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REFLEX BRADYCARDIA OCCURRING IN RESPONSE TO DIVING, NASOPHARYNGEAL STIMULATION AND OCULAR PRESSURE, AND ITS MODIFICATION BY RESPIRATION AND SWALLOWING

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SUMMARY

1. Digital pressure applied to the eyes evokes reflex bradycardia in human subjects and anaesthetized dogs. The bradycardia is most pronounced when breathing stops.

2. In the dog oculo-cardiac reflex bradycardia is mediated by vagal stimulation and by sympathetic withdrawal.

3. Oculo-cardiac reflex bradycardia in the dog is reduced by central neural inspiratory activity and by the excitation of pulmonary afferents by inflation of the lungs. In human subjects it is reduced when inspiratory efforts are made against a closed glottis.

4. Nasopharyngeal stimulation with water evokes reflex bradycardia in the anaesthetized dog. This bradycardia is reduced by central neural inspiratory activity and by the excitation of pulmonary afferents by inflation of the lungs.

5. Bradycardia occurs in normal human subjects during immersion of the face in water ('diving'). This bradycardia is reduced when inspiratory efforts are made against a closed glottis.

6. Swallowing evokes transient tachycardia in human subjects. During diving or the application of ocular pressure, swallowing reduces the reflex bradycardia which these evoke.

INTRODUCTION

Brief stimuli delivered to the carotid chemoreceptors and baroreceptors evoke prompt reflex bradycardia when they are timed to occur in the expiratory phase of the respiratory cycle, but have little or no effect on heart rate when given during inspiration. (Koepchen, Wagner & Lux, 1961; Haymet & McCloskey, 1975). The bradycardia evoked in these reflexes is mediated mainly by the vagus nerves, although withdrawal of cardiac sympathetic tone can also be involved and is similarly modulated by the respiratory cycle (Davis, McCloskey & Potter, 1977). The respiratory modulation of these reflexes is attributable to two mechanisms: there is an inhibitory effect on the reflexes arising from activity of inspiratory neurones in the central nervous system (Koepchen *et al.* 1961; Davidson, Goldner & McCloskey, 1976), and there is a further inhibitory effect arising from the excitation of intrapulmonary receptors during air flow into the lungs (Gandevia, McCloskey & Potter, 1978). Presumably both inhibitory mechanisms usually operate together.

In the present study we have investigated other stimuli known to evoke bradycardia, and have looked for modulations of reflex effectiveness attributable to central and peripheral reflex respiratory mechanisms. The reflexes studied here were the oculo-cardiac reflex (Aschner, 1908), the diving reflex (Andersen, 1966) and a reflex arising from naso-pharyngeal stimulation (cf. Angell James & Daly, 1972), in experiments on normal awake human subjects and anaesthetized dogs. We also report observations on the changes in heart rate which accompany swallowing in man, and discuss these in the light of our other findings.

METHODS

Animal experiments. Experiments were performed on eighteen adult dogs of both sexes, weighing 9–19 kg. Eleven of these animals were also used in the study of chemoreceptor and baroreceptor reflexes described in the preceding paper (Gandevia *et al.* 1978). The animals were anaesthetized with intravenous chloralose (α-chloralose: British Drug Houses; 80 mg/kg). In each dog the trachea was cannulated low in the neck. A nylon cannula was inserted in a femoral vein for administration of anaesthetic or drugs. Rectal temperature was maintained at 37–39 °C.

Arterial blood pressure was measured through a cannula in a lingual artery using a Statham P23AC transducer, and was recorded on one channel of a Grass polygraph pen recorder. The electrocardiogram and the beat by beat heart rate (Grass 7P4 cardiotachometer, triggered from the e.c.g.) were recorded. Respiratory activity was measured according to the requirements of the particular experiment. In some animals an indication of tracheal air flow was obtained by measuring the pressure at the tip of a wide (2 mm i.d.) catheter inserted into the trachea, using Statham transducer. Alternatively, respiratory activity was measured by a bag-in-box method similar to that described by Donald & Christie (1949) in which the animal inspired through a valve from a bag enclosed in an airtight box into which expired air was led; pressure in the box, which was related to the tidal volume of the breath taken, was measured using a Grass PT5A volumetric pressure transducer. In paralysed animals, respiratory activity was recorded from the central end of the cut and de-sheathed phrenic nerve using platinum electrodes. This phrenic neural discharge was integrated using a Grass 7P3B preamplifier ('leaky' integrator; time constant 0·05 sec).

Two reflexes were tested in anaesthetized dogs, the oculo-cardiac reflex and the reflex elicited by nasopharyngeal stimulation. The oculo-cardiac reflex was elicited by an experimenter placing the thumbs on the closed eyelids of the animal and pressing. No measurement of the pressure used was made, but similar pressures were applied to awake human subjects in other experiments (see below) without causing great discomfort. Nasopharyngeal stimulation was achieved by a method similar to that described by Angell James & Daly (1972). A cuffed, endotracheal tube was inserted into the trachea pointing rostrally and advanced until its tip lay in the nasopharynx. The tube was then tied firmly into the trachea, and the cuff was inflated. Tap water at room temperature was led into this tube when nasopharyngeal stimulation was required, and was collected through a rubber glove tied over the muzzle as it flowed out of the nose and mouth. The rate of flow of water was usually 1 l./min.

Human experiments. Experiments were performed on eight healthy subjects of both sexes, including the three authors. Electrocardiograms were measured from conventional limb leads, and beat by beat heart rate was obtained using a Grass 7P4 cardiotachometer triggered from the e.c.g. Both were recorded on the polygraph. For both diving reflex and oculo-cardiac reflex studies subjects were usually seated. Diving reflexes were elicited by the subject's immersing the nose and mouth in a small bowl of cold water. Oculo-cardiac reflexes were elicited by an experimenter placing the thumbs on the subject's closed eyelids and pressing. The pressure was firm, but subjects were instructed to tell the experimenter as soon as any discomfort occurred; all the results reported here were obtained in experiments in which the subjects reported no discomfort.

In the diving experiments the subjects, of course, stopped breathing while their faces were immersed. In the experiments on the oculo-cardiac reflex subjects were instructed to stop breathing while the eye pressure was applied (see Results). Although we noted no consistent differences between reflexes elicited with the breath held in inspiration and those in which it was

held in expiration, we usually asked our subjects to commence breath-holding at the normal end-expiratory point. In the course of a 'dive' or of application of eyeball pressure during a breath-hold, subjects performed one of two simple manoeuvres in response to an instruction from an experimenter. The subjects were required either to swallow or to make a single inspiratory effort against a closed glottis ('false breath'). Swallowing was used in these experiments because it had been found that resting subjects breathing normally increase their heart rates by 10–30 beats/min for a few beats on each occasion they swallow (Fig. 1; see also Miller & Sherrington, 1916).

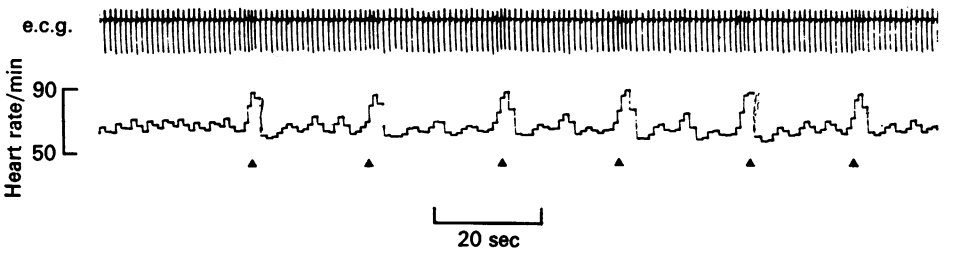

Fig. 1. Effects of swallowing on heart rate. Records show e.c.g. and heart rate of a normal resting human subject. The subject swallows on six occasions, marked by filled triangles.

RESULTS

Oculo-cardiac reflex

Animal experiments. The typical response to the application of pressure to the eyes in the anaesthetized dog was a bradycardia. In some dogs this was considerable, in others it was slight, but in all there was an enhancement of respiratory sinus arrhythmia accompanying the bradycardia. In about twelve of eighteen dogs tested, there was also some reduction in the rate and depth of breathing during the application of pressure.

In order to test the effectiveness of the oculo-cardiac reflex during periods in which an animal was not breathing, Hering–Breuer inflation reflexes were evoked by occluding the trachea at the end of normal inspirations. When the lungs were thus held inflated the animal made no attempt to breathe for many seconds, during which time the effectiveness of the reflex could be tested. Typically, there was a slight increase in heart rate during a control trial of lung inflation alone. This was probably attributable to the slight fall in blood pressure which occurred during the period of positive intrathoracic pressure. When pressure was applied to the eyes during Hering–Breuer inflation apnoea, however, the bradycardia which was evoked was large, and was always larger than the bradycardia evoked by similar pressure applied during normal breathing. If both eyeball pressure and lung inflation were maintained for long enough, the animal would ultimately make an inspiratory effort. Whether such an inspiratory effort was permitted to draw air into the lungs (Fig. 2) or not (Fig. 4), it was accompanied by a sudden and marked reduction in the bradycardia which then returned as the inspiratory effort concluded. Responses of this type are shown in Fig. 2; similar responses were obtained in all dogs in which oculo-cardiac reflexes could be evoked (fourteen of eighteen dogs).

The oculo-cardiac reflex bradycardia was mediated mainly by the vagus nerves. This was shown in six dogs in which previously marked bradycardia was all but

abolished following vagotomy or administration of atropine (1–2 mg/kg). In all six, however, slight bradycardia was still evoked by eyeball pressure, and this was attributable to withdrawal of sympathetic neural tone, as it was no longer evoked after administration of propranolol (1 mg/kg). In three of the six animals in which bradycardia due to sympathetic withdrawal could be demonstrated, there was a greater response elicited when eyeball pressure was applied during the expiratory

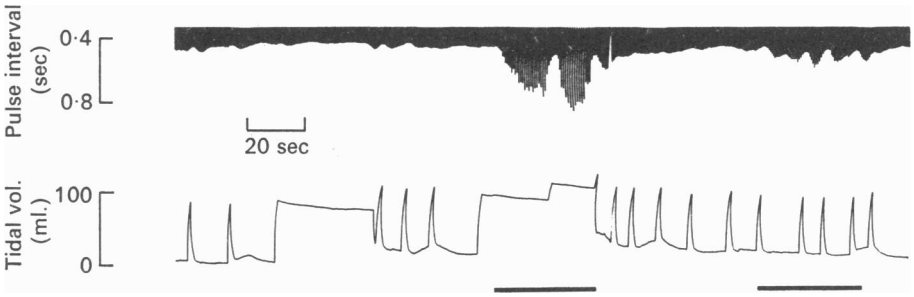

Fig. 2. Dog, anaesthetized with chloralose. Records show pulse interval and tidal volume (measured by bag-in-box method, inspiration upwards). Marker bars at bottom of figure show periods during which digital pressure was applied to the eyes. On two occasions the tidal volume record shows inspiratory apnoea, achieved by occluding the expiratory line. Inspiratory apnoea or ocular pressure alone evoke little change in heart rate. When ocular pressure is applied during a period of inspiratory apnoea, there is marked bradycardia; note that this bradycardia is transiently reduced when the inspiratory apnoea is interrupted by a breath.

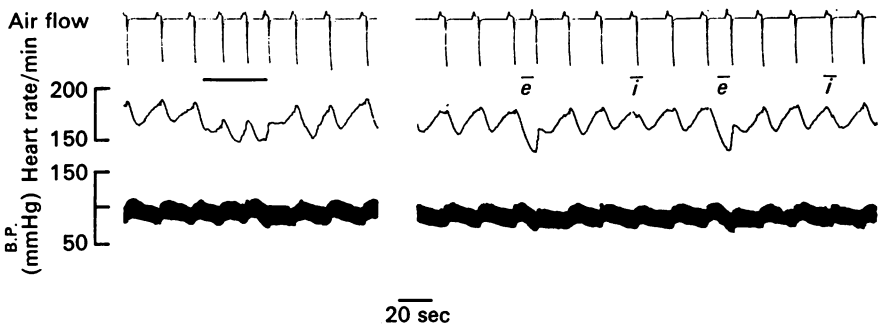

Fig. 3. Dog, anaesthetized with chloralose; bilateral vagotomy. Records show tracheal air flow (inspiration upwards), heart rate and arterial blood pressure. Marker bars below the air flow record show periods in which pressure was applied to both eyes. In the panel at the left the bradycardia in response to sustained ocular pressure is seen. In the panel at the right the bradycardia evoked by ocular pressure applied only during the expiratory phase of breathing (*e*) is compared with that evoked by similar pressure applied only during inspiration (*i*).

phase of breathing than when it was applied during inspiration. This effect is shown in Fig. 3, and is similar to respiratory modulation of sympathetic responses to chemoreceptor and baroreceptor stimuli described by Davis *et al.* (1977).

In seven dogs more elaborate experiments were performed in order to elucidate the respiratory mechanisms responsible for reducing oculo-cardiac bradycardia whenever an inspiration occurred (e.g. Fig. 2). These animals were paralysed with D-tubo-

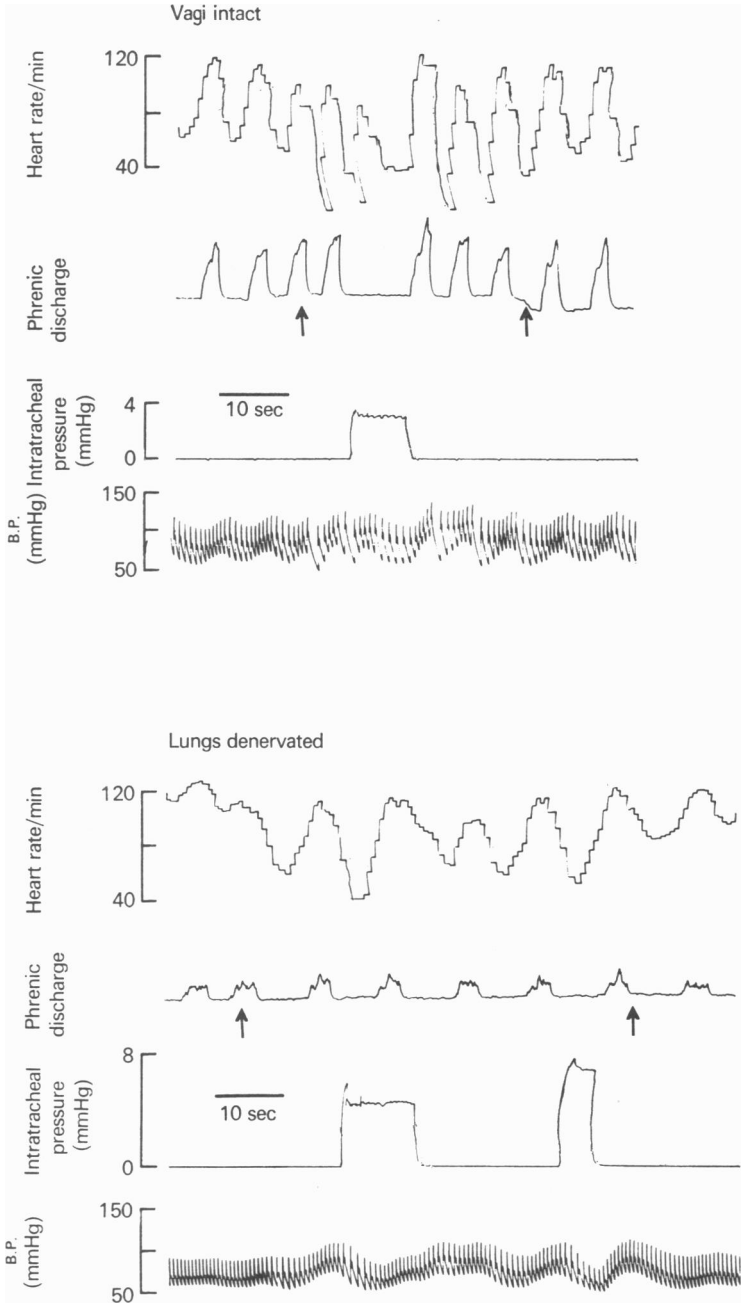

Fig. 4. Dog, anaesthetized with chloralose, paralysed with D-tubocurarine. Records of heart rate, integrated phrenic neural activity, intratracheal pressure and arterial B.P., obtained during periods of temporary cessation of artificial ventilation, are shown. Ocular pressure was applied between the arrows. In the upper panel the response to ocular pressure is seen: there is a pronounced bradycardia which is reduced on each occasion that inspiratory (phrenic) activity occurs. The bradycardia is also reduced as the lungs are inflated, but returns while the inflation is maintained. In the lower panel the response to ocular pressure is seen after surgical denervation of the lungs; bradycardia is again evoked but reduced on each occasion that inspiratory activity occurs. The bradycardia is no longer reduced by lung inflation, nor does inflation inhibit inspiration as it did before pulmonary denervation.

curarine (1–2 mg/kg) and artificially ventilated on oxygen with a Starling 'Ideal' pump. From time to time the pump was stopped and experiments on the oculo-cardiac reflex were performed. Respiratory activity in the paralysed animals was indicated by the integrated phrenic electroneurogram. When pressure was applied to the eyes in these animals, oculo-cardiac reflex bradycardia occurred. With each

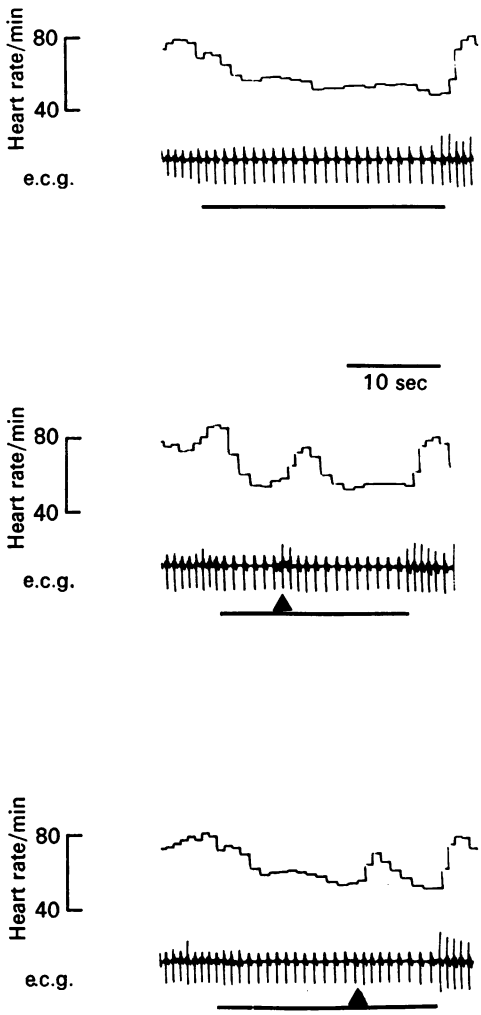

Fig. 5. Records show the heart rate and e.c.g. from a normal human subject during three periods of voluntary apnoea in which bilateral ocular pressure was applied (marker bars). The upper panel shows a control response. The middle panel shows the effect of the subject taking a 'false breath' (see text) at the filled triangle. The lower panel shows the effect of the subject swallowing at the filled triangle.

inspiratory effort indicated by the phrenic electroneurogram, the bradycardia was reduced, but it returned when the effort concluded. When the lungs were inflated within 1–2 sec to a pressure of 4–8 mmHg by an experimenter blowing into a tube connected to the tracheal cannula, this again reduced the oculo-cardiac reflex

bradycardia. The bradycardia returned within a few seconds of the air flow into the lung even when the lung inflation was maintained. Such inflations were associated with a silencing of the activity in the phrenic nerve, presumably because of the Hering–Breuer inflation reflex. In all seven dogs the lungs were then surgically denervated by cutting the left vagus nerve in the neck, and the right pulmonary vagus through a thoracic incision at the level of the fourth rib (Gandevia *et al.* 1978); this procedure denervated the lungs, but maintained cardiac vagal innervation on

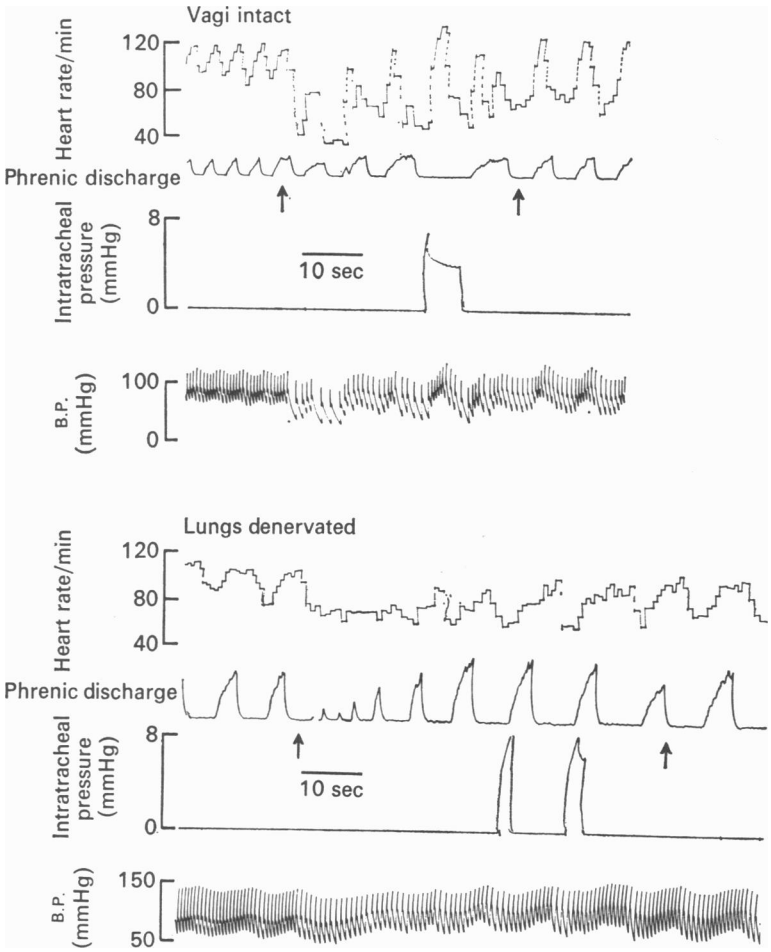

Fig. 6. Dog, anaesthetized with chloralose, paralysed with D-tubocurarine. Records of heart rate, integrated phrenic neural activity, intratracheal pressure and arterial blood pressure, obtained during periods of temporary cessation of artificial ventilation, are shown. Nasopharyngeal stimulation was applied between the arrows. In the upper panel the response to nasopharyngeal stimulation is seen: there is a pronounced bradycardia which is reduced on each occasion that inspiratory (phrenic) activity occurs. The bradycardia is also reduced as the lungs are inflated but returns while the inflation is maintained. In the lower panel the response to nasopharyngeal stimulation is seen after surgical denervation of the lungs; bradycardia is again evoked but reduced on each occasion that inspiratory activity occurs. The bradycardia is no longer reduced by lung inflation.

the right side. Repeating the initial experiment after pulmonary denervation showed that oculo-cardiac reflex bradycardia still occurred and was still reduced with each inspiratory effort, but was no longer reduced by lung inflation. A complete set of findings from a typical experiment are shown in Fig. 4.

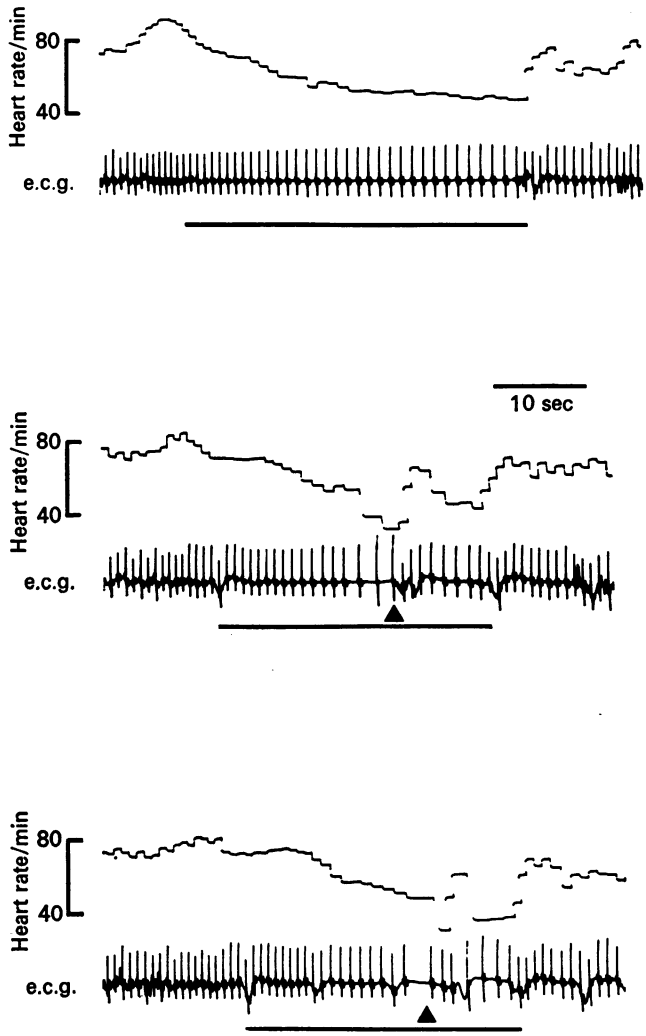

Fig. 7. Records show the heart rate and e.c.g. from a normal human subject during three periods of voluntary apnoea in which the face and mouth were immersed in water (marker bars). The upper panel shows a control response. The middle panel shows the effect of the subject taking a 'false breath' at the filled triangle. The lower panel shows the effect of the subject swallowing at the filled triangle.

Human experiments. Oculo-cardiac reflex bradycardia was evoked on many occasions in each of eight normal volunteer subjects. The bradycardia was easier to demonstrate and was more marked during breath holding. This finding is consistent with the observations in the dog, reported above (e.g. Fig. 2). We found no consistent

difference in the magnitude of the bradycardia evoked when breath-holding was performed in the inspiratory or expiratory position.

The oculo-cardiac reflex bradycardia was reduced whenever a subject took a breath, despite the continued application of pressure to the eyes. The bradycardia was also reduced if the subject made a 'false breath', or it the subject swallowed. Fig. 5 shows results from a typical subject.

Nasopharyngeal stimulation

Experiments similar to those on the oculo-cardiac reflex described above were performed in ten dogs. The passage of cold water through the nasopharynx was associated with a pronounced bradycardia which was reduced with each inspiratory effort, and enhanced in periods of Hering–Breuer inflation apnoea.

Seven of the dogs were paralysed with D-tubocurarine, artificially ventilated on oxygen, and then tested with nasopharyngeal stimulation in periods during which the pump was temporarily stopped. In these animals nasopharyngeal stimulation evoked a prompt reflex bradycardia which was reduced on each occasion that the phrenic electroneurogram indicated inspiratory activity. The bradycardia was also reduced by the inflation of the lungs (to 4–8 mmHg within 1–2 sec), although the bradycardia returned despite maintained static inflation of the lungs (see Fig. 6). Inflation of the lungs in this way was associated with silencing of the phrenic electroneurogram, presumably because of a Hering–Breuer inflation apnoea. Denervation of the lungs abolished the effects of lung inflation.

Diving

Eight normal volunteer subjects participated in many 'dives' in which they immersed the nose and mouth in a bowl of cold water. Each dive was associated with a reflex bradycardia, and we found no consistent differences in the responses to dives in which the breath was held in inspiration and those in which the breath was held in expiration. Most subjects normally swallow at the moment of immersing the face in water and find it most difficult to prevent themselves from doing this (Ebbecke, 1943). Diving bradycardia came on more slowly in dives which commenced with a swallow and was reduced whenever a subject took a 'false breath', or swallowed. Fig. 7 shows results from one subject.

DISCUSSION

It is known that stimulation of the carotid chemoreceptors or baroreceptors can evoke reflex bradycardia, but that such reflex effects are wholly or partly blocked during the inspiratory phase of breathing by central neural inspiratory mechanisms (Koepchen *et al.* 1961; Davidson *et al.* 1976) and by the central actions of sensory nerves from the lungs (Gandevia *et al.* 1978). In the present study we examined reflex bradycardia evoked by other stimuli and found that it, too, could be reduced by central neural inspiratory activity or by the actions of sensory nerves from the lungs. The central and reflex effects of breathing upon mechanisms which slow the heart appear, therefore, to be generally effective and not to be specifically confined to baroreceptor and chemoreceptor reflexes. It may be, however, that the reflexes we have examined here act by augmenting either the baroreceptor or chemoreceptor

reflexes, so that the generality of the effect may be only apparent. It has been suggested, for instance, that diving bradycardia occurs through a facilitation of arterial chemoreceptor reflexes (e.g. Jones & Purves, 1970; Daly, Elsner & Angell James, 1977) or of baroreceptor reflexes (Andersen & Blix, 1974).

The mechanisms by which the respiratory modifications of reflex bradycardia act were considered fully in the preceding paper (Gandevia *et al.* 1978), and the analysis offered there requires no elaboration in the light of the present results. The most clear-cut experiments were those performed in paralysed dogs (see Figs. 4 and 6), in which it was shown that bradycardia was reduced during the discharge of central inspiratory neurones unaccompanied by any movement of the lungs, or during air flow *into* (but not *out of*) the lungs in the absence of central inspiratory drive activity. Denervation of the lungs established that the effects of air flow were mediated by sensory nerves from the lungs, and the observation that the reduction in bradycardia was not sustained during a static inflation suggests that the intrapulmonary receptors responsible had a considerable phasic sensitivity.

The oculo-cardiac reflex was first described by Aschner (1908) and Dagnini (1908) and is evoked by pressure on the eyeball or traction on the extrinsic muscles of the eyes. It has its afferent limb in the trigeminal nerve and efferent limbs in the vagus and (as shown here) sympathetic nerves. The reflex is of obscure significance and was used here because it provides a mechanism for evoking bradycardia which is not apparently dependent upon arterial baroreceptor or chemoreceptor reflexes. The reflex is sometimes used by the physician attempting to terminate supraventricular tachycardia and our observations suggest that the manoeuvre will be more likely to succeed if the patient is asked to hold his breath while it is performed. Aserinsky & De Bias (1963) reported that oculo-cardiac reflex bradycardia is suppressed by artificial ventilation, a finding which we would attribute to the phasic activity of intrapulmonary receptors. As severe bradycardia or even cardiac arrest attributable to the oculo-cardiac reflex can occur during ophthalmic surgery (Katz & Bigger, 1970), a precaution suggested by these observations would be for anaesthetists to increase the frequency of lung inflations during surgical manipulations of the eye.

The reflexes elicited by diving in man and by nasopharyngeal stimulation in the dog are similar (Angell James & Daly, 1972). It is known that diving bradycardia in man (Daly & Angell James, 1975) and in the duck (Andersen, 1963) is reduced when a breath is taken, even if the inspired gas does not alter the blood gas tensions. Our experiments suggest that the reduction is brought about by both central inspiratory and phasic pulmonary afferent mechanisms. Recent studies by Bamford & Jones (1976) in the duck lead also to this conclusion. In the episodes of diving and oculo-cardiac reflex bradycardia in which 'false breaths' were taken (Figs. 5 and 7), both mechanisms could have been operating, although the pulmonary distortion was presumably much less than would have occurred during a normal breath. As with the oculo-cardiac reflex, there are known to be hazards in surgical procedures from cardiac arrhythmias attributable to reflexes from nasopharyngeal and endotracheal stimulation (Katz & Bigger, 1970), and again the present findings suggest that more frequent lung inflations imposed during such procedures should give some protection.

Swallowing was a manoeuvre which was used in an attempt to activate central respiratory neurones without causing an accompanying distortion of the lungs. It is

known that many medullary neurones which discharge in association with inspiration also discharge at the commencement of a swallow (Hukuhara & Okada, 1956; Sumi, 1963). The same neurones discharge when a stimulus known to induce swallowing is presented in a paralysed animal (Sumi, 1963). These inspiratory neuronal discharges may be associated with the very slight inspiratory effort ('Schluckatmung') which is a characteristic early event in swallowing (Doty, 1968). The association of tachycardia with swallowing was observed by Miller & Sherrington (1916) and has been demonstrated here (Fig. 1). Central inspiratory neural activity accompanies swallowing, and we have shown here that similar activity reduces bradycardia. It follows, as was shown in Figs. 5 and 7, that swallowing reduces oculo-cardiac or diving reflex bradycardia. Attempts were also made, in many experiments on three domestic ducks, to look at the effects of swallowing on diving bradycardia in these diving animals but they could not be induced to swallow while diving.

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