (renal artery), the vascular resistance increasing by 10·7±1·9%. In all these vascular territories, the increase in resistance is highly significant ($P<0·001$). The number of observations in each group of experiments differs widely because this study is still in progress. It is therefore premature to make a quantitative comparison of the size of the resistance changes in the different vascular territories. By contrast, however, are the changes in resistance in the vascular bed of the common carotid artery, the mean increase being only $5{\cdot}8 \pm 5{\cdot}8\%$. This is not significantly different from zero ($P > 0{\cdot}3$).

Control experiments were carried out to establish that these vascular responses were primary reflexes from the nose and not secondary either to inhibition of breathing or to the alteration in arterial blood pressure. It was found in addition that the responses were abolished by denervation of the vascular bed, by the ganglionic blocking agent hexamethonium and by the adrenergic blocking agent guanethedine, indicating that they are mediated by sympathetic adrenergic nerves. None of these procedures affected the respiratory responses. The application of a local anæsthetic to the nasal mucous membrane, however, abolished the respiratory, cardiac and vasomotor effects.

Similar vasomotor responses were observed in experiments in which the nasal mucous membrane was electrically stimulated, and in those using ammonia, ether or tobacco smoke (Fig 3).

Changes in Cardiac Output

The reason for the arterial blood pressure falling on occasions during stimulation of the nasal mucous membrane, in spite of vasoconstriction occurring in a large proportion of the systemic vascular bed is due to the overriding effect of a concomitant reduction in cardiac output. This is illustrated by Fig 5 in which the blood flow in the

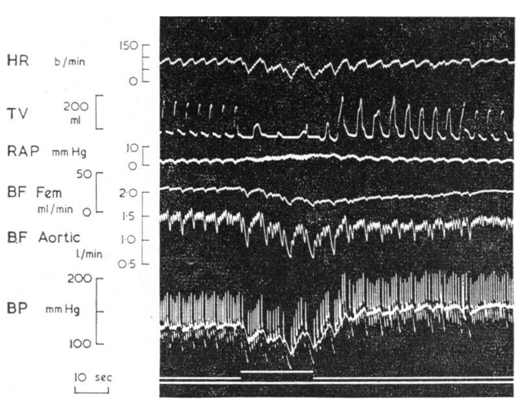

Fig 5 *Effects of stimulation of the nose by water at 5°C on cardiac output. Dog, male, 16·8 kg.* HR, *heart rate;* TV, *tidal volume (inspiration upwards);* RAP, *right arterial pressure;* BF, *blood flow;* Fem, *femoral artery;* Aortic, *ascending aortic;* BP, *arterial blood pressure and mean blood pressure. Time calibration, 10 sec*

ascending aorta was recorded. This was taken as a measure of the cardiac output but does not include the coronary blood flow which is only about 5% of the total left ventricular output. At the same time as the cardiac output diminished from 1·5 to 1·1 litres/min, a reduction in femoral blood flow and an increase in resistance in the femoral vascular bed occurred.

Summary: We have confirmed that stimulation of the nasal mucous membrane causes reflexly a reduction in breathing or apnœa in the expiratory position, bradycardia and hypertension or hypotension. In addition we have shown that reflex vasoconstriction occurs in the skin, muscle, splanchnic vascular bed and kidney, but not in the carotid circulation, combined with a reduction in cardiac output. It will be evident therefore that these responses differ from those in a vasovagal faint in which the prominent features are bradycardia and vasodilatation, not vasoconstriction (Barcroft *et al.* 1944).

Reflexes of Other Parts of the Upper Respiratory Tract

Stimulation of the mucous membrane of other parts of the upper respiratory tract also causes reflex effects involving respiration and the cardiovascular system, but the responses differ in some respects from those elicited from the nose. For instance, digital pressure on the mucous membrane in the vault of the pharynx in the region of the eustachian orifice stimulates breathing in the apnœic anæsthetized cat and dog (Cook 1964).

The laryngeal mucosa is probably the most sensitive part of the respiratory tract and its stimulation causes coughing and also bradycardia, bronchoconstriction and hypertension (Nadel & Widdicombe 1962). Cardiac arrest may occur as a result of surgical interference with the larynx under inadequate anæsthesia (Burstein 1949, Cook 1964). Recently two new types of respiratory reaction have been described and collectively termed the 'pharyngeal respiratory reflexes'. These are evoked from the laryngopharyngeal region ('laryngopharyngeal cough') and the epipharyngeal region ('aspiration reflex'), and give rise to coughing and sneezing, and a series of inspiratory effects without accompanying expiratory efforts respectively. Both reflexes are accompanied by hypertension (Tomori & Widdicombe 1969). Reflexes from the upper respiratory tract have been reviewed by Widdicombe (1963).

Physiological Function of Nasal Reflexes

Ramos (1960) found that blowing air through the nose caused changes in breathing and he concluded that in normal respiration rhythmic excitation of the fifth nerve afferents would contribute to the smoothness of respiratory movements, sharing this action with other afferents, particularly those in the vagus nerves subserving the Hering-Breuer respiratory reflex. With stronger irritants the combination of apnœa, laryngeal spasm and bronchoconstriction must serve a protective function preventing the agent gaining access to the lungs.

Although it is difficult to ascribe any useful function to the accompanying cardiovascular responses of strong excitation of the nasal mucous membrane, it is of interest to compare them with those occurring in aquatic mammals and diving birds during submersion. These animals are capable of remaining submerged for prolonged periods of time through cardiovascular adaptations. Immediately the head is immersed in water apnœa, bradycardia, vasoconstriction and a reduction in cardiac output occur and it has been established that one important mechanism giving rise to these responses is stimulation of receptors in and around the nose, the afferent pathway involved being the trigeminal nerves (*see* Andersen 1966). Similar responses occur in man on immersing the face but it appears that the receptors involved are largely those in the skin (Kawakami *et al.* 1967).

The sequence of events is shown diagrammatically in Fig 6. The reflex responses occurring as a result of stimulation of the receptors in and around the nose are indicated by the continuous lines and include apnœa in the expiratory position through inhibition of the respiratory centres, bradycardia and vasoconstriction. Then, as a result of inhibition of respiration, a gradual fall in the arterial blood P_{O_2}, a rise in P_{CO_2} and a reduction of pH occur, leading to asphyxia. These changes in the blood now excite the arterial chemoreceptors situated in the carotid and aortic bodies and, as shown in Fig 6 by the interrupted lines, their combined stimulation causes reflex bradycardia and vasoconstriction through stimulation of the cardio-inhibitory and vasomotor centres respectively (Angell James & Daly 1969). These responses therefore reinforce those elicited by stimulation of the nasal receptors.

The afferent pathways for the chemoreceptor reflexes are the carotid sinus nerves (branches of the glossopharyngeal nerves) and aortic nerves (branches of the vagus nerves).

Another reflex response resulting from stimulation of these chemoreceptors is an increase in respiration, but the fact that apnœa persists during diving at a time when the carotid and aortic bodies are being increasingly stimulated would suggest that their excitatory impulses are inhibited centrally, presumably by the afferent impulses from nasal receptors. This point, however, requires further study. But there must eventually come a time when asphyxia becomes so great that impulses from the peripheral arterial

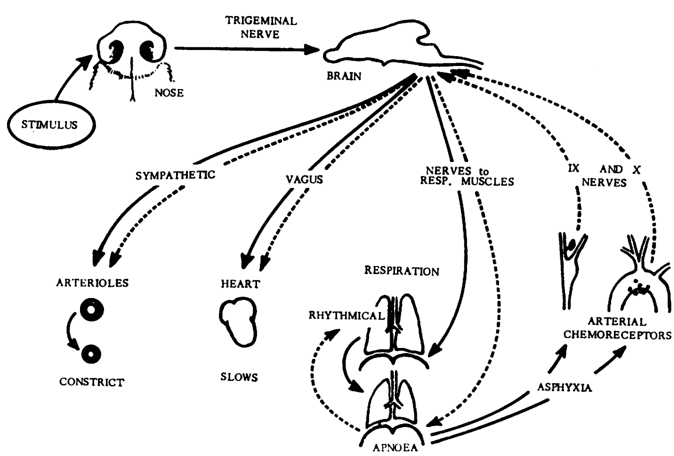

Fig 6 *Diagrammatic representation of the reflex responses occurring in mammals during submersion. The water stimulus to receptors in and around the nose leads to apnœa, bradycardia and vasoconstriction (continuous lines). The apnœa, in turn, causes arterial hypoxia and hypercapnia (asphyxia) which stimulates the carotid and aortic body chemoreceptors. Their combined excitation reflexly reinforces the cardio-inhibitory and vasoconstrictor responses (interrupted lines). Eventually asphyxia acting through the peripheral arterial chemoreceptors and central chemoreceptors will stimulate respiration (interrupted lines). For details,* see *text*

chemoreceptors and the central chemoreceptors break through the central block and cause breathing to start again (Fig 6). Before this break-through occurs, the animal must, of course, surface.

The intense peripheral vasoconstriction through stimulation of nasal receptors and arterial chemoreceptors, in those tissues that can withstand relatively prolonged periods of hypoxia, causes redistribution of the diminished cardiac output to the brain and the cardiac muscle, which are known to be damaged by even brief periods of hypoxia. In this way the oxygen available in the circulating blood and lungs during the period of submersion is made available to those tissues requiring it most urgently, and this mechanism is one of the most important by which diving animals can remain submerged for such prolonged periods of time.

From the foregoing it is apparent that the nasal cardiovascular reflexes serve a purposeful function in these diving mammals.

Acknowledgments: We wish to express our thanks to Mr D R Bacon for technical assistance. This work was generously supported by grants from the Medical Research Council, the Royal Society and St Bartholomew's Hospital Endowment Fund.

REFERENCES

Adam J (1900) *Glasg. Hosp. Rep.* 3, 151
Allen W F (1929) *Amer. J. Physiol.* 88, 620
Amdur M O, Melvin W W & Drinker P (1953) *Lancet* ii, 758
Andersen H T (1966) *Physiol. Rev.* 46, 212
Barcroft H, Edholm O G, McMichael J & Sharpey-Schafer E P (1944) *Lancet* i, 489
Beyer H (1901) *Arch. Anat. Physiol. (Lpz.) Anat. Abt.* Suppl. p 261
Braus H (1960) Anatomie des Menschen. Berlin
Brodie T G & Russell A E (1900) *J. Physiol. (Lond.)* 26, 92
Brubaker A P (1919) *J. Amer. med. Ass.* 73, 585
Burstein C L (1949) Fundamental Considerations in Anæsthesia. New York
Camps F E ed (1968) Gradwohl's Legal Medicine. 2nd ed. Bristol; p 347
Cook W R (1964) *J. small Anim. Pract.* 5, 309
Corbett J L, Kerr J H & Prys-Roberts C (1969) *J. Physiol. (Lond.)* 210, 51P
Crew A (1933) The Old Bailey. London
Dabney V (1915) *Trans. Amer. Acad. Ophthal. Otolaryng.* p 53
Dixon W E & Brodie T G (1903) *J. Physiol. (Lond.)* 29, 97
DuBois A B & Dautrebande L (1958) *J. clin. Invest.* 37, 1746
Ebbecke U (1944) *Klin. Wschr.* 23, 141
Ebbecke U & Knüchel F (1943) *Pflügers Arch. ges. Physiol.* 247, 255
Ellis M (1936) *Proc. roy. Soc. Med.* 29, 527
Francis A (1902) *Lancet* ii, 1053
François-Franck Ch-A
(1876) *C.R. Ass. franç. Av. Sci.* 5, 806
(1889) *Arch. Physiol. norm. path.* Ser. 5, 1, 538
Gardner E (1942) *Med.-leg. Rev.* 10, 120
Grove W E (1922) *Ann. Otol. (St. Louis)* 31, 913
Hamilton W F, Woodbury R A & Harper H T jr (1944) *Amer. J. Physiol.* 141, 42
Harris A S (1939) *Ann. Otol. (St. Louis)* 48, 311
James J E A & Daly M de B (1969) *J. Physiol. (Lond.)* 201, 87
Kawakami Y, Natelson B H & DuBois A B (1967) *J. appl. Physiol.* 23, 964
Killian G (1910) *Dtsch. med. Wschr.* 36, 1868
Kratschmer F (1870) *S.-B. Akad. Wiss. Wien, math.-nat. Kl.* 62, 147
Loeb H W (1923) *Trans. Amer. Laryng. rhin. otol. Soc.* p 226
Malcomson K G (1959) *J. Laryng.* 73, 73
McAuliffe G W, Goodell H & Wolff H G (1945) *Res. Publ. Assoc. nerv. ment. Dis.* 23, 185
McBride P (1887) *Brit. med. J.* i, 205
Nadel J A & Comroe J H jr (1961) *J. appl. Physiol.* 16, 713
Nadel J A & Widdicombe J G (1962) *J. appl. Physiol.* 17, 861
Rall J E, Gilbert N C & Trump R (1945) *J. Lab. clin. Med.* 30, 953
Ramos J G (1960) *Acta physiol. lat.-amer.* 10, 104
Russetzki J (1925) *Gaz. Hôp. (Paris)* 98, 549
Sluder G (1919) *J. Amer. med. Ass.* 73, 589
Tomori Z (1965) *Physiol. bohemoslov.* 14, 84
Tomori Z & Widdicombe J G (1969) *J. Physiol. (Lond.)* 200, 25
Watson E R (1922) *Trans. med.-leg. Soc. (Lond.)* 16, 134
Widdicombe J G (1963) Handbook of Physiology. American Physiological Society, Washington, D.C.; Sect. 3, 1, 585

DISCUSSION

Mr K Greer Malcomson (*Bristol*) said he had read a paper on the vasomotor activities of the nasal mucous membrane to the Section ten years previously (1959, *J. Laryng.* 73, 73) in which he had demonstrated threshold stimuli unlike those used by Dr Angell James and Professor Daly.

He found that the sneezing reflex was invested in the parasympathetic component of nasal innervation, via the greater superficial petrosal nerve, the vidian nerve or the nerve of the pterygoid canal, relaying in the sphenopalatine ganglion whence the postganglionic fibres went to the nasal mucous membrane. Stimulation of the branches of the trigeminal going to the nose produced reflexes in the jaw and neck muscles but there was no sneezing.

It was generally believed that in a minority of people sneezing had a psychosomatic basis, and Mr Malcomson supported this. It had a parallel in dogs who might express pleasure at the return of their owner by the usual fussing plus sneezing.

The following paper was also read and may be published in a later issue of *Proceedings:*

Painful Glomus Tumours of the Larynx
Mr Douglas Ranger and Professor A Thackray