are calculated. We again see that turnover rates are approximately 3 times as high in midgut gland, gut and stomach compared to the rest animal.

For comparison, respiration rates for different organs for the terrestrial pulmonate snail *Helix pomatia*, as presented by other authors[6], are summarized also in Table II, column 3. Respiration rate obviously goes more or less parallel to carbon turnover rate. Organs of the pallial complex show about 3 times higher a respiration rate than the rest animal and reproductive organs show again a rather low value. The incorporation and turnover rates of the organs correspond to some extent to the results for rats and mice: the highest being in liver, then kidney, spleen, lung, muscle, and the lowest in the brain. In crustaceans (*Orconectes limosus*), the succession: gut, midgut gland, stomach, muscle was found, when ^{14}C-glucose was injected into the hemolymph[7].

If half-life times of the midgut gland, stomach and gut of *Ancylus fluviatilis* are converted to mammal temperature of 38 °C by assuming a Q_{10} of 2, values of 2.3–5.3 days are found, which nearly equal values for mice and for *Orconectes* when converted[3]. On the other hand, respiration rates are about 2500 µl O_2/g · h for mice, 40 µl O_2/g · h for *Orconectes*[3] and 200 µl O_2/g · h for *Ancylus fluviatilis*[8], the latter both at 18 °C. Assuming again a Q_{10} of 2, mice have a respiration rate about 15 times as high a *Orconectes* and about 3 times as high as *Ancylus*. Such calculations have to be regarded with caution, as neither invertebrate can exist at 38 °C and any assumed value of Q_{10} is arbitrary. But nevertheless it now seems to be a general phenomenon that invertebrates, although showing lower respiration rates than mammals, have quite comparable incorporation and carbon turnover rates, if converted to the same temperature.

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Carotid Chemoreceptor Influence on the Cardiac Sympathetic Nerve Discharge

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Summary. Pure stimulation of carotid chemoreceptors induces, as a primary effect, the general spontaneous activation of both the cardioexcitatory and the cardioinhibitory medullary centres. These effects have been shown by keeping constant those parameters which, when modified, produced secondary effects hiding the investigated primary ones.

Chemoreceptor stimulation of carotid glomus provokes in spontaneously breathing animals an increased activation of the respiratory centres, hypertension and tachycardia[1,2]. When the animals are thoracotomized or curarized and artificially ventilated, the same chemoreceptor stimulus results in bradycardia[3–5]. It is generally accepted that the tachycardia observed in the first experimental condition is ascribable to the increase of ventilation provoked by the chemoreceptor stimulus, which is to say that the tachycardia is a secondary response to the increase of pulmonary stretch receptor activity[4,6,7]. The primary effect of carotid chemoreceptor stimulation on the heart should consist in a decrease of its frequency: such a bradycardic effect appears unmasked if the animal is not allowed to increase its ventilation.

As far as the mechanisms of this bradycardia are concerned, beside the well defined increase of the vagal activity[8–11], there is still some disagreement as to whether there is any participation of the cardiac sympathetic activity in this response. Among others, DOWNING and SIEGEL[12] affirm that the cardiac sympathetic nerve does not show any contribution, while ALANÌS et al.[13] have described experiments in which a decrease, an increase or no change in the cardiac sympathetic activity occurred.

The present experiments have been undertaken in order to analyze the role of the cardiac sympathetic nerve in this reflex bradycardia.

Methods. The experiments were performed on 20 cats anaesthetized with urethane (250 mg/kg) and chloralose (30 mg/kg), thoracotomized along the midline and artificially ventilated. The arterial blood pressure was recorded from right subclavian artery. The electric activity from the central cut end of the inferior cardiac nerve and of the phrenic nerve was recorded by means of an AC preamplifier and an integrator following the technique of ALANÌS et al.[13]. Tracheal CO_2% was continuously monitored and the ventilation was adjusted as to keep the end-tidal CO_2% at the control level throughout the experiment. When required, the cat's arterial blood pressure was kept constant by connecting the abdominal aorta (at the bifurcation) to a large plastic tube which led to a big reservoir containing buffered saline solution. The pressure in the reservoir was fixed at the same level as the mean blood pressure the animal exhibited just before each trial. The heart was paced, when necessary, by electrical impulses at a suitable frequency, delivered through a couple of ring-shaped platinum electrodes fixed to the anterior wall of the right atrium. NaCN solution at a concentration of 50 µg/ml was injected by means of an infusion pump, through the cannulated thyroid artery.

Results and discussion. The NaCN injection in thoracotomized-artificially ventilated cats (Figure A) evoked the usual increase in phrenic discharge. At the same time the sympathetic discharge exhibited an abrupt, short-lasting increase followed by modifications that were synchronous

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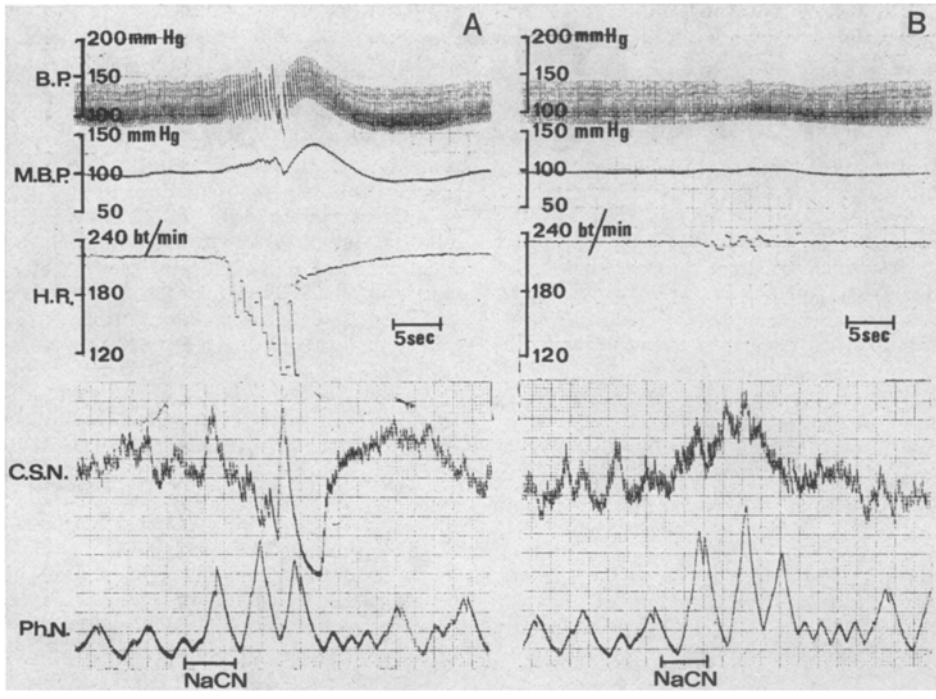

Effect of carotid chemoreceptor stimulation on the cardiac sympathetic nerve discharge. At the signal 0.2 ml of a NaCN solution (50 μg/ml) was injected through the right thyroid artery. Cat: urethane (250 mg/kg) and chloralose (30 mg/kg) anaesthesia, open chest, artificial ventilation. A) control; B) heart electrically paced, blood pressure kept artificially constant. Traces from above: arterial blood pressure (B.P.), mean arterial pressure (M.B.P.) heart rate in beats per min (H.R.), integrated discharge activity of the right cardiac sympathetic nerve (C.S.N.) and of the right phrenic nerve (Ph. N.).

and opposite-sign to those of the arterial pressure. We believe the first abrupt increase of the sympathetic discharge was due to the effect of the chemoreceptor stimulation; subsequently such an effect was hidden and counteracted by the pressoreceptor influences, every time there was a change in the arterial systemic blood pressure level.

In a successive trial (Figure B), both the arterial blood pressure and the heart frequency were kept constant (see methods) in order to make evident the pure effect of the same chemoreceptor stimulation on the cardiac sympathetic activity. In this situation, the sympathetic discharge showed a significant increase, lasting approximately the same time as the respiratory activation.

The comparison of the two trials shows that the phase of bradycardia shown in Figure A and the increase in sympathetic nerve discharge observed in B were both happening at the same latency from the cyanide injection and synchronously with the increase of the phrenic discharge. This means that, during carotid chemoreceptor stimulation, both parasympathetic and sympathetic supply to the heart were activated at the same time.

At the end of the experiments, the Hering nerve ipsilateral to the side of NaCN injection was cut. Baroceptor and chemoreceptor activity was recorded from the peripheral end of the nerve. The NaCN administration produced a clear increase of chemoreceptor activity and did not modify the baroreceptor discharge; the same injection had no effect whatsoever on blood pressure, heart rate and respiratory activity. This proves that the effects observed on these parameters before cutting the Hering nerve were due to the pure stimulation of carotid chemoreceptors, and neither direct stimulation of central structures nor of other chemoreceptor areas was involved.

These data show clearly that carotid chemoreceptor stimulation produces a general activation of both the cardioactivatory and the cardioinhibitory medullary centres.

Retinal Blur and Midbrain Cell Response[1]

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Summary. The responses of certain photically responsive cells of the superior colliculus were found acutely dependent on the focal condition of the retinal image.

Although refractive errors are by far the most common anomalies of the visual system[2], little is known of their effects directly on visual pathway coding. An approach explored here was to induce, through refractive error, known degrees of retinal image blur and to compare the resulting pathway responses with those associated with optically best (i.e., no refractive error) conditions.

Material and method. The responses cited are from among a cumulative sample of more than 300 neurons now studied within the rabbit mesencephalon and visual

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