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EFFECTS OF INTRANASAL APPLICATION OF THE LOCAL ANAESTHETIC XYLOCAINE ON VESTIBULO-, OPTO-COLLIC AND POSTURAL REFLEXES OF THE HEAD, AND ON TONIC IMMOBILITY IN HOMING PIGEONS: IMPLICATIONS FOR EXPERIMENTS ON PIGEON HOMING

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Summary

Spraying lidocaine (Xylocaine), a local anaesthetic, into the nasal cavity of homing pigeons is a technique widely used to study the role of olfaction in pigeon homing. Studies in the laboratory revealed that Xylocaine suppressed vestibular and optokinetic nystagmus, roughly for as long as it suppressed olfaction, interfered with control of the head posture when the body was tilted in the sagittal plane, and prolonged tonic immobility. Amplitude and duration of both optokinetic and vestibular nystagmus were affected to the same extent. The effects are most probably caused by the drug's rapid absorption *via* the mucous membranes in the nasal cavity and its transport in the blood to brain structures involved in integrating vestibular and visual inputs. The results provide room for non-olfactory explanations of the manifold effects on pigeon homing following administration of local anaesthetics to the nasal chambers.

Introduction

Since Papi *et al.* (1971) hypothesized that olfaction is the basic sensory mechanism underlying pigeon navigation, the involvement of olfaction in pigeon homing has been investigated by various methods of olfactory manipulation (see Papi, 1986, for a review).

Because lidocaine (Xylocaine=XC) is able to produce a reliable and reversible nerve block, it has been widely used in medicine and dentistry since its introduction by Löfgren (1948). Schmidt-Koenig and Phillips (1978) and Dornfeldt (1979) were the first to study pigeon homing by administering a local anaesthetic

Key words: Xylocaine, lidocaine, intranasal anaesthesia, vestibulo-collic reflexes, opto-collic reflexes, pigeon homing, tonic immobility.

(XC) to the nasal chambers. Subsequently, other authors also rendered homing pigeons anosmatic by applying local anaesthetics (XC and Gingicaine) to the nasal cavity. Either initial orientation or homing performance or both were affected. This was true especially in studies performed in Italy, whereas effects were less consistent or even nonexistent in studies performed in Germany (see Papi, 1986; Wiltschko and Wiltschko, 1989). Wiltschko *et al.* (1986, 1987*a*,*b*) showed that the anaesthetic had significant effects on initial orientation only in particular cases, for example when the pigeons had been housed in a garden loft in Frankfurt (FRG), while there was a marked decrease in homeward orientation when the pigeons had been housed in a wind-exposed loft on the roof of a 19-m high building nearby (Wiltschko and Wiltschko, 1989). There was consistently a significant decline in homing speeds of the pigeons from both lofts. The effects induced by XC were indistinguishable from those induced by Gingicaine.

After releasing the homing pigeons, one of us (KD) studied the possible non-olfactory side-effects of intranasal anaesthesia. Sometimes pigeons treated with XC were unable to maintain their upright position in the normal manner and had an unusually relaxed body musculature. A greater portion of the pigeons (just above the 5% significance level) compared with untreated controls landed after release within view of the releaser. Similarly, Wiltschko *et al.* (1987a) reported that anaesthetized birds flew low and landed within view more frequently than did untreated ones.

Direct experiments revealing such effects have not been performed, although this technique has become established as a standard tool in pigeon homing research. In an attempt to fill this gap, we focused our attention on the following questions. (1) Does intranasally applied XC exert any effects on compensatory turning movements of the head in response to passive rotations of the pigeon (vestibular nystagmus)? (2) Does XC affect tracking-saccadic head movements in response to rotations of large-field visual stimuli (optokinetic nystagmus)? (3) Does XC influence the control of head posture? (4) Does XC affect tonic immobility, which is a useful index of fear (Gallup, 1977; Jones, 1986)? All questions are answered affirmatively; the first three questions are probably relevant for course control while flying home; the fourth question allows an assessment of the pigeon's general well-being.

Materials and methods

Animals

The homing pigeons used in the experiments were housed in the loft of the Zoological Institute of the University of Göttingen. They were allowed to feed, drink and fly out *ad libitum*. They were 1–4 years old and experienced in homing.

Application of XC

A normal spray push (i.e. about 10 mg of XC containing 1.04 mg of lidocaine hydrochloride as the active agent, 0.001 mg of cetylpyridinium chloride, and a

propellant) was sprayed through the cannula of the commercial spray bottle (Astra Chemicals) up each nostril into the nasal cavity (insertion depth about 5 mm).

Device for stimulation

The pigeon was placed in a 'normal' (horizontal) position on a turntable surrounded by a cylinder with black and white stripes of 7° width (as described by Bilo, 1977). The wings were fixed with adhesive tape. The rotational axis was vertical and approximately between both labyrinths. The rotational stimulus consisted of constant positive (beginning) and negative (ending) acceleration and an intermediate phase of zero acceleration. The rotations were produced by a velocity servo system (Mattke) controlled by ramp–plateau–ramp-like command signals from a function generator. For optokinetic stimulation, the striped cylinder was rotated around a vertical axis: this was also electronically controlled.

Recording and quantification of muscle activity

Electrical activity of neck muscles was recorded by a standard technique (see Bilo, 1977). Muscular activity was quantified by a diode rectifying the electromyograms, involving low-pass filtering by an RC-element (time constant 0.17 s). In two series of experiments all muscular responses were integrated over a time interval of 20 s by an RC-element with an extremely high time constant. Thus, the discharge of the capacitor during integration was negligible. The response integral depended on the amplitude, frequency and duration of the post-rotatory nystagmus.

Registration of head movement

The frequency of the head movements was quantitatively determined by a photoresistor measuring the light reflected by a segmented paper corona glued to the pigeon's head. The amplitude of head movement was only qualitatively determined.

Fixation of the head to the turntable

A segment of a metal sheet was glued to the skull with Uhu and attached with a clamp to the turntable, keeping the head in the normal position. This and the preparation for recording muscular activities were performed under Fluothane anaesthesia (Bilo *et al.* 1972).

Experiments on the control of static head posture

The pigeon's legs were fixed in a cleft of a sitting block and its wings were fixed with adhesive tape. A paper pointer was glued to the head. The bird on the block, which was mounted in front of a vertical disc with scale units of 1°, was manually tilted about a transverse horizontal axis roughly through the body's centre of gravity. The succession of angular positions was −20°, 20°, −40°, 40°, −60°, 60°, −80°, 80° (− counterclockwise; + clockwise). It was repeated twice for each bird

(eight pigeons). Except when the position of the sitting block was being changed, the bird was kept in darkness. One minute after tilting the pigeon on the block, a flash photograph was taken. XC was then applied (see above) and 5 min later three further measurements in the same body position were carried out.

The negatives were projected onto a screen by a slide projector. Body and head positions were measured on this screen. The angle of the pointer with respect to the horizontal axis in the first measurement on the untreated pigeon (i.e. body position=0°) was subtracted from all the other angles measured on the same animal (see sketch in Fig. 5). The difference in the mean head position of each pigeon before and after application of XC was tested using the paired *t*-test.

Measurement of tonic immobility

The experimenter firmly grasped the pigeon with both hands, turned it upside-down and pressed it for 10 s onto the bottom of a cardboard box (50 cm×50 cm×60 cm, external illumination through a window made of translucent parchment with a 60 W bulb). The experimenter then cautiously withdrew his hands and covered the top of the box with a plate on which a camera was mounted in a hole. The pigeon was observed through the camera but could not see the experimenter. The time (in seconds) from withdrawal of the hands until the pigeon rose to its feet was measured with a watch. This period, commonly referred to as tonic immobility time, was determined in 18 homing pigeons (11 males, 7 females) selected at random in the loft; each pigeon was measured in a random sequence on consecutive days between 09:00 h and 12:00 h, once under the control condition (i.e. without treatment) and once under the drug's influence.

Results

Effect of XC on the vestibular head nystagmus

Head freely movable

Fig. 1 shows records selected from a series of experiments with pigeon D204. Fig. 3A shows the discharge integrals of the neck muscles on the right side for this bird. These result from summing the action potentials of the muscles over an interval starting at the onset of the deceleration of the turntable and ending 10 s later.

Response of the untreated pigeon. During non-negative acceleration of the rotation, the pigeon attempted to maintain its prior direction of view. This activity, commonly called vestibular head nystagmus, consists of two phases occurring serially and alternately: slow, smooth, counter-rotational movements and fast head turnings in the direction of rotation, overtaking the rotation. Because of the relatively high velocity and the recording technique used, the records largely consist of fast movements declining in frequency; the slower movements cannot be seen distinctly in the initial part of the rotation.

During rightward rotation, head nystagmus was associated with discharges of the neck musculature on the left side. A short time after the onset of the rotationa

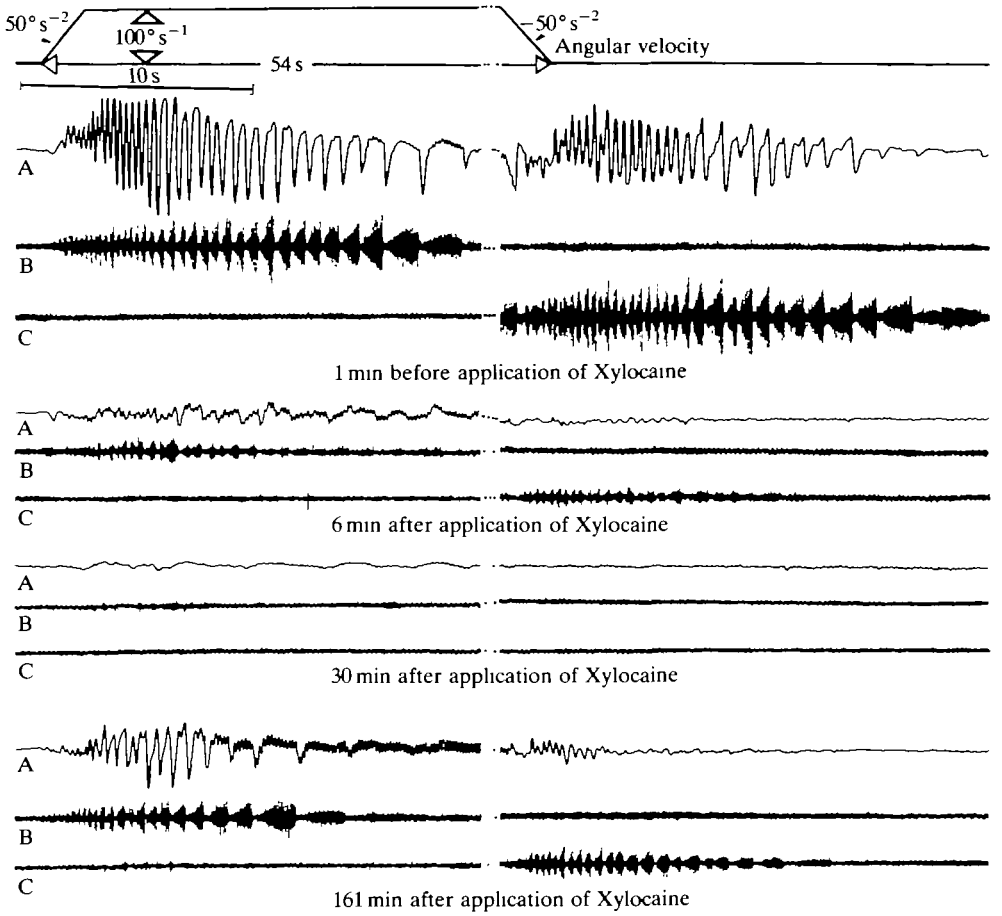

Fig. 1. Vestibular head nystagmus before and after application of Xylocaine in pigeon D204 with freely movable head. Top row: rightward rotation stimulus. (A) Photocurrent records of the head movement (the superimposed tremor-like signal is an artefact); (B) electromyogram of the neck musculature of the left side; (C) electromyogram of the neck musculature of the right side.

deceleration, similar movements of the head were recorded with declining frequency. These are referred to as post-rotatory vestibular head nystagmus. They were associated with discharges on the right side similar to those previously recorded from the left side during rotation. As for the head nystagmus, the frequency of the underlying discharges of the neck muscles was highest at the beginning of the rotational acceleration and deceleration; thereafter it declined. Initially, the amplitudes of the head movements and that of the gross muscular discharges increased to a maximum; they then decreased to nearly zero towards the end of the period.

Response of the treated pigeon. Six minutes after the first application of XC the amplitude of both head movements and of gross discharges of the neck muscles

drastically decreased. The nystagmus and the activity of the neck muscles were less regular and of shorter duration. Ten minutes after application of XC the nystagmus disappeared entirely. Gradual recovery began 90 min later (see Fig. 3A).

Fig. 1 shows recordings 6, 30 and 161 min after application of XC. The nystagmus was still suppressed after 30 min. It reappeared after 161 min, but was less pronounced than at the onset of the experiment. Since the head nystagmus and the relevant activity of the neck musculature was also becoming smaller in the controls during the course of this experiment, presumably because of a habituation effect, a precise determination of the duration of the effect of XC was not possible. In the experiment under consideration, the effect lasted for at least 100 min (see also below).

To establish another control condition, we injected tap water through both nostrils 184 min after administration of XC. The nystagmic activity decreased for only a few minutes and the effect did not resemble the drastic effect produced by XC.

Head fixed to the turntable

Pigeons treated with the anaesthetic often kept their beaks higher than controls (see Figs 5, 6). Since the head nystagmus depends on how the pigeon holds its head with respect to the sagittal plane, the head was fixed to the turntable in its 'normal' position.

Figs 2 and 3B show the results obtained with pigeon D570.

Response of the untreated pigeon. The amplitude of the rhythmic activity of the neck muscles (termed 'nystagmus') is similar to that of pigeon D204, though the frequency is markedly higher. As the frequency of the nystagmus in pigeons with freely movable heads was less than in those with fixed heads (D. Bilo, unpublished data), we conclude that the higher frequency was due to the head's fixation. Unfortunately, only one bird was tested; hence, no definitive conclusion is possible.

Response of the treated pigeon. The effect of XC in this experiment resembled that in the experiment described above, though it differed in some respects. Following application of XC the nystagmus did not disappear completely. The integral of the post-rotatory response dropped to about 30 % of the initial value and, within an hour, it returned to 43 % of the initial measurement (Fig. 3B). When the treatment was repeated, the response integral dropped to 40 % of its value prior to treatment, but recovered to 90 % within an hour. After the third application, the integral declined to 34 % of its initial value, rising to 85 % after 60 min. Using tap water, instead of XC, was not effective in this pigeon.

Fig. 2. Effect of Xylocaine on the muscle activity associated with vestibular head nystagmus of pigeon D570 with head fixed to the turntable. Top row: rightward rotation stimulus. Electromyograms recorded from the neck musculature of the left side during rotation prior to deceleration and electromyograms recorded from the neck musculature of the right side thereafter are separated from each other by dotted lines.

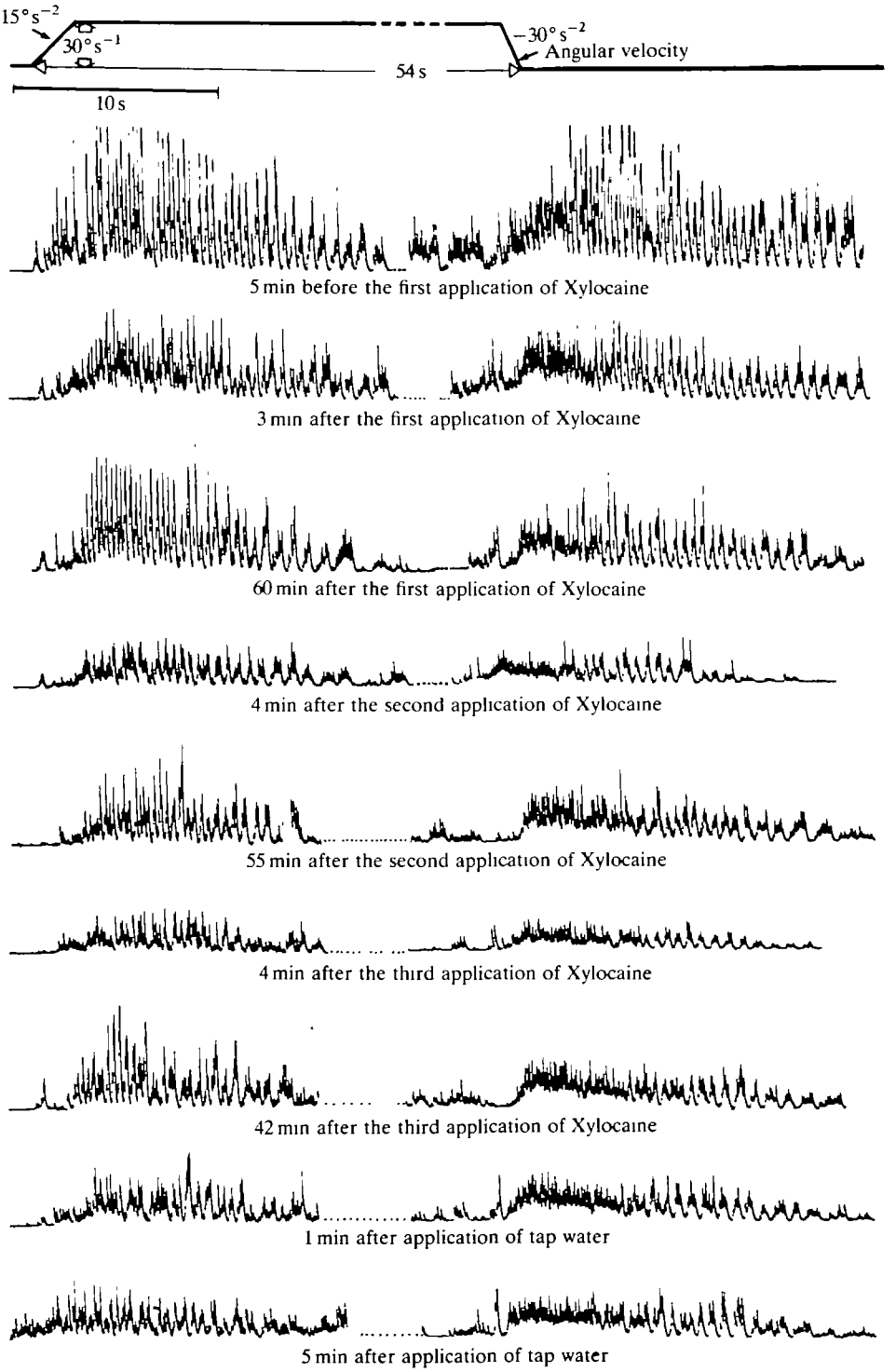
15°s⁻²
30°s⁻¹
−30°s⁻²
Angular velocity
54 s
10 s
5 min before the first application of Xylocaine
3 min after the first application of Xylocaine
60 min after the first application of Xylocaine
4 min after the second application of Xylocaine
55 min after the second application of Xylocaine
4 min after the third application of Xylocaine
42 min after the third application of Xylocaine
1 min after application of tap water
5 min after application of tap water

Fig. 2

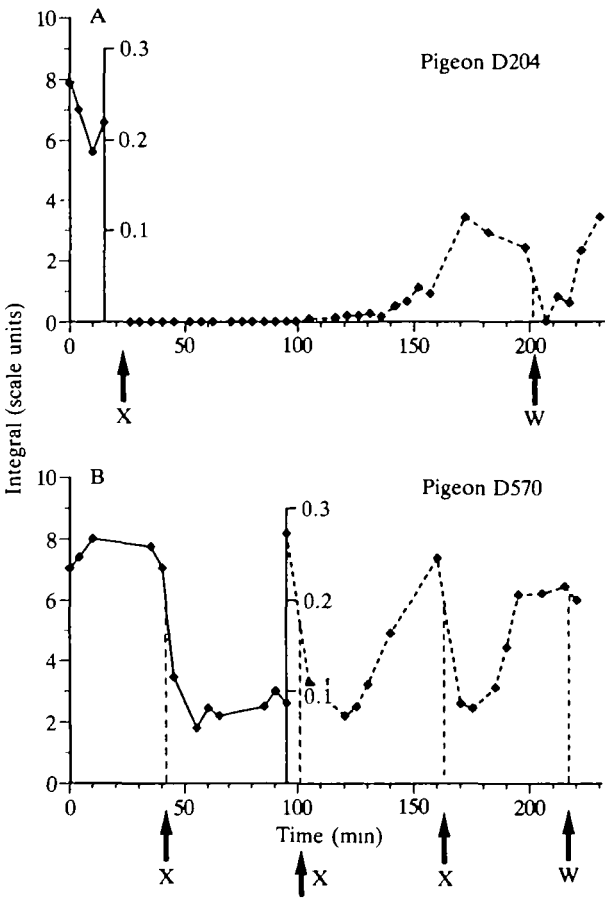

Fig. 3. Integrals of post-rotatory electromyograms of the right-side neck muscles associated with vestibular head nystagmus in response to one rotation stimulus as shown in the top rows of Figs 1 and 2 for two pigeons: (A) D204, (B) D570. Ordinate scales are arbitrary, linear and at 15 min (A) and 95 min (B), the ordinate scales were magnified 30 times for clearer demonstration of the effect. X, application of Xylocaine; W, tap water injection.

Effect of XC on the optokinetic head nystagmus

The responses to optokinetic stimuli (amplitude $30°s^{-1}$, duration 54 s) were investigated only in pigeons with fixed heads.

Response of the untreated pigeon

When the striped cylinder was rotated clockwise (or counterclockwise) the right (or left) muscles in the neck were rhythmically active. The nystagmus reached its full strength after some seconds. Its amplitude varied considerably and decreased gradually during continued stimulation.

In contrast to the vestibular nystagmus, the optokinetic nystagmus lasted only a few seconds, and the left-hand neck muscles remained inactive during and after

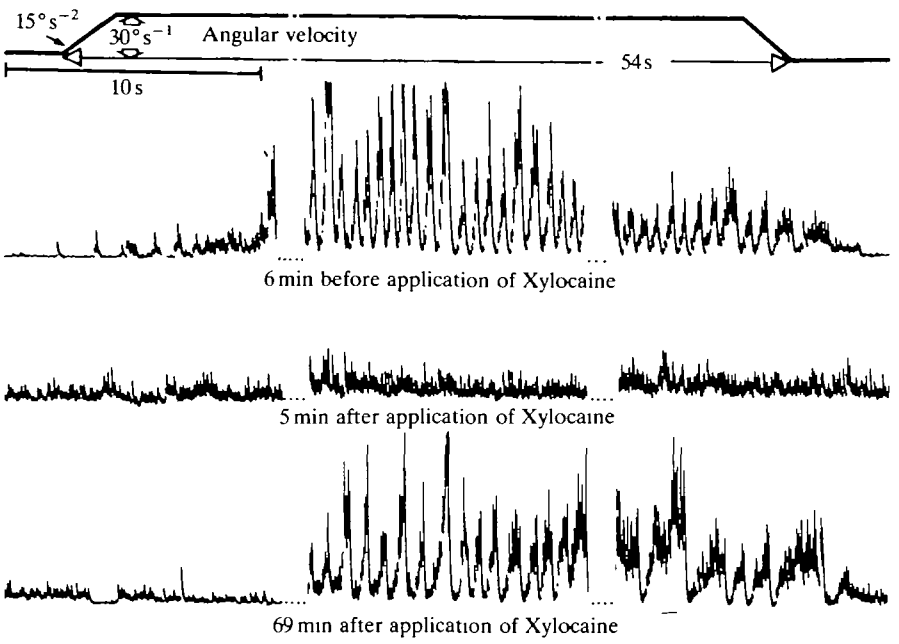

Fig. 4. Effect of Xylocaine on optokinetic head nystagmus. Top row: rightward rotation of the striped cylinder. Below: electromyograms of the neck musculature on the right side. The records are typical examples selected from experiments on pigeon D598 with its head fixed.

rightward rotation. The discharges of the neck muscles on the right side continued for some seconds after cessation of stimulation.

Response of the treated pigeon

XC affected the optokinetic and the vestibular nystagmus similarly. The effect was, however, difficult to assess because both normal and treated pigeons became sleepy, so we had to prevent them from falling asleep (i.e. closing their eyes) by clapping our hands.

Table 1 contains data characterizing the effect of XC on the optokinetic nystagmus in three pigeons. Fig. 4 shows recordings selected from the series conducted with pigeon D598.

Duration of the effect of XC

In the cases in which the nystagmus had been completely suppressed, the minimum duration Δt_{min} of the effect was determined (see Table 1 for definition of Δt_{min}). The mean of the six values from three pigeons was 34±4 min (S.D.).

For the vestibular nystagmus only two values of Δt_{min} were determined: pigeon D204, Δt_{min}=84 min; pigeon D598, Δt_{min}=103 min. The minimum duration of the effect seemed to be larger in vestibular than in optokinetic nystagmus. The duration of the effect was independent of the number of prior XC applications.

Table 1. *Effect of Xylocaine on the optokinetic nystagmus*

Pigeon	t (min)	Δt_{min} (min)	Description of activity
D504	0		Intense nystagmus
	4		First XC application
	8		No nystagmus
	41	33	Slight nystagmus
	96		Intense nystagmus
	112		Second XC application
	116		Slight nystagmus
	120	47	No nystagmus
	163		Slight nystagmus
	197		Intense nystagmus, amplitude larger than before last application
	221		Third XC application
	225		Slight nystagmus
	232	37	No nystagmus
	262		Slight nystagmus
D588	0		Intense nystagmus
	4		First XC application
	9		Slight nystagmus
	32	36	No nystagmus
	45		Slight nystagmus
	71		Intense nystagmus
	75		Control experiment with tap water
	78		Slight nystagmus
	86		Intense nystagmus
	90		Second XC application
	94		No nystagmus
	115		No nystagmus
D598	0		Intense nystagmus
	7		First XC application
	12		No nystagmus
	23	11	Slight nystagmus
	133		Intense nystagmus
	139		Second XC application
	144		No nystagmus, basal activity level higher
	193	49	Slight nystagmus
	208		Intense nystagmus, amplitude and frequency less than before the second application

Δt_{min}, minimum time that the effect of Xylocaine (XC) lasted, i.e. the time between the first test after XC application and the reappearance of the optokinetic nystagmus.

The maximum duration of the XC effect (i.e. the time passing after the first application until the recurrence of the initial amplitude) was often not measurable because the XC effect was superimposed on a habituation effect that also reduced the amplitude in the untreated pigeons.

The maximum XC effect on optokinetic nystagmus was approximately determined for three pigeons: pigeon D598 (first application), Δt_{max}=121 min; pigeon D588 (first application), Δt_{max}=62 min; pigeon D504 (second application), Δt_{max}=81 min. The mean Δt_{max} was 90±28 min (S.D.). Thus, the XC effect persisted for about 90min. The latency of the effect was less than 4 min.

Effect of XC on the control of stationary head posture

Fig. 5 shows the head posture of eight pigeons following tilting of the body by angles ranging from −80° to +80° before and after XC application. On average, the slope of the graph is 0.7, indicating that the tilted body position was only

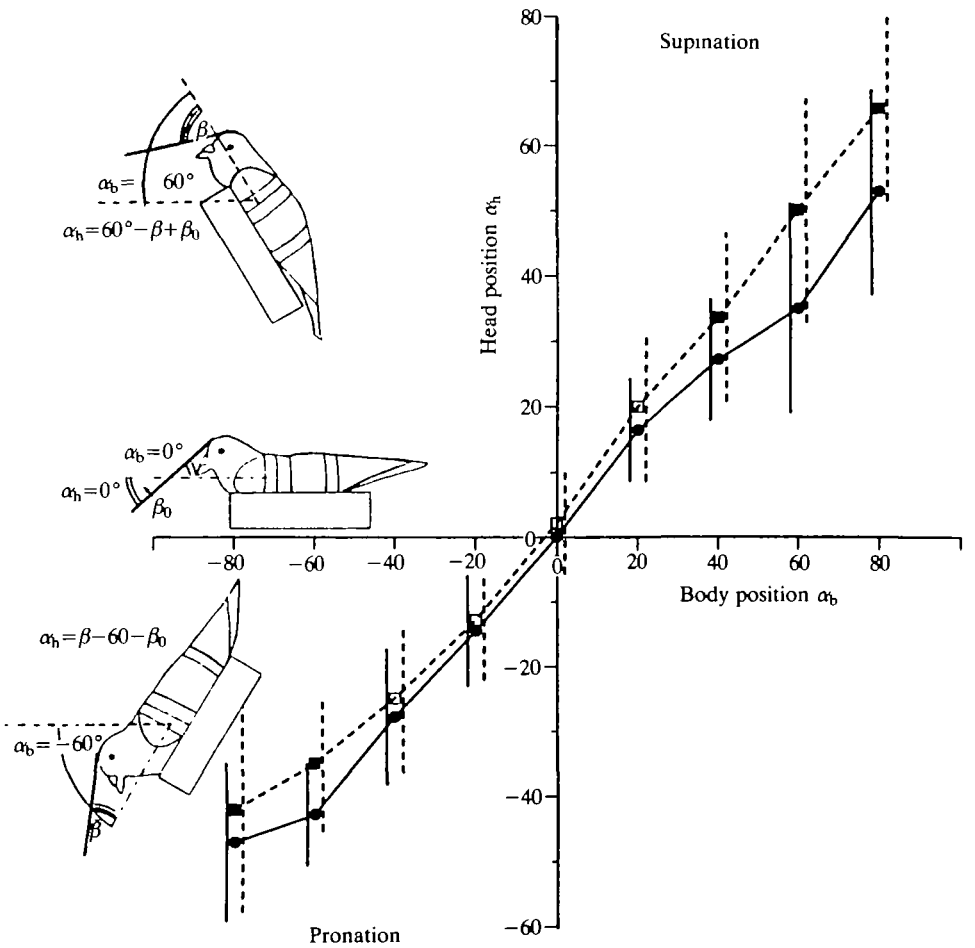

Fig. 5. Mean angle and mean angular deviation of the head position in relation to the body position after clockwise rotation (+) and counterclockwise rotation (−) around a transverse axis, before (solid line) and after (dashed line) application of Xylocaine in eight pigeons. Significant differences (at least $P<0.05$, paired t-test) are marked by closed squares. The sketch illustrates how the measured angles were transformed into the plotted ones.

slightly compensated for by moving the head. In the case of complete compensation, the line should be parallel to the abscissa, whereas the lack of any compensation would be reflected by a line with a slope of 1. The poor performance of the system controlling the stationary head posture was possibly due to the arousal state, which essentially decreases in the dark.

Effect of XC

The graph of the data obtained from treated pigeons is slightly different from that of the data obtained from controls. The following absolute differences between the means were significant: 5° and 12° ($P<0.05$), when the body was pronated (at −80° and −60°, respectively), and 7° ($P<0.05$), 15° and 13° ($P<0.01$, paired t-test), when the body was supinated (at 40°, 60° and 80°, respectively). In this series, the vestibular postural head control in the sagittal plane was improved if the body was pronated, but deteriorated if the body was supinated; the effect may be described as head rotation in the sagittal plane upwards.

The experimental design in this series does not control possible effects due to habituation or fatigue *per se*. However, the individual measurements (for instance, in two body positions: −60° and 60°; see Fig. 6) show that the curves of the head positions are roughly parallel to the abscissa, on a lower level before application of XC than after it. This demonstrates an elevated head position under the influence of the drug rather than any effects due to habituation or fatigue.

Influence of XC on tonic immobility

Of the 18 pigeons tested, 16 showed longer tonic immobility times when treated with the drug than when untreated. The median value, and first and third quartile of the sample of immobility times are 5.3, 3.7 and 10.6 min, respectively, when untreated, and 25.0, 6.3 and 144 min, respectively, when anaesthetized. The medians are significantly different (Wilcoxon matched-pairs signed-rank test: $P<0.01$).

Since every animal served as experimental as well as control in a random order, habituation, fatigue and longer-lasting after-effects of a preceding treatment with XC can be ruled out *a priori* as possible explanations of this effect.

Discussion

The data presented here show that spraying XC into the nasal cavity markedly reduced vestibular and optic control of the head, as shown by changes in head nystagmus, in head posture and in the discharge patterns of the neck musculature associated with nystagmic activity. Amplitude and duration of optokinetic and of vestibular nystagmus were all similarly affected.

The mean recovery rate (90 min) roughly matched the rates of recovery from anosmia reported in the literature on pigeon homing. XC (at one-third of the present concentration) applied intranasally through the choane eliminated

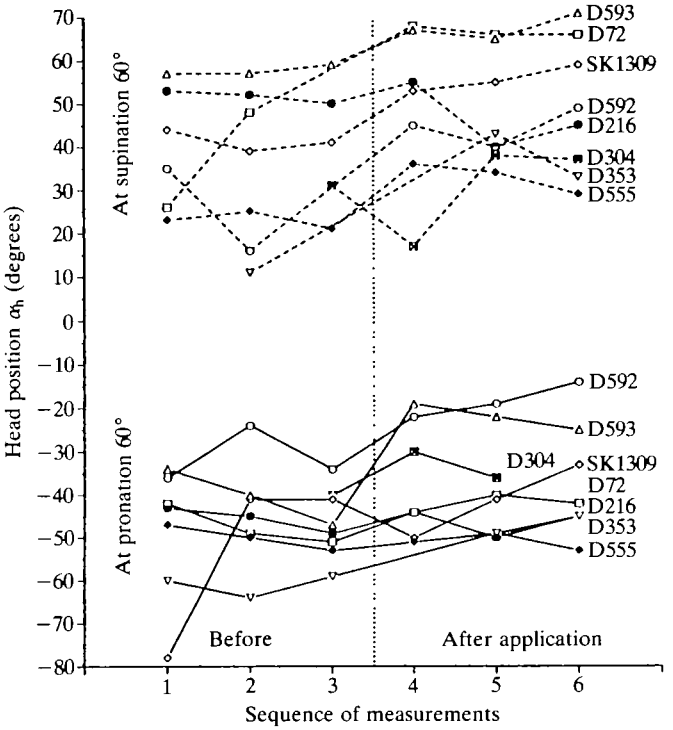

Fig. 6. Sequence of individual head positions in eight pigeons at 60° supination (top, dashed lines) and at 60° pronation (bottom, solid lines) before (left-hand side) and after (right-hand side) application of Xylocaine; angles have been defined as in the legend of Fig. 5; the missing points were when the photographs showed the bird's head out of the sagittal plane of the body.

the conditioned response to amyl acetate, a strong olfactory stimulant, in the laboratory for 60–90 min (Schmidt-Koenig and Phillips, 1978). Applying XC (at the same concentration used here) through the nostrils suppressed the unconditioned acceleration of the heart frequency in response to amyl acetate for 30–60 min (Wallraff and Foà, 1981).

The following discussion of possible reasons for the XC effect focuses on three lines of evidence reported in the literature.

First, in a variety of controlled laboratory situations, both pigeons and rodents alter some behaviour when their olfactory input is greatly diminished by olfactory bulbectomy or nerve cuttings. The effects are not readily explicable in terms of olfactory perception loss alone. Their understanding is facilitated on the basis of such concepts as emotionality, motivation and attentional behaviour; this brings to mind limbic structures and the reticular core as implicated brain sites (see Wenzel, 1974, for a review). Yet neither effects on nystagmic activities nor on postural head control as a result of olfactory manipulation have been previously reported.

Second, the trigeminal and the olfactory nerves innervate the nasal cavity in

overlapping areas. The ophthalmic nerve, part of the trigeminal nerve system, is the principal sensory nerve in the ocular orbit and the nasal cavity (see Bubien-Waluszewska, 1981, for anatomical details). Blocking these nerve branches possibly affects oculomotor functions. As magnetic fields affect pigeon navigation only while birds can smell atmospheric odours (Wallraff *et al.* 1986), odour perception and magnetic sensibility may be linked to each other in an unknown way, so that the loss of the former capability might lead to cessation of the latter one as well. Alternatively, anaesthesia might interfere directly with magnetic field detection because in the bobolink, a migratory bird, responses to magnetic stimulation were recorded from the ophthalmic and supraorbital nerves (Beason and Semm, 1987). The strong effects on reflexes controlled by the vestibular system, however, seem to support the following hypothesis.

Third, local anaesthetics applied to mucous membranes produce plasma levels that, when plotted against time, simulate those attained after rapid intravenous injection (Adriani *et al.* 1985). Morbid or fatal complications after administering XC to the nasopharyngeal and laryngeal surfaces of human patients reportedly occurred far more frequently than during general surgery (Kilian, 1974). The main adverse effects of XC are on the central nervous system; they are believed to be due to blocking of the inhibitory cortical synapses (Covino, 1972).

Intravenous injection of XC leads to reversible suppression of tinnitus and to changes in the function of the vestibular system in humans. This implicates structures in the brain that are involved in processing auditory and vestibular information as possible sites of XC action (Merchant and Kirtane, 1986).

In the light of these reports we think it likely that a substantial portion of the drug was rapidly absorbed *via* the intranasal mucous membranes, hence entering the blood stream. Although it could not directly penetrate the rigid labyrinth capsule, it readily passed the blood–brain barrier, which is not an obstacle for anaesthetics (Chernick, 1976). It probably affected brain centres responsible for integrating visual and vestibular inputs, i.e. the nuclei vestibulares, the formatio reticularis and/or the cerebellum (e.g. Precht, 1978).

That anaesthetized pigeons – upon release – were sometimes incapable of maintaining their erect position in the cage calls to mind such symptoms in humans suffering from sea-sickness or other motion-induced illnesses caused by a mismatch of vestibular signals from both ears. Particularly at higher ambient temperature (>27°C), the body musculature in pigeons treated with XC seemed to be relaxed. This is probably caused by a decrease in spontaneous activity in the vestibular apparatus, which is responsible for maintaining and regulating the body muscle tonus. Thus, we suggest that both the above effects are functionally linked to the interference with the vestibular system.

The methodology of intranasal anaesthesia has been studied in rabbits and pigeons. In rabbits, intranasally applied lidocaine proved to be less toxic than other local anaesthetics (Aström and Persson, 1961). This animal study confirms lidocaine's reputation as a 'good drug' among physicians, though medical literature contains ample evidence for adverse effects produced by lidocaine. In

pigeons, XC appeared to be more effective in suppressing the cardiac acceleration in response to odour stimuli than Gingicaine (Wallraff, 1988).

Section of the olfactory nerves reduced the duration of tonic immobility (termed 'animal hypnosis' or otherwise in the early literature) in pigeons (Wenzel and Rausch, 1977), whereas the treatment in this paper significantly prolonged it by a factor of 4.7. Thus, the XC effect, unlike the former effect, may not be linked to the suppression of olfaction. At present, tonic immobility constitutes one of the best behavioural indices of fear in animals because procedures believed to elicit fear prolong the reaction, whereas fear reducers attenuate it (see Gallup, 1977; Jones, 1986, for reviews). In this context the drug's effect seems to be associated with fear. This, in turn, may result from the indisposition owing to dysfunctions of the vestibular–ocular system reported here.

Unlike other employers of this technique, Benvenuti and Wallraff (1985) and Kiepenheuer (1985) attempted to control potential influences on flying (homing) motivation, affective behaviour or arousal in their experimental design by treating both control and experimental pigeons with XC and Gingicaine to prevent odour perception at the release site. The results obtained were interpreted, with different degrees of certainty, as evidence for site simulation by olfactory information. If, however, the birds' compass or course control was impaired, even these results do not exclude other explanations.

Since the vestibular apparatus is capable of measuring rotational and linear accelerations, its involvement in course control is possible. Bisecting the semicircular canals or removing the cochlea, lagena or sacculus does not impair pigeon homing; yet, in all these experiments some loophole remained for alternative explanations (see Schmidt-Koenig, 1979, for a review). In homing pigeons and migratory pigeon species, unlike domestic pigeons and a nonmigratory pigeon species, prolonged discharges were recorded from the cerebellum after rotation on a turntable. Hence, rotational information could play a role in orientation (Gualtierotti *et al.* 1959; Schreiber *et al.* 1962).

In conclusion, these results of intranasal application of local anaesthetics imply that the manifold, puzzling effects on homing behaviour reported in the literature are not necessarily explicable in terms of a blockage of the olfactory pathways alone, but alternatively or additionally they may be the result of non-olfactory effects based on disruptive course control, altered detour dependency and/or changes in the general well-being or motivation to fly directly home.

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References

ADRIANI, J., BEATLER, W. A., BRIHMADESAM, L. AND NARAGHI, M. (1985). Topical anesthetics: use and misuse. *Southern Med. J.* **78**, 1224–1229.

ÅSTRÖM, A. AND PERSSON, N. H. (1961). The toxicity of some local anesthetics after application

on different mucous membranes and its relation to anesthetic action on the nasal mucosa of the rabbit. *J. Pharmac. exp. Ther.* **132**, 87–90.

BEASON, R. C. AND SEMM, P. (1987). Magnetic responses of the trigeminal nerve system of the bobolink *(Dolichonyx oryzivorus)*. *Neurosci. Lett.* **80**, 229–234.

BENVENUTI, S. AND WALLRAFF, H. G. (1985). Pigeon navigation: site simulation by means of atmospheric odours. *J. comp. Physiol.* **156**, 737–746.

BILO, D. (1977). Eingangs-Ausgangsbeziehungen und Modell eines Drehreflexes der Haustaube *Columba livia*. *Biol. Cybernetics* **26**, 109–124.

BILO, D., BEST, G., SCHÖNENBERGER, I. AND NACHTIGALL, W. (1972). Methode der Halothan-Inhalationsnarkose bei Vögeln (Taube und Wellensittich). *J. comp. Physiol.* **79**, 137–152.

BUBIEN-WALUSZEWSKA, A. (1981). The cranial nerves. In *Form and Function in Birds*, vol. 2 (ed. A. S. King and J. McLelland), pp. 385–438. New York: Academic Press.

CHERNICK, W. S. (1976). Local anesthetics. In *Drill's Pharmacology in Medicine* (ed. J. R. Dipalma), pp. 190–210. New York: McGraw-Hill.

COVINO, B. G. (1972). Local anesthesia. I. *New Engl. J. Med.* **286**, 975–1035.

DORNFELDT, K. (1979). Beeinträchtigung der Navigation der Brieftaube (*Columba livia*) durch Xylocain-Behandlung der Nasenhöhle. *Verh. dt zool. Ges.* **72**, 218.

GALLUP, G. G. (1977). Tonic immobility: the role of fear and predation. *Psychol. Rec.* **27**, 41–61.

GUALTIEROTTI, T., SCHREIBER, B., MAINARDI, D. AND PASSERINI, D. (1959). Effect of acceleration on cerebellar potentials in birds and its relation to sense direction. *Am. J. Physiol.* **197**, 469–474.

JONES, R. B. (1986). The tonic immobility reaction of the domestic owl: a review. *Wldl. Poult. Sci.* **42**, 82–96.

KIEPENHEUER, J. (1985). Can pigeons be fooled about the actual release site position by presenting them information from another site? *Behav. Ecol. Sociobiol.* **18**, 75–82.

KILIAN, H. (1974). Komplikationen bei peripheren Nervenblockaden. In *Die peripheren Leitungsanästhesien* (ed. H. Nolte, J. Meyer and J. Wurster), pp. 97–121. Stuttgart: Georg-Thieme Verlag.

LÖFGREN, N. (1948). *Studies on Local Anesthetics. Xylocaine: A New Synthetic Drug.* Stockholm: Ivar Haeggstroms.

MERCHANT, S. N. AND KIRTANE, M. V. (1986). Vestibular effects of intraveneous lidocaine used in the treatment of tinnitus. *J. Lar. Otol.* **100**, 1249–1253.

PAPI, F. (1986). Pigeon navigation: solved problems and open questions. *Monitore zool. ital.* **20**, 471–517.

PAPI, F., FIORE, L., FIASCHI, V. AND BENVENUTI, S. (1971). The influence of olfactory nerve section on the homing capacity of carrier pigeons. *Monitore zool. ital.* **5**, 265–267.

PRECHT, W. (1978). *Neuronal Operation in the Vestibular System.* Berlin, Heidelberg, New York: Springer-Verlag.

SCHMIDT-KOENIG, K. (1979). *Avian Orientation and Navigation.* London: Academic Press.

SCHMIDT-KOENIG, K. AND PHILLIPS, J. B. (1978). Local anesthesia of the olfactory membrane and homing in pigeons. In *Animal Migration, Navigation, and Homing* (ed. K. Schmidt-Koenig and W. T. Keeton), pp. 119–124. Berlin, Heidelberg, New York: Springer-Verlag.

SCHREIBER, B., GUALTIEROTTI, T. AND MAINARDI, D. (1962). Some problems of cerebellar physiology in migratory and sedentary birds. *Anim. Behav.* **10**, 42–47.

WALLRAFF, H. G. (1988). Olfactory deprivation in pigeons: Examination of methods applied in homing experiments. *Comp. Biochem. Physiol.* **89**A, 621–629.

WALLRAFF, H. G. AND FOÀ, A. (1981). Pigeon navigation: Charcoal filter removes relevant information from environmental air. *Behav. Ecol. Sociobiol.* **9**, 67–77.

WALLRAFF, H. G., PAPI, F., IOALE, P. AND BENVENUTI, S. (1986). Magnetic fields affect pigeon navigation only while birds can smell atmospheric odors. *Naturwissenschaften* **73**, 215–217.

WENZEL, B. M. (1974). The olfactory system and behavior. In *Limbic and Autonomic Nervous Systems Research* (ed. L. V. DiCara), pp. 1–40. New York: Plenum Press.

WENZEL, B. M. AND RAUSCH, L. J. (1977). Does the olfactory system modulate affective behavior in the pigeon? In *Tonic Functions of Sensory Systems* (ed. B. M. Wenzel and H. P. Zeigler). *Ann. New York Acad. Sci.* **290**, 315–340.

WILTSCHKO, R. AND WILTSCHKO, W. (1989). Pigeon homing: olfactory orientation – a paradox. *Behav. Ecol. Sociobiol.* **24**, 163–173.

WILTSCHKO, W., WILTSCHKO, R., FOÀ, A. AND BENVENUTI, S. (1986). Orientation behavior of pigeons deprived of olfactory information during the outward journey and at the release site. *Monitore zool. ital.* **20**, 183–193.

WILTSCHKO, W., WILTSCHKO, R. AND JAHNEL, M. (1987*a*). The orientation behaviour of anosmic pigeons in Frankfurt a.M., Germany. *Anim. Behav.* **35**, 1324–1333.

WILTSCHKO, W., WILTSCHKO, R. AND WALCOTT, C. (1987*b*). Pigeon homing: Different effects of olfactory deprivation in different countries. *Behav. Ecol. Sociobiol.* **21**, 333–342.