

“Velocity leakage” in the pigeon vestibulo-ocular reflex

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Abstract. The transfer characteristics of the vestibulo-ocular reflex (VOR), and of the semicircular canal primary afferents (SCPAs) that drive it, have been studied in several species. In monkeys and cats, the dominant time constant describing horizontal VOR dynamics (τ_{hv}) is longer than that (τ_c) of horizontal SCPAs. This lengthening of the time constant has been attributed to a “velocity storage” mechanism that has been modeled as a positive feedback loop in the VOR pathways. We have studied the transfer characteristics of horizontal and vertical VOR and SCPAs in unanesthetized pigeons. In this species the dominant time constants of both the horizontal and vertical VOR (τ_{hv} and τ_{vv}) are shorter than τ_c . This finding indicates that time constants characterizing the lower frequency response of the VOR can be lengthened or shortened depending on the species. We propose that in the pigeon the “velocity leakage” mechanism can be modeled by substituting negative feedback for positive feedback in the model of the VOR pathways. Negative feedback can also account for the further shortening of τ_{hv} and τ_{vv} as VOR gain increases with arousal. Additionally, making the negative feedback loop nonlinear can model the dependency of lower frequency VOR phase on amplitude, and skew in VOR waveforms. Pigeon VOR and SCPA dynamics also differ in their adaptive properties and higher frequency behavior. A predominance of input from highly adaptive SCPAs is proposed to account for the increased adaptation of the vertical VOR as compared with SCPAs overall. A pure time-delay associated with VOR operation can explain the phase lag of the VOR relative to SCPAs at higher frequencies.

1 Introduction

The function of the vestibulo-ocular reflex (VOR) is to stabilize the retinal image during head rotations by producing eye rotational velocities that are equal and opposite to head velocities over the frequency range of

naturally occurring head rotations (Mayne 1950). This function is faithfully served by the horizontal and vertical VOR (HVOR and VVOR, respectively) of alert rhesus monkeys, for example, since at a frequency of 1.0 Hz, the gain of eye velocity relative to head velocity is approximately 1, and the phase is near zero (Furman et al. 1982; Correia et al. 1985). The VOR is driven by head rotational velocity signals which originate in horizontally and vertically oriented semicircular canal (SC) vestibular receptors and are transmitted into the brainstem by SC primary afferents (SCPAs). For certain mammalian species, the frequency range of the HVOR is actually broader, by being extended to lower frequencies, than that of the SCPA signal that drives it.

This broadening is illustrated by the finding that the dominant time constant, which describes most of the dynamics, is longer for the HVOR (τ_{hv}) than for the SCs (τ_c), the latter being estimated from the responses of SCPAs. In rhesus monkeys, for example, τ_{hv} and τ_c were determined to be 23 and 6 s, respectively, using simultaneously recorded HVOR eye rotation and horizontal SCPA unit responses to step stimuli (Büttner and Waespe 1981). Using frequency responses, τ_{hv} was found to be 12 s (Furman et al. 1982). In the squirrel monkey, τ_{hv} and τ_c were determined to be 19 s (Paige 1983) and 6 s (Fernandez and Goldberg 1971), respectively. In the cat, τ_{hv} and τ_c were determined to be 15 s (Robinson 1976) and 4 s (Melvill Jones and Milsum 1970), respectively. Lengthening of the value of the dominant time constant for the HVOR in monkeys and cats has been attributed to a theoretical construct called “velocity storage” (Raphan et al. 1979). Velocity storage has been modeled (Robinson 1981) as a positive feedback pathway that prolongs the neural activity that is initiated in the brainstem by the SCPA response.

Lengthening of the dominant time constant of the VVOR (τ_{vv}) relative to τ_c is less clear. Fernandez and Goldberg (1971) showed that τ_c was nearly the same for horizontal and vertical SCPAs. Assuming this holds true for the rhesus monkey, τ_c for vertical SCPAs should also be 6 s, as it is for horizontal SCPAs in this species (Büttner and Waespe 1981). Matsuo and Cohen (1984), using nystagmic responses to steps, determined that

$\tau_{vv} = 17$ s for upward slow-phase velocity responses and $\tau_{vv} = 9$ s for downward slow-phase velocity responses. Correia and associates (1985), using sinusoidal oscillations (frequencies = 0.01, 0.1, and 1.0 Hz), found no up-down asymmetry in the nystagmic responses and no lengthening of the value of the average time constant ($\tau_{vv} = 6$ s).

In a series of experiments using the unanesthetized pigeon (Anastasio et al. 1985; Anastasio and Correia 1988), we were able to compare the transfer characteristics of SCPAs and the VOR in the same preparation. We found that τ_{hv} did not differ from τ_{vv} in the pigeon. However, both of these VOR time constants were shorter than τ_c . We found that $\tau_{hv} = \tau_{vv} = 4$ s and $\tau_c = 10$ s, a reduction of 2.5 times. We have evaluated several models to explain the "velocity leakage" observed in the pigeon and will present and discuss the most parsimonious model.

Other differences between the response properties of SCPAs and VOR in the pigeon will also be accounted for. Response adaptation in the VVOR is greater than that of the HVOR or of the SCPAs as a population. We propose that a predominance of input from more highly adaptive SCPAs can account for the increased adaptation of the VVOR as compared with the HVOR and SCPAs overall. At higher frequencies, the SCPA frequency response exhibits a phase lead while the VOR exhibits a lag. We suggest that a pure time-delay, associated with synaptic, conduction, and neuromuscular mechanisms along the VOR pathway, is responsible for the observed differences in SCPA and VOR higher frequency responses.

2 Transfer characteristics of the SCPAs and the VOR in pigeons

2.1 Experimental preparation

The data to be modeled are the frequency responses of SCPAs obtained using extracellular unit recordings (Anastasio et al. 1985) and VOR eye movements measured using the magnetic search coil technique (Anastasio and Correia 1988), taken in essentially the same chronic, unanesthetized pigeon preparation. The frequency response data were obtained using sinusoidal rotational stimuli over the frequency range extending from 0.03 to 6.0 Hz at a peak rotational (angular) velocity of 12 deg/s. The VOR was tested in pigeons that were either pharmacologically aroused (using amphetamine) or drug free (i.e., normal). The frequency responses of SCPAs were tested in normal pigeons only.

Rotations were delivered in the horizontal plane for horizontal SCPAs and for the HVOR, in the sagittal plane for vertical SCPAs, and in the coronal plane for the VVOR. The planes chosen for testing VOR have behavioral significance since, in the lateral-eye pigeon, horizontal and vertical eye rotations are made in the horizontal and coronal planes, respectively. Vertical SCPAs were tested in the sagittal rather than the coronal plane for purely technical reasons; unit recordings could be more

reliably maintained during rotation in the sagittal rather than the coronal plane. Due to canal geometry in vertebrates (Ezure and Graf 1984), the vertical canals (anterior and posterior) are stimulated approximately equally with rotations in either the sagittal or coronal planes. The axes of rotation were strictly vertical and head-centered in all cases. This prevented coupling with the otolith system, which is known to modify VOR time constants in monkeys (Raphan and Cohen 1981). The responses were tested with the pigeons either blind-folded (SCPAs) or in total darkness (VOR). This prevented coupling with the optokinetic system (Raphan et al. 1979).

2.2 Best-fit transfer functions

Transfer functions fit to both the SCPA and VOR frequency responses were based on the second-order, torsion-pendulum model of the SC (van Egmond et al. 1949; Wilson and Melvill Jones 1979):

$$\frac{D}{\ddot{H}} = \frac{\tau_l \tau_s}{(s\tau_l + 1)(s\tau_s + 1)} \quad (1)$$

which relates cupular deflection (D) to head rotational acceleration (\ddot{H}), and where τ_l and τ_s are the long and short time constants of the SC, respectively, and s is the Laplace operator ($s = j\omega$, where $j = \sqrt{-1}$, $\omega = 2\pi f$, and f is the head rotational frequency in Hz). The value of the short time constant in the torsion pendulum equation of the SC in the pigeon (τ_s) has been estimated to be 0.003 s (Money et al. 1966; Dickman and Correia 1989b). Thus, the higher frequency pole [$\tau_s/(s\tau_s + 1)$] of the torsion-pendulum equation will describe the response of the SCs only at frequencies above about 50 Hz ($1/2\pi\tau_s$), which is outside the range of our data (up to 6.0 Hz maximum frequency). The value of the long time constant (τ_l) has been estimated from the responses of SCPAs in pigeons to be about 10 s (Landolt and Correia 1980; Anastasio et al. 1985; Dickman and Correia 1989a). Evaluating equation (1) with this value of τ_l indicates that over most of the frequency range studied (frequencies greater than about 0.02 Hz or $1/2\pi\tau_l$), the SCs will respond to head rotational acceleration (\ddot{H}) by producing a cupular deflection (D) that is proportional to head rotational velocity (\dot{H}). Within the range of head rotational frequencies employed in our experiments on unanesthetized pigeons, the hydromechanical response of the SCs can be described approximately by the following first-order transfer function:

$$\frac{D}{\dot{H}} = \frac{s\tau_c}{(s\tau_c + 1)} \quad (2)$$

where the canal (SC) time constant (τ_c) is equal in value to the long time constant (τ_l), and both sides have been multiplied by s so that cupular deflection (D) is related to head rotational velocity (\dot{H}).

Equation (2) forms the basis for the transfer functions describing the frequency responses of the SCPAs and the HVOR and VVOR in unanesthetized pigeons. These transfer functions, which also include terms describing other neurobiological dynamic characteristics, are given

below for the SCPAs (Anastasio et al. 1985):

$$\frac{A}{\dot{H}} = \frac{s\tau_c}{(s\tau_c + 1)} s^k (s\tau_z + 1) \quad (3)$$

and for the HVOR and VVOR (Anastasio and Correia 1988):

$$-\frac{\dot{E}}{\dot{H}} = G_v \frac{s\tau_v}{(s\tau_v + 1)} s^k e^{-s\tau_d} \quad (4)$$

where A is the instantaneous firing rate of SCPAs which is related to cupular deflection (D), \dot{E} is eye rotational velocity, \dot{H} is head rotational velocity, τ_c and τ_v are the dominant time constants of the SCPAs and VOR, respectively, τ_z is the time constant of the cupular velocity operator, τ_d is the time constant of VOR delay, k is the exponent of the fractional order adaptation operator, and s is the Laplace operator. The term G_v represents the frequency-independent (or forward-loop) gain of the VOR. The frequency-independent gain of the SCPAs was normalized to 1.

In Equation (4), VOR is defined as $-\dot{E}/\dot{H}$. Since \dot{E} is opposite to \dot{H} for the VOR, gains computed in this way are positive. Also, VOR phase is defined as zero when \dot{E} and \dot{H} are separated by 180° . Thus, the VOR response is essentially inverted before the gain and phase relative to \dot{H} are computed, and this facilitates comparisons with the SCPA frequency response. The inverting nature of VOR is dealt with explicitly in the models presented below. The gain of the SCPAs has dimensions of impulses/s per deg/s of \dot{H} but is normalized to 1 to facilitate comparisons with the VOR frequency response.

Both the SCPAs (Anastasio et al. 1985) and the VOR (Anastasio and Correia 1988) in pigeons predominantly reflect the first-order dynamics of the SCs. They have gains that are approximately constant and phases close

to zero relative to head rotational velocity over most of the frequency range studied, with gain decreasing and phase lead increasing as frequency decreases at the lower end of the range. However, the values of their dominant time constants (τ_c and τ_v , respectively) are quite different, as considered below. Another transfer characteristic common to both the SCPAs and the VOR is across-frequencies or fractional order adaptation. In the frequency domain, this term takes the form of a fractional order differentiator (s^k ; $0 < k < 1$) and describes a gain that gradually increases with frequency, and a constant phase lead between zero and 90° .

An additional operator was necessary to fit adequately each of the SCPA and VOR higher frequency responses. For the SCPAs, a zero term representing cupular velocity sensitivity ($s\tau_z + 1$) was included (Fernandez and Goldberg 1971). This term describes a higher frequency (above 2.0 Hz) gain enhancement and phase advance noted in the frequency responses of most SCPAs. For the VOR, a pure time-delay term ($e^{-s\tau_d}$) was included (Milsum 1966). The VOR eye movement response to head movement is not instantaneous, due to synaptic, conduction, and neuromuscular delays along the VOR pathway. In the frequency domain, the dynamic consequences of this time delay consist of a phase lag at higher frequencies unaccompanied by a gain change (Milsum 1966). This dynamic characteristic has been observed in the frequency responses of the HVOR and VVOR in both normal and aroused pigeons (Anastasio and Correia 1988; Fig. 1).

Equations (3) and (4) were fit to the frequency response data of SCPAs (Anastasio et al. 1985) and the VOR (Anastasio and Correia 1988), respectively. Values for the parameters are given in Table 1. The transfer functions were evaluated, incorporating the values from Table 1, and are plotted in Fig. 1, where the dynamics of

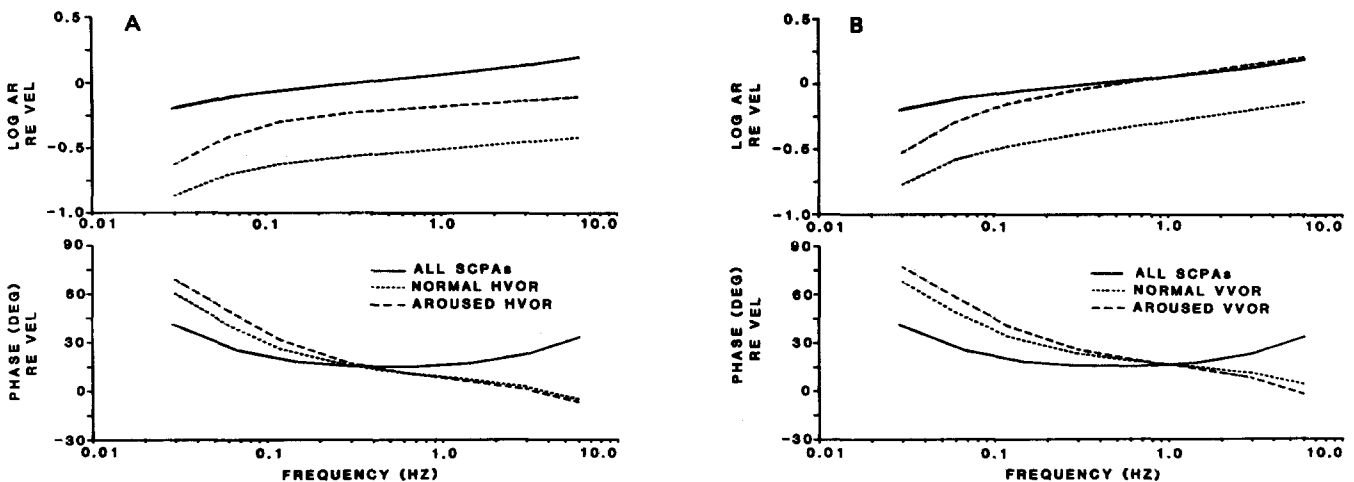


Fig. 1A, B. A comparison of the dynamics of semicircular canal primary afferents (SCPAs) and the horizontal vestibulo-ocular reflex (HVOR, A) and vertical vestibulo-ocular reflex (VVOR, B) of normal and aroused pigeons. Log amplitude ratio (AR) and phase curves, plotted relative to velocity (RE VEL), represent transfer functions fit to the respective frequency response results. Transfer functions had the general forms given in (3) and (4). The curves shown incorporate the parameter values given in Table 1. Actual VOR log(AR) values are plotted; the SCPA log(AR) curve was normalized. Phase leads are given as positive values, phase lags as negative

Table 1. Parameter values for transfer functions describing the frequency response data of semicircular canal primary afferents (SCPAs) and the horizontal and vertical vestibulo-ocular reflex (HVOR and VVOR, respectively) in pigeons

	τ_c	k	τ_z	
SCPAs	9.7	0.13	0.01	
	τ_v	k	τ_d	G_v
Normal HVOR	4.4	0.11	0.007	0.26
Normal VVOR	4.3	0.19	0.006	0.37
Aroused HVOR	3.0	0.09	0.007	0.56
Aroused VVOR	3.0	0.18	0.008	0.85

Transfer functions describing the frequency responses of the SCPAs and HVOR and VVOR are shown in (3) and (4), respectively. Parameter values from this table were used to evaluate those transfer functions, and the resulting curves are plotted in Fig. 1. τ_c , τ_v are dominant time constants for the SCPAs and VOR, respectively; k is the exponent of the fractional order adaptation term; τ_z is the time constant of the cupular velocity-sensitive zero term; τ_d is the time constant of the pure time-delay term; G_v is the frequency-independent gain of the VOR

the SCPAs in normal pigeons can be compared to those of the HVOR (Fig. 1A) and VVOR (Fig. 1B) in normal and aroused pigeons. Note that the gain (AR) of the SCPAs has been normalized to 1 [$\log(\text{AR}) = 0$], but actual VOR gains are presented as defined above.

2.3 Frequency response comparisons

The most important difference between the transfer characteristics of the SCPAs and the VOR (Table 1) is that the dominant time constant of the VOR response (τ_v) is shorter than that of the SCPAs ($\tau_c = 10$ s). This is even more true for aroused pigeons ($\tau_v = 3$ s) than for normal pigeons ($\tau_v = 4$ s). This difference in dominant time constants can be appreciated by observing that the gain “rolls-off” at a higher frequency, and the phase lead is greater in the low to middle frequency range, for the VOR response as compared with the SCPA response (Fig. 1).

Another striking difference in SCPA and VOR dynamics occurs in the higher frequency region (above 2.0 Hz), where the SCPA response exhibits a phase advance (and a slight gain enhancement), while the VOR response exhibits a phase delay without a gain decrement. This phase delay does not differ much for the HVOR and VVOR in both normal and aroused pigeons.

A third notable difference in SCPA and VOR behavior involves across-frequencies adaptation. The values of k are similar for the SCPAs and the HVOR ($k = 0.1$) but are higher for the VVOR ($k = 0.2$). The k values do not differ much between normal and aroused birds. This difference in fractional order adaptation can be appreciated by observing that the slopes of the gain curves are slightly greater for the VVOR than for the HVOR or SCPAs. Also, the VVOR phase curves (Fig. 1B) are offset more in lead and cross the SCPA phase curve at a higher frequency than the HVOR curves (Fig. 1A), even though both the HVOR and VVOR have similar τ_v and τ_d values (Table 1).

3 A control systems model of the pigeon VOR

The model schematized in Fig. 2 is adapted from the one proposed by Robinson (1981). That model accounted for signal processing along the HVOR pathway in monkeys, whereas the model in Fig. 2 concerns both the HVOR and VVOR pathways in pigeons. Other differences between the monkey and pigeon models will be noted below. The elements that compose the basic VOR system and are depicted in the model include the semicircular canals (SCs), SCPAs, vestibular nuclei (VN) neurons, extra-ocular muscle motoneurons, extra-ocular muscles, and the eyeball itself (Lorente de N6 1933; Wilson and Melvill Jones 1979). The model indicates how head velocity (\dot{H}) could be transformed into VOR eye velocity (E) through a series of transformations involving neural signals carried by canal afferents (A), VN neurons (N), the optokinetic system (O), and eye muscle motoneurons (M).

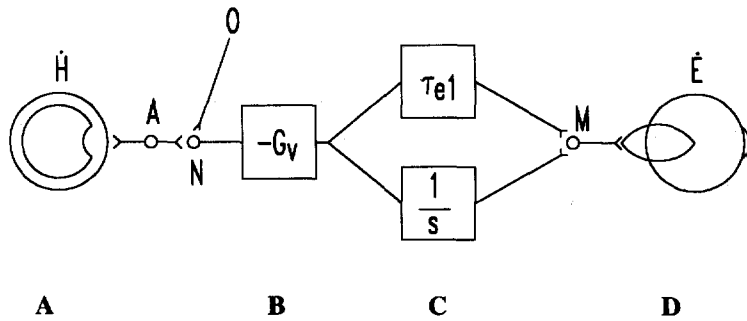
3.1 Level of the semicircular canal primary afferents

The transfer function describing the relationship between SCPA instantaneous firing rate and head velocity is shown in Fig. 2A. This is (3) described above, with the exception that the exponent of the fractional order adaptation term (k) has been replaced by k_c for the canals, to distinguish it from a similar exponent used to modify adaptation in the VOR (k_v). The s^k terms are used in the pigeon VOR model to describe across-frequencies adaptation, whereas the adaptation operator [$s\tau_a/(s\tau_a + 1)$] (Young and Oman 1969) was used in the monkey VOR model (Robinson 1981) to describe lower frequency adaptation.

3.2 Level of the vestibular nuclei

The transfer function in Fig. 2B describes the response of a presumed set of VN neurons to SCPA input in the pigeon. This transfer function is that of a *lead* compensator (Milsum 1966), which essentially exchanges the dominant time constant of the canal response (τ_c) for the *shorter* time constant (τ_v) of the VOR response in the pigeon. The analogous transfer function in the monkey VOR model (Robinson 1981) is that of a *lag* compensator, which exchanges τ_c for the *longer* τ_v . This transformation is thought to occur at the level of the VN, since horizontal canal-driven VN neurons in monkeys have average time constants closer in value to τ_{hv} than to τ_c (Buettner et al. 1978; Waespe and Henn 1979). In pigeons, however, no direct measurements of the frequency response of VN neurons have been made.

Lag compensation in the monkey VOR is due to the action of the velocity storage mechanism (Raphan et al. 1979), which has been modeled as *positive* feedback involving VN neurons (Buettner et al. 1978) and the optokinetic system (Robinson 1981). We propose that lead compensation in the pigeon VOR is due to the action of the velocity leakage mechanism which we model as an analogous *negative* feedback loop. The



$$\frac{A}{\dot{H}} = \frac{s\tau_c}{(s\tau_c + 1)} s^{k_c} (s\tau_z + 1) \quad \left| \quad \frac{N}{A} = -G_v \frac{\tau_v (s\tau_c + 1)}{\tau_c (s\tau_v + 1)} \quad \right| \quad \frac{M}{N} = \frac{(s\tau_{e1} + 1)}{s} \quad \left| \quad \frac{\dot{E}}{M} = \frac{s}{(s\tau_{e1} + 1)(s\tau_{e2} + 1)} s^{k_v} e^{-s\tau_d}$$

where:

$$\tau_z = \tau_{e2}$$

$$G_v = G_{hv} \text{ or } G_{vv}, G_{hv} \neq G_{vv}$$

$$k_v = k_{hv} \text{ or } k_{vv}, k_{hv} \neq k_{vv}$$

E

F

$$\frac{\dot{E}_h}{\dot{H}} = -G_{hv} \frac{s\tau_v}{(s\tau_v + 1)} s^{(k_c + k_{hv})} e^{-s\tau_d} \quad \left| \quad \frac{\dot{E}_v}{\dot{H}} = -G_{vv} \frac{s\tau_v}{(s\tau_v + 1)} s^{(k_c + k_{vv})} e^{-s\tau_d}$$

Fig. 2. A schematic diagram of the pigeon HVOR and VVOR. Also shown are generalized transfer functions describing the SCPA frequency response (A), the velocity leakage mechanism (B), the oculomotor neural integrator and direct pathway in parallel (C), and eyeball and associated dynamics (D). Transfer functions describing HVOR and VVOR frequency responses are shown in E and F, respectively. Multiplication of the transfer functions in A–D will result in the transfer functions of the HVOR and VVOR (E and F). \dot{H} , head rotational velocity; A, SCPA head rotational velocity signal; N, vestibular nucleus (VN) neuron head rotational velocity signal; O, possible input to VN from the optokinetic system; M, extra-ocular motoneuron eye rotation command signal; \dot{E} , eye rotational velocity; \dot{E}_h , horizontal eye rotational velocity; \dot{E}_v , vertical eye rotational velocity; s, Laplace variable; τ_c , canal time constant; τ_z , cupular velocity zero time constant; τ_v , VOR time constant; τ_{e1} , middle frequency eyeball lag time constant; τ_{e2} , higher frequency eyeball lag time constant; τ_d , pure VOR time-delay time constant; G_v , forward-loop VOR gain; G_{hv} , forward-loop HVOR gain; G_{vv} , forward-loop VVOR gain; k_c , fractional exponent for canal adaptation term; k_v , fractional exponent for VOR adaptation modifier; k_{hv} , fractional exponent for HVOR adaptation modifier; k_{vv} , fractional exponent for VVOR adaptation modifier

negative feedback loop is shown in Fig. 3 and considered further below. The gain of the VOR (G_v) is arbitrarily assigned to the VN level. It is negative in the model to indicate that the VOR commands eye velocity to be opposite to head velocity.

3.3 Level of the eye muscle motoneurons

The transfer function in Fig. 2C describes the response of eye muscle motoneurons to VN neuron input in the pigeon. In monkeys, eye muscle motoneurons carry signals proportional to both eye velocity and position during VOR and appear to receive both a direct and a mathematically integrated vestibular signal (Skavenski and Robinson 1973). The neural integrator of the oculomotor system, in parallel with the direct pathway, produces a lead element in addition to integration (Robinson 1981), which is shown in the transfer function of the motoneurons. The direct pathway is assumed to have a gain of τ_{e1} , equal in value to the time constant describing middle frequency eyeball plant dynamics (see below). Recordings from eye motor nuclei in birds indicate that both the eye velocity and position components are also

present but may be carried by separate motoneurons (Letelier et al. 1987).

3.4 Level of the eyeball plant

The relationship between motoneuron discharge rate and the eye position that results from this neural command in monkeys can be described by a second-order, double-lag transfer function (Keller 1973):

$$\frac{E}{M} = \frac{1}{(s\tau_{e1} + 1)(s\tau_{e2} + 1)} \quad (5)$$

where the values of the time constants in seconds have been estimated as 0.179 for τ_{e1} and 0.016 for τ_{e2} . Thus, eyeball plant dynamics contribute a middle $[1/(s\tau_{e1} + 1)]$ and higher $[1/(s\tau_{e2} + 1)]$ frequency lag, due to the elastic and viscous properties of the orbital suspensory tissues and eye muscles. Equation (5) forms the basis for the transfer function shown in Fig. 2D. Since the pigeon results are reported relative to head velocity, both sides of (5) have been multiplied by s in Fig. 2D. Also given as part of the eyeball plant transfer function are the pure time-delay ($e^{-s\tau_d}$) and fractional order adaptation modifier (s^{k_v})

terms. The adaptation term at this final level models any difference in VOR adaptation and average SCPA adaptation.

3.5 Level of the vestibulo-ocular reflex

The transfer functions of the HVOR and VVOR, in Fig. 2E and F, respectively, can be obtained by multiplication of the intermediate transfer functions given in Fig. 2A, B, C, and D. This amounts to a transformation of the SCPA head velocity signal into eye velocity during the VOR, as they have been observed in pigeons. Thus, multiplication of A and B in Fig. 2 will swap the dominant time constant of the SCPA response (τ_c) for that of the VOR (τ_v) and will also produce an inversion of the signal and contribute gain G_v . Gain differs for the HVOR (G_{hv}) and VVOR (G_{vv}), as indicated in Fig. 2. Similarly, multiplication of C and D in Fig. 2 will produce cancellation of the middle frequency lag in the eyeball plant dynamic response (Skavenski and Robinson 1973) and will also cancel the free s terms.

The pure time-delay operator ($e^{-s\tau_d}$) remains in the transfer functions of the HVOR and VVOR. In the frequency domain, the consequences of this time delay consist of a phase lag at higher frequencies unaccompanied by a gain change (Milsum 1966). This characteristic is apparent in the frequency responses of the VOR as shown in Fig. 1. At higher frequencies, the VOR should be influenced by the higher frequency lag of the eyeball plant. This lag should contribute a higher frequency phase lag and gain attenuation. These dynamic characteristics are *not* apparent in the frequency response of the pigeon VOR; the higher frequency phase lag *without* a gain attenuation can be accounted for by the pure time delay. The frequency response of the pigeon SCPAs exhibits a phase lead and gain enhancement at higher frequencies (Fig. 1). These characteristics also are *not* apparent in the transfer function of the VOR. It has been suggested that the gain enhancement and phase lead inherent in the SCPA head rotational velocity signal could compensate for the gain attenuation and phase lag expected from the higher frequency response of the eyeball plant (Fernandez and Goldberg 1971). It is possible that the cupular velocity term ($s\tau_z + 1$) compensates the higher frequency lag of the eyeball plant [$1/(s\tau_{e2} + 1)$] in pigeons. If this is true, then τ_z and τ_{e2} should be equal in value, as indicated in Fig. 2. The average value of τ_z for pigeon SCPAs is 0.01 s as given in Table 1.

Across-frequencies adaptation, characterized by a gradually increasing gain and a constant phase lead over the entire bandwidth, is apparent in the SCPA and VOR frequency response curves of Fig. 1. The degree of adaptation is quantified by the value of the exponent (k) of the fractional order adaptation operator (s^k). Response adaptation is about the same for the HVOR as for SCPAs on average ($k = 0.1$) but is about twice as high for the VVOR ($k = 0.2$) (Table 1). To account for these differences, an additional adaptation term has been provided in the model. This term (s^{k_v}) can modify SCPA adaptation (s^{k_c}). Additionally, k_v takes different values for HVOR (k_{hv}) or VVOR (k_{vv}). Multiplication of the

fractional order adaptation operator of the SCPAs by that of either the VVOR or HVOR will simply result in addition of the exponents. In this way, the adaptive properties of the SCPA head rotational velocity signal will be changed from k_c to $(k_c + k_{vv})$ for the VVOR and from k_c to $(k_c + k_{hv})$ for the HVOR. If the k values as determined from frequency response data in the pigeon are taken as 0.2 for the VVOR, 0.1 for the HVOR, and 0.1 for the SCPAs, then the adaptive properties of the SCPA head rotational velocity signal will be appropriately modified if k_{vv} is equal to 0.1 and k_{hv} is equal to zero.

4 The velocity leakage mechanism

A neural, negative feedback loop at the level of the VN is proposed to account for velocity leakage in the pigeon VOR. This loop is illustrated in detail within a model of the pigeon vestibulo-optokinetic system in Fig. 3. The VOR portion (Fig. 2) of the vestibulo-optokinetic model (Fig. 3) has been simplified. Dynamic terms related to cupular velocity sensitivity, adaptation, neural integration, eyeball mechanics, and delay have been omitted to focus attention on the processing of vestibular and optokinetic signals at the VN level by the velocity leakage mechanism. This model is analogous to the model of the monkey vestibulo-optokinetic system proposed by Robinson (1981), in which the velocity storage mechanism is thought to be shared between the vestibular and optokinetic systems. The pigeon and monkey vestibulo-optokinetic models differ in several important respects, as noted below.

The goal of the vestibulo-optokinetic system is to match the velocity of gaze ($\dot{G} = \dot{E} + \dot{H}$) with that of the visual scene or world (\dot{W}), so as to maintain retinal image stability during head rotation. The \dot{H} signal enters the system after being transduced by the canals, which are represented by the simplified canal (SC) transfer function [Eq. (2)] at the bottom of Fig. 3. The function of the optokinetic system is to reduce retinal slip ($\dot{e} = \dot{W} - \dot{G}$) by using it as an error signal in a feedback servomechanism which is illustrated by the outer negative feedback loop in Fig. 3. The optokinetic loop is thought to include the VN in monkeys (Robinson 1981) and is arranged this way in the pigeon model (Fig. 3) as well.

4.1 Time Constant shortening by negative feedback

The negative feedback loop proposed to underlie velocity leakage in the pigeon VOR is illustrated by the inner loop in Fig. 3. The function of this loop is to *shorten* the time constant of the SCPA signal to that of the VOR. Feedback is thought to occur at the level of the VN as in the monkey (Buettner et al. 1978). The negative feedback pathway includes the forward-loop VOR gain (G_v), two inverters (-1), a saturation element (SAT), and a first-order lag element, with feedback gain G_f , that is assumed to have the same time constant as the canals [$G_f/(\sigma_c + 1)$]. This negative feedback loop is analogous to

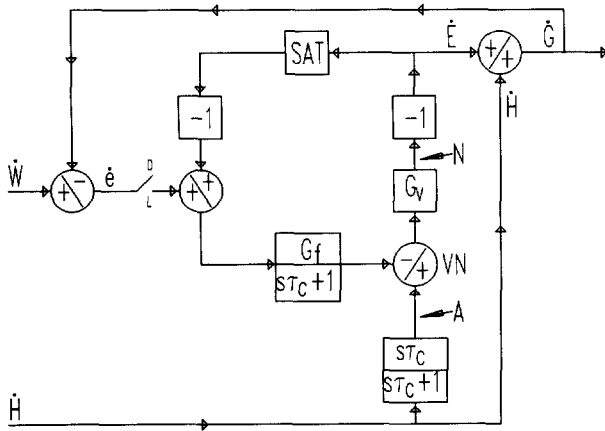


Fig. 3. A schematic diagram of the vestibulo-optokinetic system in the pigeon, detailing the velocity leakage mechanism. Head rotational velocity input (\dot{H}) arrives at the VN after passing through a simplified SCPA transfer function. The VN eye velocity command signal (\dot{E}) is negatively fed back through a low-pass (lag) element via the inner loop. The low-pass element is assumed to have the same time constant as the SCPA transfer function (τ_c). Other inner loop operators include forward loop gain (G_v), feedback gain (G_f), two inverters (-1), and a saturation element (SAT). A retinal slip signal (\dot{e}), which is the difference between gaze ($\dot{G} = \dot{H} + \dot{E}$) and world (\dot{W}) rotational velocity, may sum with the inner loop feedback signal after its second inversion. The dark/light (D/L) switch opens and closes the optokinetic pathway, which forms the outer negative feedback loop. A and N are SCPA and VN neuron head rotational velocity signals, respectively

a positive feedback loop that was proposed (Buettner et al. 1978; Robinson 1981) to *lengthen* the SCPA time constant to that of the VOR in primates. The transfer function of the VN is shown as a lead compensator in Fig. 2B. The lead compensator transfer function can be equated with the transfer function obtained by evaluating the feedback loop:

$$G_v \frac{\tau_v (s\tau_c + 1)}{\tau_c (s\tau_v + 1)} = G_v \frac{1}{1 + G_v G_f} \frac{(s\tau_c + 1)}{\left(s \left(\frac{\tau_c}{1 + G_v G_f} \right) + 1 \right)} \quad (6)$$

The above expression indicates that the ratio of τ_v to τ_c should be equal to the reciprocal of the quantity $(1 + G_v G_f)$ or, equivalently, that τ_v should be equal to τ_c divided by the quantity $(1 + G_v G_f)$ for the pigeon VOR. If G_v is set to 1, and if τ_c for the pigeon SCPA frequency response is taken as 10 s, and τ_v is taken as 4 s for the HVOR and VVOR in normal pigeons, the analysis indicates that G_f should be 1.5. The model also predicts that the gain of the pigeon velocity leakage negative feedback loop ($G_v G_f$) should be approximately 1.5.

4.2 Response of the VOR in darkness and light

Simulation results from the pigeon vestibulo-optokinetic model (Fig. 3) are illustrated in Fig. 4. Shown in Fig. 4A are the responses to a head rotational velocity step of one unit (upper solid line, signal \dot{H}) in the dark (optokinetic

switch is open in Fig. 3) of the SCPAs (dashed curve in Fig. 4A, signal A) and VN neurons (solid curve in Fig. 4A, signal N). The time constant of the VN response is shorter than that of the SCPAs due to the action of the negative feedback loop. The VN response is not purely exponential because of the saturation nonlinearity, which is considered further below. The VN response to the same stimulus in the light (optokinetic switch is closed in Fig. 3) is shown in Fig. 4B (solid curve, signal N). This response has an optokinetic component (dashed curve, signal O). With the gain values chosen ($G_v = 1$, $G_f = 1.5$), the hypothetical closed-loop gain of the pigeon optokinetic component at the VN level will be 0.375 [closed-loop optokinetic gains: inner loop, $G_f G_v / (1 + G_f G_v) = 0.6$; outer loop, $0.6 / (1 + 0.6) = 0.375$].

This low value for the optokinetic component at the VN level in the pigeon is due to the gain reduction effects of the two negative feedback loops: the inner velocity leakage loop and the outer optokinetic loop. In the monkey vestibulo-optokinetic model (Robinson 1981), the inner velocity storage feedback loop is positive, and the closed-loop optokinetic gain is 0.8 (with an inner, positive loop feedback gain of 0.75). Thus, the proposed negative feedback loop underlying velocity leakage in the pigeon would cause a reduction in optokinetic response gain at the VN level, assuming, as for velocity storage in the monkey, that the velocity leakage mechanism is shared between the vestibular and optokinetic systems. The possibility that the pigeon optokinetic system might partly circumvent the velocity leakage mechanism is considered in the Discussion. Another notable difference between the pigeon and monkey vestibulo-optokinetic models concerns the point at which the retinal slip error signal enters the inner loop. In the monkey model, retinal slip enters the inner feedback loop *before* it is reinverted. In the pigeon model, because inner loop feedback is negative, the retinal slip signal must enter the inner loop *after* it is reinverted.

4.3 Effects of nonlinear feedback

The pigeon VOR is largely linear (Anastasio and Correia 1988). One exception is the dependence of phase lead on stimulus amplitude at lower frequencies. At a frequency of 0.03 Hz, pigeon VOR phase lead decreased from 72° to 53° as the stimulus amplitude increased from 6 to 30 deg/s peak rotational velocity. Over a similar range of stimulus amplitudes, τ_v as determined from the step responses of the HVOR in normal rabbits was shown to increase as the stimulus amplitude increased (Baloh et al. 1983). The effect on VOR phase of an increase in τ_v will be to decrease the amount of phase lead in the low to middle frequency range. It has been suggested (Raphan et al. 1979; Baloh et al. 1983) that the VN feedback pathway saturates at higher levels of stimulus amplitude. Equation (6) specifies that $\tau_v = \tau_c / (1 + G_v G_f)$, where G_f is the gain of the VN negative feedback pathway. If this feedback pathway saturates at higher stimulus amplitudes, then G_f would effectively decrease, τ_v would increase, and the VOR phase lead at low frequencies would decrease, thereby accounting for the observed nonlinear

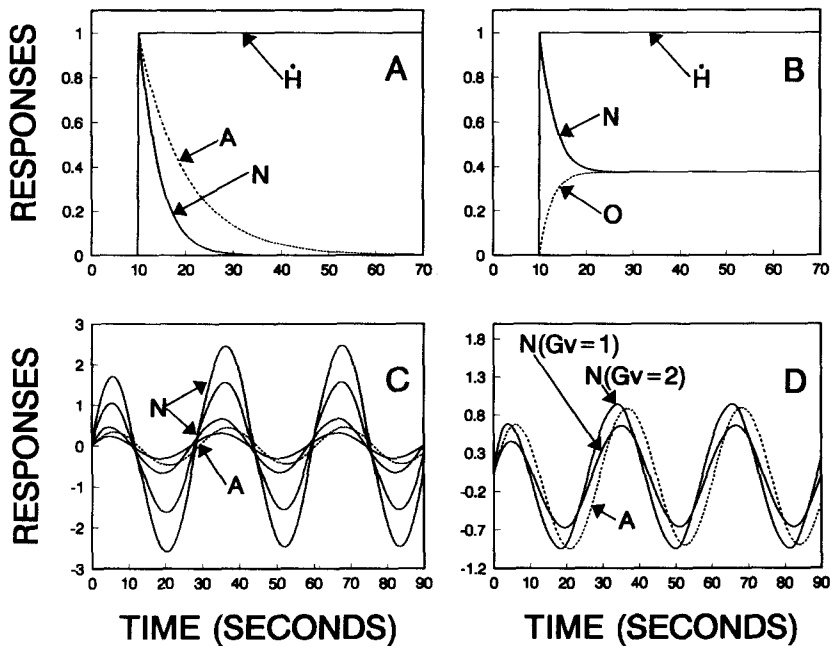


Fig. 4A–D. Simulations of the vestibulo-optokinetic, velocity leakage model shown in Fig. 3. Labels on curves correspond to variables as defined in previous figures and equations. Illustrated are responses of model elements to head rotational velocity step (A and B, upper solid lines, signal \dot{H}) and sinusoidal (C and D, \dot{H} signals not shown) stimuli. A SCPA (dashed curve, signal A) and VN (solid curve, signal N) responses to a step of one unit in the dark (D/L switch open in Fig. 3). The VN response decays faster than the SCPA response due to velocity leakage. B VN response (solid curve, signal N) to a step of one unit in the light (D/L switch closed in Fig. 3) and contribution to this response of the optokinetic system (dashed curve, signal O). The closed-loop optokinetic gain is only 0.375. C VN responses (four solid curves, N signals) to sinusoidal head rotations in the dark at 0.03 Hz and 0.5, 1.0, 2.0, and 3.0 units and SCPA response (dashed curve, signal A) at 0.5 units for comparison. Phase lead of VN relative to SCPA response decreases as head rotation amplitude increases due to saturation in velocity leakage feedback loop. D VN responses to sinusoidal head rotation in the dark at 0.03 Hz and 1.0 unit under normal (smaller amplitude solid curve, signal N with $G_v = 1$) and aroused (larger amplitude solid curve, signal N with $G_v = 2$) conditions. SCPA response (dashed curve, signal A) also shown for comparison. Phase lead of VN relative to SCPA response increases under aroused conditions due to increased efficacy of the velocity leakage feedback loop

dependency of VOR phase lead on stimulus amplitude at lower frequencies in pigeons.

The effects of saturation on VOR responses in the model are illustrated in Fig. 4C. Head rotational velocity is delivered in the dark at a frequency of 0.03 Hz, and peak amplitudes are set at 0.5, 1.0, 2.0, and 3.0 units. The hard saturation element (SAT) in Fig. 3 has a slope of 1 between arbitrarily set limits of ± 0.5 , and a slope of zero outside of these limits. The responses at the VN level (four solid curves in Fig. 4C, N signals) lead the SCPA response (dashed curve, signal A) at the lowest peak head velocity, but this phase lead is reduced as the peak head velocity is increased. The phase lead decrease is accompanied by a slight skewing of the VN response waveform, such that the first quarter cycle of the sinusoidal response in either direction is longer than the second quarter cycle. A similar skewing was also observed in the HVOR and VVOR of normal and aroused pigeons (Anastasio and Correia 1988). The opposite occurs in monkeys for stimuli at 0.02 Hz and amplitudes greater than 120 deg/s. HVOR phase lag decreases as peak sinusoidal head rotational velocity increases, and this phase lag decrease is accompanied by a skewing where the first quarter cycle in either direction is shorter than the second quarter cycle (Paige 1983).

The model thus reflects the property that velocity leakage in pigeons becomes less effective as the amplitude of peak head rotational velocity is increased. Velocity leakage would presumably become ineffective for sufficiently high amplitude head rotational velocities (the VOR lower frequency response would then primarily reflect that of the SCPAs). Considering the velocity leakage mechanism as a low-pass (lag), saturating negative feedback pathway can account for its apparent function, which is to diminish the pigeon VOR response to low frequency, low amplitude head rotations.

4.4 Effects of arousal

The differences between the VOR in normal and aroused pigeons are that gain (G_v) is increased while τ_v is decreased in the latter as compared with the former (Anastasio and Correia 1988; Fig. 1; Table 1). The model can explain this relationship between gain and time constant. Equation (6) specifies that $\tau_v = \tau_c / (1 + G_v G_f)$. An increase in G_v resulting from the effects of increased arousal will produce a further shortening of τ_v in aroused as compared with normal pigeons. Equation (6) was used to calculate the value of G_f for HVOR or VVOR, using values for τ_c , τ_v , and G_v in normal pigeons from Table 1.

These values of G_f were then used to estimate τ_v in aroused pigeons, by substituting the G_v values for aroused birds. The estimated values of τ_v in aroused pigeons are 2.7 and 2.5 s for the HVOR and VVOR, respectively, which are close to the measured values of 3.0 s for both.

The effects of doubling G_v on the sinusoidal responses at the VN level in the model (signal N in Fig. 3) are shown in Fig. 4D. Head rotation in the dark is again 0.03 Hz, and peak amplitude is one unit. With $G_v = 1$, the VN response (smaller amplitude solid curve, signal N with $G_v = 1$) leads the SCPA response (dashed curve, signal A) and is slightly skewed. Increasing G_v to 2 produces a similar response which is larger and exhibits more phase lead (larger amplitude solid curve, signal N with $G_v = 2$). The increase in phase lead indicates that τ_v has been further reduced. For the pigeon VOR, skewness and phase amplitude dependency did not increase under aroused conditions, even though the response amplitude was higher (Anastasio and Correia 1988). This indicates that the degree of saturation in the velocity leakage feedback pathway may be reduced as the VOR gain is increased. In the model, the saturation limits were arbitrarily increased to $+/- 0.8$ for aroused conditions. Other modeling studies suggest that saturation in bilateral VOR feedback pathways may be due to rectification of VN neurons within the feedback loops (Anastasio 1991). In the pigeon, the degree of rectification (saturation) could remain constant after arousal if the increase in gain of VN neurons is accompanied by an increase in their spontaneous rates bilaterally.

5 Discussion

The analysis presented above shows that models of the VOR (Fig. 2) and of the vestibulo-optokinetic system (Fig. 3), developed originally for data from the monkey (Robinson 1981), can also be used with certain parameter adjustments to account for processing along the VOR pathway in the pigeon. For the VOR model (Fig. 2), required adjustments included changes in parameter values and substitution of an across-frequencies for a lower frequency adaptation term. For the vestibulo-optokinetic model (Fig. 3), adjustments included changing the sign of, and adding a saturation element to, the inner loop feedback pathway and moving the summing point for the optokinetic signal. This model was successful in explaining the shortening of the VOR time constant from that of the canal time constant in pigeons, its further shortening with increased gain in aroused as compared with normal birds, the dependency of VOR phase on stimulus amplitude at lower frequencies, and VOR waveform skew. Because the HVOR and VVOR have similar time constants in pigeons, essentially the same model can be used for both. This would not be the case for the primate HVOR and VVOR, which do not have the same time constants, as explained in the Introduction. Although the model can account for VOR processing in pigeons, it is less clear whether it can also do so for the optokinetic response in this species. These issues are considered further below.

5.1 Higher frequency response

The higher frequency response of the pigeon VOR appears to be dominated by a pure time delay of approximately 7 ms. In the monkey, electric stimulation of extraocular motoneurons produced eye movements with a latency of 4–5 ms (Robinson 1968). In the rabbit, electric stimulation of the vestibular nerve produced intracellularly recorded postsynaptic potentials in extraocular motoneurons with a disynaptic latency (Highstein 1973a, b). If each of the two synapses in the VOR pathway have synaptic delays of about 1 ms, then the time delay values determined from pigeon VOR eye movement frequency response data agree well with those measured electrophysiologically in mammals.

It has been proposed (Fernandez and Goldberg 1971; this study) that the cupular velocity-dependent lead characteristics inherent in the responses of SCPAs could cancel the higher frequency lag of the eyeball plant. The time constant (τ_z) of the operator describing SCPA cupular velocity sensitivity is about 0.01 s in pigeons (Table 1), and the modeling results suggest that this could also be the value of the time constant (τ_{e2}) of the higher frequency lag of the eyeball plant in this animal.

5.2 Across-frequencies adaptation

We used a fractional order operator (s^k) to model adaptation which, for the frequency responses of SCPAs and the HVOR and VVOR in pigeons, occurs over a broad frequency range. Such across-frequencies adaptation is characteristic of processes composed of many parallel operators of the form $[s\tau/(s\tau + 1)]$, each having its own distinct time constant and weighted according to the value of its time constant raised to the power $(-1 - k)$ (Thorson and Biederman-Thorson 1974). Concerning SCPA across-frequencies adaptation, it has been suggested that the profuse branching of SCPA terminals within the sensory neuroepithelium may constitute such a process (Correia et al. 1981).

Recent work on the dynamics of pigeon SCPAs over an extended frequency range (0.01 to over 100 Hz) indicates that the value of k describing adaptation in these units ranges from 0.10 to 0.23 (Dickman and Correia 1989b). In normal and aroused pigeons, the values of k describing adaptation in the HVOR and VVOR are about 0.1 and 0.2, respectively (Table 1). These values fall within the range of SCPA k values, and it appears that VOR adaptation could simply reflect that of the SCPAs, with HVOR and VVOR being driven by low and high adaptation SCPAs, respectively. This segregation of SCPA input based on adaptive properties is represented in the model of Fig. 2 by separate adaptation modifiers for HVOR and VVOR (with exponents k_{hv} and k_{vv} , respectively).

5.3 The velocity leakage mechanism

The proposed negative feedback loop responsible for velocity leakage in the pigeon VOR is assumed to reside at the level of the VN. However the actual pattern of neural

connectivity that underlies the velocity leakage feedback loop remains to be elucidated. Feedback loops could be formed within the VN complex itself, between the VN and cerebellum (Arends and Zeigler 1991), or between the VN and the periphery. There is evidence (Donaldson and Knox 1990) that proprioceptive signals from extra-ocular muscles in pigeons have excitatory and inhibitory effects on VN neurons. Indirect support for the existence of velocity storage at the level of the VN in primates is offered by the finding that the dominant time constant governing the frequency response characteristics of horizontal canal driven VN neurons is similar to τ_v for the HVOR in aroused rhesus monkeys (Buettner et al. 1978). A similar investigation of the dynamics of VN neurons in pigeons remains to be done.

In primates, the velocity storage mechanism is thought to be shared between the vestibular and optokinetic systems (Robinson 1981). The two systems are assumed to join at the VN level, because primate VN neurons also respond to optokinetic stimuli (Waespe and Henn 1979). Although pigeons clearly respond to whole-field, moving visual stimuli with optokinetic nystagmus (OKN) (Gioanni 1988a), whether or not avian VN neurons also respond to optokinetic stimuli has not been determined.

The VN signal is inverted to produce VOR eye rotation commands that are opposite to head rotations. To insure correct polarity for OKN in the pigeon vestibulo-optokinetic model (Fig. 3) the optokinetic signal must be inserted into the inner, *negative* feedback loop *after* the VN signal is reinverted. This is in contrast to the *positive* inner feedback loop in the monkey model (Robinson 1981), in which the optokinetic signal was inserted *before* the VN signal was reinverted. Optokinetic signals in pigeons are carried by neurons in the accessory optic system and pretectum (Morgan and Frost 1981; Winterston and Brauth 1985). Anatomical data in pigeons reveals extensive projections of these neurons to the VN, either directly or indirectly via the cerebellum and reticular formation (Brauth and Karten 1977; Brecha et al. 1980). The available physiological and anatomical data would be consistent with any number of possible brainstem connectivity patterns and do not, in any case, preclude the arrangement of retinal slip and vestibular signals depicted in Fig. 3.

In pigeons, available evidence does not fully support a sharing of velocity leakage between the vestibular and optokinetic systems. The shared vestibulo-optokinetic model in Fig. 3 predicts that closed-loop horizontal and vertical OKN gain in alert pigeons (with $G_v = 2$) should be 0.43. This is close to the gain value measured for vertical OKN in alert pigeons (0.45), but horizontal OKN gain is almost 1 (0.95) in these alert birds (Gioanni 1988a). These discrepancies indicate that the optokinetic system may, at least in part, circumvent the velocity leakage mechanism in pigeons. In this regard, it is interesting to note that a projection from some accessory optic neurons directly to cyclovertical extra-ocular motoneurons has been observed in birds but not in mammals (McKenna and Wallman 1985).

5.4 The role of velocity leakage

A study of the vestibulo-collic reflex (VCR) and the VOR in alert pigeons illustrates that both reflexes work together to achieve gaze stabilization (Gioanni 1988b). The VCR accounts for 80% of gaze stabilization, which may explain the relatively low gain we observed for the VOR in pigeons under most circumstances (Table 1; Anastasio and Correia 1988). It is reasonable to suppose that the function of velocity leakage would be to shape the VOR to better augment VCR (Outerbridge 1969), yet combined VCR/VOR performance is still poor at lower frequencies (Gioanni 1988b). From the available data and the model, it is difficult to escape the conclusion that the function of velocity leakage is to diminish the response of the pigeon VOR to low frequency, low amplitude head rotations. Whatever behavioral advantage velocity leakage may confer upon the pigeon remains to be discovered.

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