Mechanisms Controlling Human Head Stabilization. I. Head-Neck Dynamics During Random Rotations in the Horizontal Plane

E. A. KESHER AND B. W. PETERSON

Sensory Motor Performance Program, Rehabilitation Institute of Chicago; and Department of Physiology, Northwestern University Medical School, Chicago, Illinois 60611

SUMMARY AND CONCLUSIONS

1. Potential mechanisms for controlling stabilization of the head and neck include voluntary movements, vestibular (VCR) and proprioceptive (CCR) neck reflexes, and system mechanics. In this study we have tested the hypothesis that the relative importance of those mechanisms in producing compensatory actions of the head-neck motor system depends on the frequency of an externally applied perturbation. Angular velocity of the head with respect to the trunk (neck) and myoelectric activity of these three neck muscles were recorded in seven seated subjects during pseudorandom rotations of the trunk in the horizontal plane. Subjects were externally perturbed with a random sum-of-sines stimulus at frequencies ranging from 0.185 to 4.11 Hz. Four instructional sets were presented. Voluntary mechanisms were examined by having the subjects actively stabilize the head in the presence of visual feedback as the body was rotated (VS). Visual feedback was then removed, and the subjects attempted to stabilize the head in the dark as the body was rotated (NV). Reflex mechanisms were examined when subjects performed a mental arithmetic task during body rotations in the dark (MA). Finally, subjects performed a voluntary head tracking task while the body was kept stationary (VT).

2. Gains and phases of head velocity indicated good compensation to the stimulus in VS and NV at frequencies <1 Hz. Gains dropped and phases advanced between 1 and 2 Hz, suggesting interference between visual and mechanical components. Above 3 Hz, the gains of head velocity increased steeply and exceeded unity, suggesting the emergence of mechanical resonance.

3. At low frequencies (<1 Hz) during MA, gains were very low, and phases indicated that the head was moving with the trunk. A steady rise in gains and shift in phases toward a compensatory response were observed as frequency increased. Between 1 and 2 Hz, the response of the neck moved toward compensation as gains observed during voluntary stabilization decreased, suggesting that reflex mechanisms were becoming the predominant controller of compensatory processes at this frequency range. Around 3 Hz, mechanical resonance was observed.

4. In VS, NV, and MA, electromyographic (EMG) activity steadily decreased in gain up to 1 Hz, then continuously increased at frequencies >1 Hz. This implied sustained participation of neural mechanisms in the higher frequency range. Depending on the relative motion of the head with respect to space and to the trunk, either the vestibulocollic or cervicocollic (proprioceptive) reflex were assumed to be present in the EMG output.

5. The patterns observed in the neck responses secondary to trunk perturbations were not apparent in the response dynamics of voluntary head tracking. In VT, the most compensatory gains and phases of both head velocity and muscle EMG responses appeared at the lowest frequencies of head movement. Gains steadily declined, and phase lags increased as frequency increased.

6. We acknowledge that the contributions of the three mechanisms examined here cannot be completely separated by the paradigms used, but the data suggest that reflexes do participate in the stabilization process. Comparisons of the frequency responses of the cat and human showed that a model based on the passive mechanics of the cat's neck is applicable to these data even though experimental conditions were different. Evidence of a similar pattern of gains and phases in our data to that of the animal model allows us to conclude that the observed activity of the head with respect to the trunk in this series of experiments is indicative of a process of compensatory head stabilization as a consequence of trunk movements caused by external forces.

INTRODUCTION

The head serves as the frame of reference for detection of motion by the visual and vestibular systems, and as such, remains stable relative to the environment during natural movements like locomotion (Pozzo et al. 1990; Winter 1991). Potential mechanisms for controlling stabilization of the head and neck include voluntary movements, vestibular (VCR) and proprioceptive (CCR) neck reflexes, and system mechanics. The bulk of evidence suggests, however, that neck reflexes offer an insignificant contribution to stabilization of the head. For example, in studies of passive head angular accelerations in humans, investigators were unable to identify any reflex compensation in the horizontal and vertical planes during predictable sinusoidal rotations of subjects seated in the dark (Barnes and Rance 1974, 1975).

The predictability of the stimulus may have been the reason reflex responses did not emerge. But Guittin et al. (1986) examined the influence of attentional set on head stabilization in humans and found that the short-latency VCR and CCR produced little effective head stabilization in humans even with a white noise stimulus with a bandwidth of 0–1 Hz. Normal subjects and patients with bilateral vestibular deficit were tested on their ability to stabilize their heads while rotated horizontally. Subjects were asked to stabilize while receiving visual feedback, or were distracted with mental arithmetic. Both healthy and vestibular deficient subjects stabilized their heads best when voluntarily keeping the head coincident with a stationary visual target. Although vestibular inputs were seen to provide a necessary signal to intentional stabilization of the head in the dark, the apparent lack of head stabilization when all subjects performed mental arithmetic argued against the participation of reflexes in this paradigm.

Mechanical properties of the neck muscles were considered responsible for the greater portion of compensation to an external force that disturbed the trajectory of rapid head movement in an anesthetized, rhizotomized, and vestib-
FIG. 1. Mean ± SE of harmonic distortion and signal/noise ratio values of all subjects' head angular velocity measures during voluntary stabilization with visual input (VS), voluntary stabilization without visual input (NV), mental arithmetic (MA), and visual tracking (VT).

ullectomized monkey’s head (Bizzi et al. 1978). Neck muscle stretch reflexes (including the CCR) were found to be responsible for <10–30% of compensatory torques. Richmond and Loeb (1992) have also reported the absence of significant muscle activation by excitatory monosynaptic reflexes in the neck of alert, behaving cats.

It could appear from the data to date, that reliance on the vestibular and cervical reflexes to stabilize the head and neck may diminish as we ascend the evolutionary scale or may be related to the vertical position of the head and neck in space (Vidal et al. 1986). But weaknesses exist in the majority of studies that examined the mechanisms producing stabilization of the head. Either they were performed in reduced preparations, failed to control the task constraints (Graf and Wilson 1989), or examined parameters that were not necessarily in the range of functional movement. When Goldberg and Peterson (1986) studied an alert cat preparation at frequencies up to 4 Hz, they concluded that the VCR and CCR dominated head stabilization.

In this study we tested the hypothesis that the emergence of each mechanism controlling head stabilization is frequency dependent. Responses were recorded during rotations in the horizontal plane (yaw) to compare their characteristics with the animal data and models. Our results indicate that reflex stabilization of the head may, in fact, compensate for diminished control by voluntary mechanisms at high frequencies of externally applied perturbations. In the next paper, responses to vertical plane rotations (pitch) are examined. Preliminary results from these data have been reported previously (Keshner and Peterson 1988a,b).

METHODS

Seven subjects with no history of neurological or oculomotor problems, ranging in age from 25 to 40 yr, gave their informed consent to participate in this study. One of these subjects also participated in the vertical plane paradigm presented in the next paper (Keshner et al. 1995), and those electromyographic (EMG) data will be presented for comparison in all but Fig. 1 of both papers. Subjects were seated in a rigid, molded chair that provided support to the whole body, so that the feet were placed flat at the bottom of the chair, and the knees were raised. The chair was coupled to a high torque (500 ft lb), servo-controlled rotatory turntable (Contraves) with the earth vertical axis of rotation passing through the head. A form-fitting vacuum cast, shoulder, lap, and leg belts, and a chest-level metal gate were used to secure the subject firmly and minimized relative movement between the torso and the rotating chair. The head was free to rotate in any plane, but measures of angular velocity in the pitch and roll planes proved to be insignificant. All measures of chair velocity and position were treated as equivalent to trunk velocity and position following a pilot study (Keshner and Peterson 1988b) confirming that measures of trunk angular velocity exactly matched chair angular velocity at all frequencies. The entire apparatus was enclosed in a light-tight room.

Subjects were fitted with a triaxial angular rate sensor (Watson Industries, WI) positioned at the vertex of the head. Visual stimuli used in these experiments consisted of two projected spots. One stimulus was produced by a servo-controlled projector mounted above the subject’s head and projected onto a cylindrical wall 1.6 m in front of the subject. It provided a fixed spot for visual reference during body rotations, and a moving spot that served as a target during visual tracking runs. The other stimulus was projected from a light weight fiber optic mounted on the headband in order to provide accurate feedback of head position. Total weight of the headband assembly did not exceed 750 g.

Procedure

Subjects began each trial sitting in a stationary, vertically upright orientation. Subjects received a minimum of one complete cycle of stimulation before data collection began in each trial, so that each trial lasted ~400 s. Data were collected across a 200-s period for each of four instructional sets in the following order. Voluntary stabilization (VS) required that the subject keep the head-referenced light spot coincident with a stationary target spot while the chair was rotated. The no vision condition (NV) was performed in the dark while the subject was given the task of stabilizing the head by imagining both the stationary target spot and the head-referenced light signal. During mental arithmetic (MA), a mental calculation task was provided so that the subject’s attention was removed from the task of stabilizing while rotation in the dark was ongoing. System dynamics during voluntary movement as opposed to head stabilization was also assessed via a visual tracking task (VT). In this case the target spot was moved, the body remained stationary, and subjects were instructed to follow the moving target with the head-referenced light spot.

Stimulus

The velocity command provided to the chair was a sum of 10 sinusoids (SSN) that consisted of relatively prime (i.e., having no common divisors) harmonics of a common base frequency. This stimulus was used to characterize the subject’s response to an unpredictable stimulus. The SSN waveform used for all subjects in this study was composed of the following harmonics: 37, 49, 71, 101, 143, 211, 295, 419, 589, and 823. By using a fundamental frequency of 0.005 Hz, we were able to characterize the response for frequencies from 0.185 to 4.117 Hz. Chair velocities decreased as frequency increased as follows: 20°/s from 0.185 to 0.355 Hz, 19°/s from 0.505 to 1.056 Hz, 16°/s from 1.476 to 2.096 Hz, 15°/s at 2.947 Hz, and 13°/s at 4.117 Hz. The largest excursion of the chair (±17°) occurred at the lowest frequency of rotation. In each trial, data were collected over a single period of the fundamental so that the stimulus pattern did not repeat during data collection. For the VT condition, the light spot was driven with the same velocity command provided to the chair. Peak velocity of the laser stimulus was 30°/s, and the peak excursion was ±7°. Two of our
subjects were also tested with a predictable 0.1-Hz sinusoid in order to examine the direction of trunk rotation that produced maximal muscle EMG responses. Peak excursion of this stimulus was ±30°, and peak velocity was 18°/s.

Data collection and reduction
Feedback from a tachometer and potentiometer on the torque motors supplied measures of chair angular velocity, chair position, and laser position, respectively, and were recorded on a PDP 11/23 computer. The computer acquired analog data in synchrony with the process that generated the SSN wave to control the rotating chair. Incoming data were binned so as to produce a 64 point cycle average at each of the 10 component frequencies of the SSN wave. The resulting 64 point data records were fit with sinusoids at the component frequency (1st harmonic) and at twice that frequency (2nd harmonic) plus a constant offset (DC) term with the use of a least-squares procedure. The ratio of the amplitude of the second to that of the first harmonic gave a measure of harmonic distortion. Signal-to-noise ratio was defined as the ratio of the amplitude of the first harmonic signal to the square root of the unif variance of the least-squares fit. Signal-to-noise ratios and harmonic distortion values are presented in Fig. 1. Signal-to-noise ratios were most robust in the two nonvision conditions (NV and MA) with values approaching 10. Ratios of both visual stimulus conditions (VS and VT) increased with frequency, and only VT exhibited ratios <1 at the 2 lowest frequencies. Harmonic distortion did not exceed 30% at any frequency and was noticeably lower in MA at frequencies <1 Hz. We believe our use of linear analysis methods is supported by this data.

Surface EMG recordings were taken from the right semispinalis capitis (SEMI), right splenius capitis (SPL), and right sternocleidomastoid (SCM) muscles by pairs of Ag-Ag Cl electrodes, 4 mm diam and spaced 1 cm apart. Electrode placements have been verified anatomically and physiologically in our study of isometric head stabilization (Keshner et al. 1989). Electrode placement for SEMI is 2 cm below the occipital bone at approximately C1–C2, and 2 cm lateral to midline. The muscle is palpable when the subject performs pitch extension of the head. Electrode placement for the SCM muscle can be palpated when the subject performs contralateral rotations (i.e., leftward rotations for the right-sided muscle) of the head against resistance placed at the chin. Electrodes were placed over the SCM muscle belly, approximately one-third of its length rostral to its sternal attachment. Almost all of SPL lies underneath the trapezius and SCM muscles except for a rectangular area on the lateral portion of the neck where SPL is the most superficial muscle. This muscle can be palpated during resisted head extension with ipsilateral rotation. Electrode placements for the SPL muscle were determined by measuring 6 cm rostral to the bony prominence at C3 (approximately the C4 level), 6–8 cm lateral, and palpating for the muscle belly.

During recording, EMG potentials of the two neck muscles were amplified, band-pass filtered (10–200 Hz), full-wave rectified, and integrated (20-ms time constant). To prevent aliasing, all signals were filtered with an 8-pole low-pass Bessel filter with a corner frequency of 20 Hz (Frequency Devices, MA) before digitization. Signals were viewed on a monitor during testing and stored in digital form on a PDP 11/23 computer for later reduction and analysis.

Data analysis
Head and chair angular velocity data from all pseudorandom rotation paradigms (VS, NV, and MA) will be presented as a response ratio vector representing angular velocity of the neck with respect to (wrt) the trunk. Angular velocity of the neck was derived from the vectorial difference between head and chair angular velocities at each stimulus frequency. Gain of the response ratio vector is described by its length and is equal to the response/stimulus amplitude ratio; phase is equal to the difference between response and stimulus phase angles. In the head tracking task (VT), movement of the head reflected movement at the neck because the trunk was stationary.

In the three moving chair tests, phase is expressed relative to chair velocity. As illustrated in Fig. 2, neck velocity in the VS condition has a phase close to −180° when the neck is producing a peak leftward velocity to fully compensate for the peak rightward chair velocity, so that the head is held still. Thus a phase difference of ±180° indicates that the neck is moving in a direction that fully compensates for chair peak angular velocity. Phases of 0° indicate that the neck is not compensating, and the head is moving in phase with the chair (note the MA condition in Fig. 2). Lead of the neck with respect to the trunk is seen as a neck phase between 0 and −180°. Lag of the neck with respect to the trunk is seen as phases between −180° and −360°. A gain of one and phase of 180° represents perfect compensation of the head (or no movement of the head relative to space). Smaller gains indicate that the head is moving at smaller velocities than the chair (i.e., undercompensating for the stimulus). Gains greater than one indicate that the head is moving at higher velocities than the chair. Vector averages for the population of subjects were derived by averaging the real and imaginary components of each subject’s gain and phase values, and then calculating the gain and phase of the response ratio vector.

For the head tracking task (VT), movement of the head is more relevant than that of the neck. Thus the data will be presented as a response ratio vector indicating head velocity with respect to target velocity. In this condition, the ideal response is represented as a gain of one and phase of 0° to indicate that the head is moving in phase with the target.

EMG responses were analyzed in a more descriptive fashion. Gains of the muscle EMG responses were collected in arbitrary units and are compared only within a muscle and across instructional sets. Interpretation of phase results in each case will depend on the known actions of the muscles; thus phase responses of each muscle are presented with respect to peak chair position or velocity. As illustrated in Fig. 2, response phases around +90 and −90° in the muscles indicate responses in phase with peak left (SEMI and SPL) or peak right (SCM) position of the chair, respectively. This result is appropriate to the functional activation of these muscles because right SEMI and SPL muscles tend to produce rightward rotation of the head, and right SCM produces leftward rotation of the head (Keshner et al. 1989). A muscle response phase of 0° suggests that the muscle is responding in phase with peak rightward velocity of the trunk; a −180° phase indicates a response related to peak leftward velocity of the trunk.

RESULTS

Response characteristics during head stabilization

To characterize the muscle EMG responses, a simplified analysis was performed with a predictable 0.1-Hz sinusoid. Data from one subject in Fig. 2 illustrate that head velocity and EMG activity varied with instruction. At this low frequency of rotation, head velocity stayed close to zero as the head was voluntarily stabilized during VS. In NV, the head was not as well stabilized, and a small movement in phase with chair velocity began to appear. To keep the head stationary in space in these two conditions, the subject produced angular velocities at the neck that compensated for chair velocity (note phases close to −180°). When attention was diverted during MA, head velocity in space nearly matched that of the chair velocity in space, resulting in a
greatly diminished neck angular velocity. In VS and NV, the right SEMI muscle was lagging chair velocity, but leading leftward position of the trunk (downward peak in bottom trace). This muscle turns the head to the right and is showing peak activity close to maximum leftward trunk turning. Because the primary action of this muscle actually is extension (Keshner et al. 1989), it will not be presented in the following figures. The EMG response of the right SPL muscle was also lagging chair velocity but was phase leading peak leftward position of the trunk so that its peak activity fell between peak leftward velocity and position. In VS, right SCM activity was in phase with rightward position of the trunk and lagging peak rightward velocity by $\sim 90^\circ$ (right SCM produces a leftward rotation of the head because of its insertion point on the medial clavicle). The same EMG response was observed in NV, but in this trial the subject produced additional activity in phase with rightward chair velocity; thus phase lagged rightward velocity by only $12^\circ$ and led position of the trunk by $78^\circ$. None of the muscles exhibited a response in MA at this frequency.

In Fig. 3, the neck wrt trunk responses of all subjects to the random SSN are presented as Bode diagrams. The solid line in each diagram represents the vector average for the group, and the shaded area is $\pm 1$ SE for the vector average (the dashed line will be discussed later). Standard errors of this three-dimensional data were derived by first calculating standard errors of the real and imaginary components, and plotting these as $x$ and $y$ error bars at each gain point on the vector average. A rectangle was drawn around the error bars, and a diagonal drawn through the rectangle. The length of the diagonal was taken as a conservative estimate of error gain. The angle subtended between the average gain and the error gain was calculated as a conservative estimate of phase error.

At frequencies $< 1$ Hz, good compensation of the head (gains equal to 1 and phases equal to $-180^\circ$) occurred in the VS and NV conditions. Removing visual feedback in the NV condition caused lower response gains and more intersubject variability. Markedly different response characteristics occurred in the MA condition at low frequencies. In this condition, gains fell around 0.1 at the lowest rotation frequencies and steadily increased with frequencies $> 0.4$ Hz. In six subjects, phases began close to the compensatory $-180^\circ$ observed in VS and NV, led compensation at intermediate frequencies, and then shifted back to compensation at high frequencies. A single exception exhibited $0^\circ$ phase at low frequencies, characteristic of a purely passive mechanical response, but agreed with the other subjects above 1 Hz. At frequencies $> 2$ Hz, gains and phases in all three conditions presented a similar picture with gains exceeding 1.0. Gains and phases for the 0.1-Hz sinusoidal data shown in Fig. 2 are replotted here on the vertical axis. They demonstrate that the pattern remains consistent for both complex and simple stimuli.

In the two voluntary stabilization conditions (VS and NV), neck wrt trunk responses exhibited a noticeable drop-off to gains of 0.2–0.4 starting around 1 Hz and continuing until $\sim 2$ Hz. A coincident $90^\circ$ phase shift occurred between the stimulus frequencies of 1 and 2 Hz. Although the trend was accentuated by the vector average, individual data sets all exhibited this reduction in gain. Diminished gains and increasing phase errors suggest that voluntary mechanisms were failing to maintain good compensation of the head. Although the gain drop was not observable in MA, the slope of its rise became less steep (see arrow). Above 2 Hz in all conditions, phases dropped off, and gains demonstrated a steep rise. Because gains greater than one are indicative of a head that moves more than the trunk, we can infer the beginning of resonant oscillations at these high frequencies, and that neural control of stabilization no longer dominates the response.
Muscle EMG responses revealed modulation of neck muscle activity across all conditions. When analyzed with respect to chair velocity (see Fig. 4), EMG responses of SCM in this subject exhibited high response gains in VS and NV, and a much diminished response in MA. Examination of SCM responses across most of the subjects revealed that this muscle’s output tended to decrease by a factor of 10 in the MA condition, suggesting that it participated more in voluntary than reflex movement in the horizontal plane. SPL EMG activity exhibited a gradual decline below 1 Hz, then began to increase steeply in a consistent fashion. Such increasing activity would be suggestive of a stronger participation of this muscle in reflex head stabilizing responses as would occur if the neck reflexes were becoming predominant in this frequency range.

SCM exhibited a steady 0 to −90° response phase (between peak rightward velocity and position) in all three instructional sets, that would be expected from a muscle responding preferentially to voluntary mechanisms; responses under the control of reflexes would be expected to show frequency-related phase shifts like those seen in SPL. In the VS and NV conditions, activation of SPL underwent a phase shift from a −90° phase-related to peak rightward chair position to a +90° phase-related to leftward chair posi-

![Diagram](image-url)
tion as it approached 1 Hz. Particularly worth noting is the anticomparatory phase of SPL responses at low frequencies in the VS and NV conditions. In the predictable sinusoid condition (Fig. 2), SPL exhibits compensation that is out of phase with SCM (note that SPL produces rightward rotations of the head, whereas SCM produces leftward rotation of the head). Yet in the random stimulus conditions, SPL and SCM are roughly in phase at low frequencies. Given the low gains of these responses to random rotations, they probably do not represent voluntary activity, but rather reflex activation dominated by the CCR. This is considered further in the DISCUSSION.

In MA, SPL maintained a steady 90° phase lead of rightward chair velocity (i.e., in phase with peak left position of the chair), until the phase drop observed in all conditions above 2 Hz. The low-frequency activity is presumably mediated by the VCR, and the phase drop above 1 Hz coincides with an increasing head movement gain that exceeds 1.0 above 2 Hz (see Fig. 3 and inset in Fig. 4). At these high frequencies, the input driving the VCR will reverse, and the CCR will act in phase with this reversed VCR to help overcome head resonance. The changing activation of SPL indicated by the phase reversal seen in Fig. 4 between 1.5 and 4 Hz presumably reflects these two reflex actions.

Response characteristics of head tracking

Responses of the head and neck muscles were also examined during voluntary head tracking (VT). Perfect visual tracking was represented by a gain of one and a phase of 0° on the Bode plots of head velocity with respect to velocity of the visual target (Fig. 5). Results suggest that the subjects were most successful in matching the target at frequencies up to 1 Hz. At frequencies >1 Hz, head velocity responses exhibited decreasing gains coincident with progressive phase lags. Also plotted in Fig. 5 are the EMG responses during active head tracking. Responses of the two muscles were in phase with leftward head velocity at low frequencies of active head movement, and phase lags steadily increased with frequency.

DISCUSSION

Potential mechanisms for controlling stabilization of the head and neck include voluntary movements, vestibular (VCR) and proprioceptive (CCR) neck reflexes, and system mechanics. In this study we have tested the hypothesis that the relative importance of those mechanisms in producing compensatory actions of the head-neck motor system depends on the frequency of an externally applied perturbation. Sensory inputs and attention to the task were controlled through visual feedback and instructional sets. Important clues about the roles of these three mechanisms come from the head movement data plotted in Fig. 3. The consistency of the behavior shown across our seven subjects indicates that such roles are similar in all individuals. To interpret these data, we must first consider the expected dynamic properties of compensatory head movements generated by voluntary, reflex, and mechanical mechanisms.

The voluntary system is likely to generate a compensatory signal with phase opposite to that of the imposed trunk rotation and with a relatively long central delay on the order of 0.2 s. The data in Fig. 5 indicate that this is indeed the case for the visual tracking component. Muscle output gain is relatively constant with a suggestion of low-pass roll-off above 1.5 Hz, whereas phase shows a progressively increasing lag that may reflect this low-pass behavior and, more importantly, a large central delay. Voluntary mechanisms can only contribute to accurate head stabilization for frequencies <1 Hz. As a result, head movements in VT show a strong low-pass like falloff with increasing frequency. The slope of the gain curve in the VT condition reaches -40
the behavior of this system in a deeply anesthetized cat. Because the mechanics are dominated by head inertia and neck elasticity, the behavior has a single critical frequency at which movement makes a transition from a signal with rising gain (40 dB/decade) and anticom pensatory 0° phase to one with constant gain of 1.0 and compensatory phase of −180°. In the former regime, neck elasticity is causing the head to move along with the trunk. In the latter, the head’s inertia is causing it to stand still in space, thus effecting a perfect compensation. Between the two regimes, phase passes through −90° and gain overshoots to a value >1.0. One can see signs of such behavior at the upper end of the Bode diagrams in Fig. 3. In each case, gain is rising at ~40 dB/decade (the slope of the dashed lines) at 2–3 Hz, where it exceeds 1.0. In the NV and MA records, there is an indication that this gain increase is slowing at 4 Hz, presumably to return to 1.0. Correspondingly, phase lag relative to the stimulus is increasing from about −90° at 2 Hz to −120° to −150° at 4 Hz. Thus neither phase nor gain has reached the expected inertially dominated response of gain 1.0, phase −180°, although both parameters appear to be approaching this limit. The indication from these data is that the human head is behaving like a second-order low-pass system with a resonant frequency of 2–3 Hz.

If we accept that our MA paradigm eliminates voluntary head stabilization, the EMG signals we observed in that condition must be produced by head-stabilizing reflexes: the VCR and CCR. In the SCM muscle, reflex EMG activation is quite modest, <3% of the voluntary activation observed in the VS and NV conditions. Our impression is that reflex activity is comparable in SPL with the difference that this muscle did not exhibit additional voluntary activation in VS and NV. The rising gain of the EMG response in each muscle at higher frequencies resembles the large increase in gain of the VCR observed during whole body rotation in the decerebrate cat (Baker et al. 1985; Bilotto et al. 1982). In the present experiment, however, the phase behavior is complicated by the fact that the head is free to move on the trunk. By correlating data in Figs. 2 and 3, we can deduce what is occurring. Beginning with SPL, the compensatory low-frequency activation in phase with leftward position of the body during MA likely reflects activation of the VCR in this situation where head and body move together. The opposite phase, anticom pensatory activity seen in VS (and to a lesser extent in NV) likely reflects activation by the CCR. Compensatory head rotations seen in those conditions would both elicit and oppose the CCR. The VCR will not contribute much here because the head is held essentially stable in space, minimizing vestibular input.

By 1 Hz the circumstances to which SPL responds have changed. Phase lags, and gain decreases in the case of NV, have increased the error in head stabilization so that significant semicircular canal activation is occurring. EMG output appears to be dominated by a VCR signal with a compensatory phase. Around 2 Hz, the neck rotation phase reverses again, and the head movement gain, plotted in Fig. 3, is now >1.0 so that head movement in space is opposite to the direction of body rotation. Thus the VCR will be driven in the reverse direction, giving the observed phase. It will also be assisted by the CCR, which continues to have a phase opposing head rotation. The net effect of these two signals

FIG. 6. Head movement evoked by a sum-of-sines (0.185–4.12 Hz) horizontal body rotation in an anesthetized cat. Gains and phases of head movement were computed relative to body position. Zero degree phase and 0-dB gain indicate head movement in the same direction and of the same amplitude, respectively, as body movement. Phases near 0° and rising gains indicate that muscle stiffness is causing the head to move with the trunk. Phases near 180° and a gain of 1 (10 dB) indicate that the head’s inertia is causing it to stand still in space. Open and closed symbols represent responses obtained with normal and doubled moment of inertia of the head, respectively. Each of the 2 sets of data is fitted by a function of the form $-Is^3/(Is^3 + Bs + K)$, where $I$ is inertia of the head, $B$ is the viscous forces of the neck muscles, and $K$ is the elastic forces of the neck muscles (taken from Goldberg and Peterson 1986).

dB/decade at 2 Hz (it becomes even steeper above 2 Hz). Thus below 2 Hz, the gain and phase data closely resemble a second-order system with a natural frequency of 1 Hz plus a delay of ~0.2 s. There is no sign of the phase lead in the EMG response that appears in the cat’s VCR at higher frequencies (Bilotto et al. 1982; Goldberg and Peterson 1986; Peterson et al. 1985), presumably reflecting central compensation by VCR circuits for the low-pass properties of the head-neck motor plant.

The mechanical system is likely to exhibit the same second-order passive behavior as was observed in the cat by Goldberg and Peterson (1986). These investigators analyzed the response of a passive mechanical head-neck system and the participation of reflex elements in alert cats by considering the dynamics of the closed-loop horizontal VCR and CCR, the low-pass properties of the neck muscles, and the inertial load of the head. They found that gains of compensatory head movements elicited by horizontal body rotations in the alert cat remained relatively constant in the frequency range of 0.1–2.0 Hz and then exhibited a rapid rise when the head movement became dominated by mechanical characteristics.

Figure 6, which is reproduced from their paper, illustrates...
will be to help oppose the resonant head motions produced by head-neck mechanics.

The characteristics of the SCM response are less obvious. EMG output appears to be dominated by a compensatory signal, which is probably of voluntary origin at lower frequencies in VS and NV with an additional reflex component coming on above 1 Hz in all conditions. There are signs of the expected reversal of EMG phase when head movement gain exceeds 1.0, especially in the MA records, but this effect is less evident than in SPL. Perhaps phase advances in the reflex signal are shifting EMG output from the expected reverse compensatory phase of $-270^\circ$ (the equivalent of $+90^\circ$) to the $-130$ to $-150^\circ$ observed.

The above analysis suggests that reflexes are contributing to the behavior of the SPL and SCM muscles in our tasks. Can we then detect their contribution to head motion? This is difficult. At low frequencies, reflex activity is small and will be overwhelmed by larger contributions of voluntary mechanisms. At high frequencies the reflexes appear to be working to damp resonant motions that are generated by head-neck mechanics. The logical place to look is at an intermediate frequency in the MA condition where the voluntary contribution is minimal. As indicated by the arrow in Fig. 3, the smoothly rising gain predicted by a second-order mechanical model is interrupted around 1 Hz by a lessening of the 40-dB/decade slope. This may indicate the interaction of reflex and mechanically generated forces. The apparent dip of gains and phases in the same frequency range in VS and NV may also indicate a shift from a voluntary to a reflex mechanism for control of head stabilization. The decreased gains in VS and NV equal those of the more compensatory response of the neck in MA, suggesting that reflex mechanisms are becoming the predominant controller of head stabilization at this frequency range; increased activation of muscle EMG implies that control was generated by a neural mechanism.

Numerous studies have investigated the inherent sensitivity to stimulus frequency found in the semicircular canals and utricular otoliths, and exhibited through their reflex activity (see Schor et al. 1988 for review). In most of these studies, the closed-loop path of the horizontal VCR was made open-loop by preventing the head from turning on the neck at frequencies up to 1 Hz. In the decerebrate cat, EMG response phases of an open-loop VCR was found to lag the angular acceleration stimulus by nearly $180^\circ$ at 0.1–0.2 Hz. When tested at higher frequencies, phase responses advanced as frequency increased so that the decerebrate VCR response lagged angular acceleration by only $40^\circ$ at 4 Hz (Bilotto et al. 1982; Ezure and Sasaki 1978); this behavior suggested a change from a response related to head position to one related to head angular acceleration (Peterson et al. 1985; Schor et al. 1986). Keshner et al. (1992) have demonstrated that neck muscles in alert cats during voluntary head tracking exhibited responses related to head position, whereas the same cats exhibited responses related to head velocity during stimulation of the VCR.

When actively stabilizing or actively tracking with the head, EMG responses at the lowest frequencies led head motion as expected from the reaction-time movements observed by Guitton et al. (1986). Diminishing EMG activation and increasing phase lags as frequency increased during head tracking would be expected from a paradigm like our VT that required only voluntary control and did not supply the necessary stimulation to elicit reflex mechanisms. EMG activity in our VS and NV paradigms only dropped until $\sim 1$ Hz. Then, at frequencies $>1$ Hz, EMG activity increased with increasing head motion, from which we can infer that both the VCR and CCR were contributing to stabilization. Phase shifts from a position-related phase lag to that of a phase lead between 1 and 2 Hz further support the assumption of reflex-controlled compensation of the head. Although we acknowledge that the contributions of the three mechanisms examined here cannot be completely separated by the paradigms used, we would argue that voluntary, reflex, and mechanical mechanisms all contributed to final head position during externally imposed perturbations, each becoming dominant at a different frequency range.

We thank Drs. Robert Kearney and Daniel Guitton for assistance and insights in the early phases of this research. Drs. Fred Prior and Ronald Kettner for critical comments on this manuscript, and R. Cromwell and G. Rovai for technical assistance.

This study was supported by National Institutes of Health Grants NS-22490 and DC-01125.

Address for reprint requests: E. Keshner, Sensory Motor Performance Program, Rm 1406-E809, Rehabilitation Institute of Chicago, 345 East Superior St., Chicago, IL 60611.

Received 26 October 1992; accepted in final form 1 February 1995.

REFERENCES


