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Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficit

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Summary. The experiments were designed to test two hypotheses and their corollaries: 1. That adaptation of EMG responses to support surface rotations is due to a decrease in the gain of proprioceptively triggered long-loop stretch reflexes (Nashner 1976), and that the adaptation is dependent on a normally functioning vestibular system (Nashner et al. 1982); 2. That EMG responses to rotations are generated primarily by vestibulo-spinal reflexes triggered by head accelerations (Allum and Pfaltz 1985) and comprise a coactivation of opposing leg muscles (Allum and Büdingen 1979). Adaptation with successive dorsi-flexive rotations of the support surface was investigated in the EMG responses of the ankle muscles, soleus (SOL) and tibialis anterior (TA), as well as the neck muscles, trapezius (TRAP) and splenius capitis (SPLEN CAP), both for normal subjects and for patients with bilateral peripheral vestibular deficit. Both normals and patients who first received the stimulus with their eyes open demonstrated decreasing activation at medium latency (ML), that is, with an onset at about 125 ms, and long latency (LL) responses with an onset ca 200 ms. This was the case for both ankle and neck muscles when the EMG response areas for the first 3 and second 7 of 10 trials were compared. Ankle muscle responses in the patients were diminished in area with respect to normals both with the eyes open and with the eyes closed. Ankle torque recordings from the patients were also smaller in amplitude, and these attenuated differently from normal torque responses. Functional coupling of the opposing ML and LL SOL and TA muscle responses was confirmed by the nearly coincident onset times and significantly correlated EMG response areas. At ML, ankle torque was highly correlated with TA activity when the influence of SOL was controlled. At LL, SOL activity was highly correlated with torque when the influence of TA was controlled. The delay of torque adaptation beyond the period of ML activity in normals, but not in the patients was attributed to the proportionally balanced coactivated muscle patterns producing a consistent force output and level of stability in normals. The results indicate that the adaptation in EMG response amplitudes during a sway stabilisation task is not dependent on a normally functioning vestibular system nor on visual inputs but rather appears to be due to a generalized habituation in the postural control system. Evidence against a change in the gain of proprioceptively triggered long loop reflexes being responsible for adaptation is based on the fact that the adaptation is not restricted to the stretched SOL muscle but includes its agonist, TA, and that the adaptation is not local but also occurs in neck muscles. The results supported the hypothesis that postural reflexes to support surface rotations may well be triggered by stretch reflexes in the lower leg or neck muscles, however, their amplitude modulation is overwhelmingly under the control of vestibulo-spinal signals.

Key words: Postural control – Vestibulo-spinal reflexes – Peripheral vestibular deficit

Introduction

Three neural systems, visual, vestibular, and ankle proprioceptive have been identified as the primary afferent pathways for the control and organization of rapid postural responses in the standing human

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(Allum and Pfaltz 1985; Bussel et al. 1980; Diener et al. 1984b, c; Lestienne et al. 1977; Nashner 1976; Nashner and Berthoz 1977). Early reports concentrated on the temporal arrangement of the postural pattern in the lower limb, and emphasized that the proprioceptive input ascending from the ankle joint was directly responsible for the sway stabilizing responses taking place at 90–120 ms (Nashner 1972; Nashner 1976; Nashner et al. 1982). In this argument, the same responses that were functional when the body was induced to sway forward by rearward support surface translation, producing increased ankle dorsiflexion and a stretch on the triceps surae, would no longer be functional when the ankle was directly rotated into dorsiflexion. Then, the induced body sway would be posterior, and EMG activity from a stretched tricpes surae would only pull the center of mass further back.

"Adaptation ratios" (Nashner 1976) demonstrated that with consecutive rotations of the support surface about the ankle joint, the apparently inappropriate muscle responses diminished in size. In patients with bilateral or with unilateral vestibular deficit, the absence, or respectively, the reduction of "adaptation" during support surface rotations (see Black et al. 1983, Fig. 5 and Nashner et al. 1982, Fig. 7) was explained as an "inappropriate reweighting of normally redundant inputs" (Black et al. 1983). To the vestibular system was thus ascribed the function of an orientational reference for the rapid identification of conflicts between other sensory systems. Support for this hypothesis rested on two findings. First, patients with bilateral or unilateral vestibular deficits lost their balance when a conflict existed between the support surface and visual input (Black et al. 1983; Nashner et al. 1982). Second, the assumption of delayed head movement during rotations at the ankle joint suggested little or late contributions of labyrinthine input toward sway stabilizing responses (Nashner 1972; Nashner et al. 1979).

Considering the vestibular system only in the role of a reference system that compares the congruence of other sensory inputs during postural destabilization is not compatible with recent evidence demonstrating stimulation and participation of labyrinthine input in the early sway stabilizing responses. Acceleration patterns of the head, and analyses of muscle EMG response size have demonstrated sizable contributions of vestibular inputs toward maintaining the magnitude of early ankle muscle responses during direct ankle joint rotations (Allum 1983; Allum and Pfaltz 1985; Bussel et al. 1980). Given these findings, and the fact that the postural system should be considered a multisegmental system rather than a simple inverted pendulum rotating about the ankle joints, alternate assumptions are needed to explain the mechanisms supporting the sensitive balance of anterior and posterior forces during upright stance. One hypothesis is that agonist and antagonist muscles acting about the ankle joint are activated in a coupled mode during stabilizing reactions (Allum and Büdingen 1979).

In order to minimize oscillations at a joint, a continuous balance of the multi-directional forces must be generated despite fluctuations in the overall response size of any one of the participating muscles. It is possible, as suggested previously (Nashner 1976), that body stabilization, after dorsi-flexion of the support surface about the ankle joints, is established by keeping the response size of a single, normally destabilizing, triceps surae muscle action at the ankle joint small. This would assume however, that the antagonist tibialis anterior muscles at that joint are not responding strongly enough to pull the body beyond the vertical position in the other direction. More likely, as suggested by studies of other joints (Bizzi et al. 1982; Fel'dman 1966; Fel'dman 1981; Gielen and Zuylen 1986), the simultaneous activation of opposing muscles pulling across the ankle joint provides the equilibrating torque. A decrease in the response size of any one muscle at the joint would then be expected in the opposing muscles as well. Simultaneous reduction in antagonist muscle responses is difficult to explain as an adaptation of the proprioceptively triggered long loop reflex gain in the agonist muscle (Nashner 1976). A better explanation would be found in the actions of higher motor centers on the neural reflex circuitry receiving the visual, vestibular and ankle proprioceptive inputs. Modulation of activation patterns at joints other than the ankle, in particular the neck, would reflect the more generalized process of central habituation rather than local adaptation of stretch reflexes at the ankle joint.

The current study was designed to investigate the interaction of muscular activity elicited by visual and vestibular inputs with the ongoing mechanical responses of the head, body and foot. To that end, the magnitude of postural reflexes in the neck muscles was correlated with head accelerations, and the leg muscle reflexes with the direction and magnitude of ankle torque exerted during restabilization. Visual contributions to the adaptation process were assessed by comparing normal responses under counterbalanced eyes open and eyes closed conditions. Vestibular contributions were identified by measuring the responses of patients with bilateral peripheral vestibular deficit and comparing them with those of normal subjects.

Methods

Body tilts were induced by rotating a force measuring platform about the ankle joints of standing subjects. Subjects stood with socks on, knees locked, and arms at the sides of the body. The feet were not strapped to the platform though lateral and backwards movements were restricted by plastic guides. Ten dorsiflexion rotations followed by 12 random rotations (dorsi- or plantar flexion) were presented at a velocity of 36 deg/s and amplitude randomized between 2.8 and 3.2 degrees.

Ten normal subjects (ages 17 to 42 years) and five patients with bilateral peripheral vestibular deficit (ages 18 to 43 years) were tested first with eyes open (EO1), and then repeated the test with eyes closed (EC2) after a 5 min rest pause. A second group of ten normal subjects was tested with eyes closed first (EC1), followed by eyes open (EO2). All subjects were given Romberg and Unterberger tests for cerebellar or vestibular dysfunction. Any prospective "normals" showing abnormal responses were not included in these experiments. Patients were selected for testing on the basis of normal computer tomography brain scans, the absence of a nystagmus response to caloric irrigation of both ears, the absence of horizontal and vertical vestibular-ocular reflexes in response to 20 deg/s^2 acceleration amplitude, triangular velocity profile of cycle interval 24 s, and no other pathological neurological symptoms. In one patient onset of the deficit occurred after influenza, one patient had bilateral section of the vestibular nerves as a result of acoustic neurinoma surgeries, and in three patients the etiology was unknown. Four of the patients had participated in an earlier experiment on a platform.

Apparatus

Platform angle was measured with a potentiometer. Head angular accelerations were measured with an angular acceleration transducer (Systron Donner model 8160 bandwidth 0.007 to 8 Hz) mounted on top of an integral motorcycle helmet fitted with an inflatable inner lining. When inflated, the inside of the helmet pressed tightly against the cheekbones and base of the skull without restricting head movements. Body angular acceleration was measured with a second angular accelerometer from the same manufacturer. This transducer was matched in frequency responses to the first, and mounted on a breastplate stabilized by a shoulder harness.

Pairs of electrodes spaced 2 cm apart were placed to record surface EMG signals from the soleus (SOL), tibialis anterior (TA), upper trapezius (TRAP), and splenius capitis (SPLEN CAP; posterior to sternocleidomastoid and anterior to trapezius) muscles on the right side of the body. The signals were bandpass filtered between 75 Hz and 2 kHz, amplified by at least a factor of 2000, full wave rectified and smoothed with a time constant of 10 ms before being sampled every ms by the analog to digital convertor of a laboratory computer. All other signals, right ankle torque, head and body accelerations, and platform angle and acceleration were sampled every 2 ms. Data 100 ms prior to, and 600 ms following the onset of the command signal for each rotation were stored directly on the computer disk for later offline averaging.

Incremental torque (with respect to a torque exerted during quiet standing) acting at the sole of each foot was measured with a strain gauge system and low pass filtered at 80 Hz. For display purposes only, the two torque signals were added together, low pass filtered at 5 Hz, and presented to the subject at eye level on an oscilloscope screen with a gain of 4 Nm/div. Subjects were instructed to stand first in a comfortable upright position with knees locked and then to lean back slightly until the TA muscle was continuously active, thereby producing a 4 to 8 Nm change in ankle torque (except for one patient who was not comfortable leaning back). The reference torque for incremental torque was then reset, and provided a guide helping the subject to realign body inclination prior to each rotation. The subjects were instructed to return quickly to the upright position on onset of a rotation. During eyes closed trials, subjects were informed through high and low puretone sounds if their incremental torque was not within 2 Nm of the reference level.

Data analysis

Offline, the 10 responses to continuous dorsiflexion rotations for the eyes open and eyes closed conditions were individually analysed for response latencies. Three responses to dorsiflexion rotation in the random condition were selected for analysis, each following at least two plantar flexion rotations. Average records were computed for the first 3 and last 7 of the 10 successive dorsiflexion trials. Acceleration and torque records were filtered with a zero phase shift, low pass, 10th order Butterworth filter whose corner frequency was set at 62.5 Hz.

Three latencies were recorded for the ankle muscles (short, medium and long). Latencies were read from single responses and referred to the time of the first inflexion in the platform velocity trace. In general (see results), short latency (SL) onset for bursts of EMG activity were taken as the first time the activity remained significantly above, or crossed the average prerotation level of EMG activity. Medium latency (ML) onset was defined as the second burst of EMG activity. Long latency (LL) onset was taken 75 ms following onset of ML activity since this was the approximate time at which a third burst occurred in TA muscles (Allum and Pfaltz 1985). Two latencies were recorded for the neck muscles (ML and LL), and represented the initial burst and 75 ms following onset of EMG activity.

The area under a burst of EMG activity was calculated by trapezoid integration for the individual trials (data for Fig. 3), for the average of the first 3 trials, and for the average of the second 7 trials (data for Tables 2 and 3). For the ankle muscles, area of the SL activity was measured from the onset of the burst to the onset of TA ML activity. Area of ML activity was defined as extending 75 ms from the onset of TA ML activity. LL activity was measured from 75 to 150 ms after ML onset. The area of ML neck muscle EMG activity was recorded 75 ms from onset of TRAP ML activity, and from 75 to 150 ms for LL activity. The average level of EMG activity 100 ms before platform rotation was used as reference level for area calculations, except for individual trials where absolute zero activity was employed as reference.

Changes in right incremental ankle torque were measured over 3 time periods commencing 65 ms after rotation onset. The 65 ms starting point was chosen as an easily identified local maximum due to the stretch response of intrinsic muscle properties as the platform decelerated (Allum and Mauritz 1984). The three intervals were 65–150, 150–225 and 225–300 ms. Each interval commenced approximately 25 ms after the onset of SL, ML and LL ankle muscle activity. A delay of 25 ms corresponds to the period required for EMG activity in a muscle to develop into a force response (Allum and Mauritz 1984; Stein and Bawa 1976). The amplitude of a distinct peak of backwards pitching head acceleration at ca 260 ms from rotation onset was measured by calculating the difference between its peak value and that of the proceeding peak forward pitching head angular acceleration (see Fig. 1).

Significant differences between the area of EMG response bursts, amplitude of head angular acceleration, and change in ankle torque were examined through a *t*-test for paired comparisons for the first three trials, second three trials and last four trials of continuous dorsiflexion rotations, and the three dorsiflexion

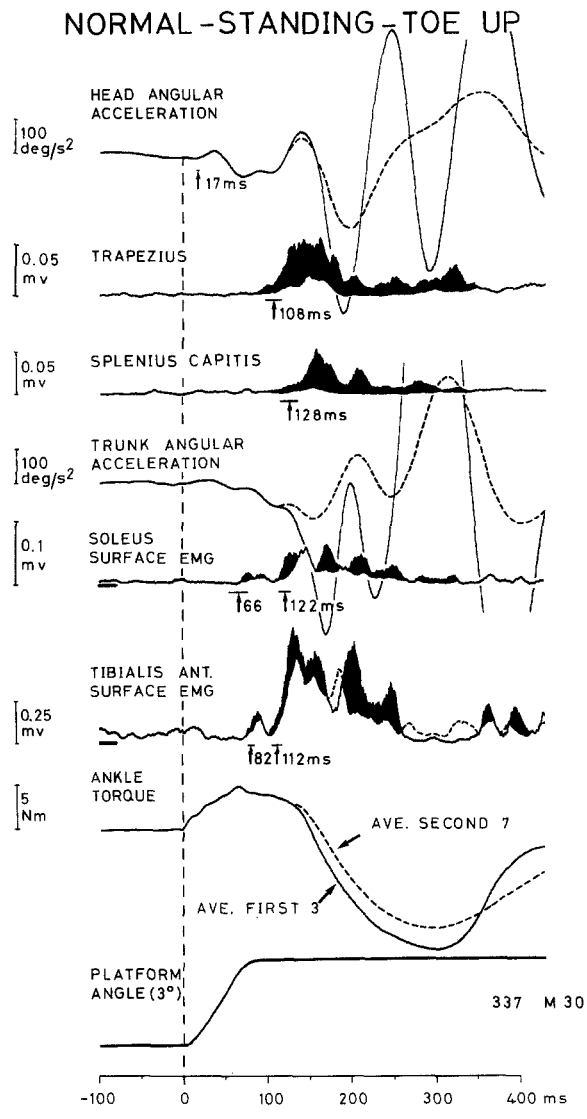
trials during random rotations. Since no significant differences were found between trials 4–6 and 7–10, a multivariate analysis of variance (MANOVA) with repeated measures was subsequently performed on the difference between the mean of the first 3 and second 7 trials of dorsiflexion rotation for the normal subjects and the patients. To determine the effects of presentation order that eyes open and eyes closed conditions might impose on muscle responses, a MANOVA was also performed between the means of the two normal groups (EO1 and EC1). This statistical analysis performs univariate F tests on each dependent measure while taking into account the correlations between the multiple dependent variables (Myers 1979). Hypotheses that were tested in both MANOVA runs included the change in response of each dependent variable over repeated presentations of the stimulus, the effect of having the eyes open or closed, and the difference between the responses of normal subjects and patients. Paired comparisons between the means of each dependent variable were then tested for significance on each experimental question at the $p < 0.05$ level with a Bonferroni t-test, using the mean squares for error calculated by the MANOVA. The Bonferroni t-test results are reported in this paper.

A stepwise linear regression analysis was applied to the individual mean values of the first three and last seven trials of SL, ML, and LL EMG activity areas. With ankle torque as the dependent variable and TA and SOL response areas as the predictor variables, the respective bivariate linear regression of torque on each and both muscles was calculated, that is:

$$\text{Torque} = (K_T * TA) - (K_S * SOL) + \text{constant}.$$

In order to represent this equation in two dimensions, the predictor constant (for example, K_S) that emerged from the bivariate regression of the muscle with the lesser correlation was then held constant during the univariate regression of torque. The torque value plus this predictor constant multiplied by the score of the more weakly correlated variable (for example, SOL) was regressed n the more strongly predictive variable (for example, TA); i.e. the dependent variable is (Torque + (K_S * SOL)), and the predictive variable is TA. ML TRAP was used as the predictor variable in a single regression analysis of head angular acceleration amplitudes at ca 260 ms. Coactivation between the muscles TA and SOL, and TA and TRAP was tested by means of a linear regression analysis on the SL, ML, and LL EMG response areas of respective muscle pairs.

Fourier analysis of 512 ms of head and body angular acceleration records commenging 25 ms after the onset of platform rotation was performed using standard FFT techniques (Bendat and Piersol 1971). From this analysis the power spectrum of head accelerations and the gain and phase of head angular accelerations with respect to body angular accelerations were determined. Gain was defined as the peak amplitude of head acceleration divided by peak amplitude of body acceleration at the two largest peaks in the power spectra.



Fi.g 1. Average responses of a 30 year old normal to dorsiflexing rotations when standing with eyes open. The averages of the first three trials (solid line) and next seven trials (broken line) are plotted, with the difference between the means of the EMG curves shown as a filled area when there is a reduction in activity. Absolute zero EMG activity is marked with a short horizontal bar at the beginning of the EMG traces except when activity prior to rotation onset was practically zero. The zero latency reference is given by a vertical dashed line, and corresponds to the first inflexion of platform velocity. Average values of EMG burst and head acceleration onset latencies are marked by a vertical arrow, 1 standard deviation by a horizontal bar above the arrow. An upward deflection of the head and trunk acceleration traces indicates backwards pitching angular acceleration. Increasing plantar flexion force on the platform is represented by an upward deflection of the ankle torque curve

Results

Stabilizing strategies in normal and vestibular deficient subjects

Typical response patterns for each muscle, ankle torque, and head and body angular acceleration are illustrated in Figs. 1 and 2 for a normal subject and a patient with a bilateral vestibular deficit. Each figure presents the averaged responses of a single subject for the first three and next seven trials of consecutive dorsiflexion rotations. The responses in Fig. 1 are from a member of the group of normals that received the rotational stimulus with eyes open first (NORMS

Table 1. Mean short (SL) and medium (ML) latencies of muscle onset (ms)

	SOL mean (sd)		TA mean (sd)		TRAP mean (sd)	SPLENCAP mean (sd)
Norms:						
	SL	ML	SL	ML	ML	ML
EO 1st	59	126	84	122	117	128
	(14)	(10)	(8)	(8)	(18)	(27)
EC 1st	55	119	87	124	113	132
	(17)	(20)	(7)	(11)	(24)	(27)
EC 2nd	54	121	86	121	111	136
	(14)	(14)	(7)	(9)	(29)	(25)
Bilats:						
EO 1st	54	122	93	129	122	125
	(12)	(17)	(7)	(10)	(29)	(34)
EC 2nd	57	128	90	130	128	122
	(12)	(23)	(10)	(10)	(30)	(39)

EO1); all subjects in the patient group were stimulated with eyes open first (BILATS EO1).

The pattern of muscle activation demonstrated in all experimental groups was consistent with previous reports (Allum 1983; Allum and Pfaltz 1985). Following the stretch to the triceps surae by the dorsiflexion rotation of the foot, a myotatic response appeared in the normal population (NORMS) in SOL at an average latency of 56 ms. This response was termed the short latency (SL) action of SOL. The inclined position of the subjects produced clearer TA responses (Allum 1983), in particular, a response in TA followed that of SOL at an average latency of 86 ms. We have termed this the SL response of TA since it was the first burst of EMG activity in TA and appeared consistently earlier than the medium latency (ML) response times. Concurrent bursts of SOL, TA and TRAP muscles appeared in the range of ML responses ca 120 ms, and were shortly followed by a burst in SPLEN CAP. Mean latencies and the standard deviations of each muscle response are presented in Table 1 for each experimental group.

After small accelerations, commencing between 18 and 22 ms, that were temporally correlated with the platform acceleration, head angular acceleration uniformly continued with a backwards pulse followed by a forward pitching of body angular acceleration. The forward pitch of the body followed the onset of SL activity in TA. A large change in ankle torque on the platform acting to pull the body forward, was initiated shortly following the onset of ML TA and SOL activity. A larger backwards pitch of head angular acceleration followed the period of the ML activity in the TRAP muscle. As will be discussed in a following section, this backwards acceleration pulse, peaking at a mean latency of 266 ms, began to decrease in amplitude with successive platform rotations (see Fig. 1), so that in some subjects it was difficult to identify in the average records of the second set of 7 trials. TRAP activity also diminished greatly at the same time.

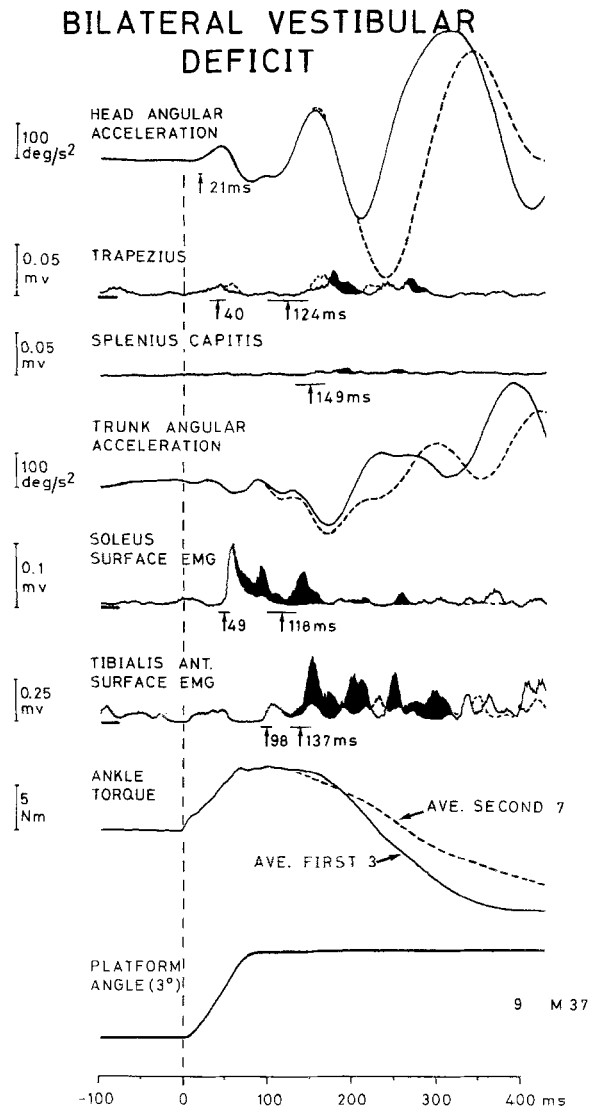


Fig. 2. Average responses of the first three and next seven trials of a 37 year old patient with a bilateral peripheral vestibular deficit which commenced 1.5 years prior to tests. The layout is identical to Fig. 1. The black filled areas of the EMG bursts record the differences between the mean responses in the two sets of trials when a response reduction occurs over trials. Note the diminished EMG activity, head and trunk accelerations, and the more shallow torque trace compared to Fig. 1

The patient data sustains the general relationships between muscle onset and the direction of torque and angular acceleration. Patients differ however in three respects that are evident in Fig. 2. First,

Table 2. Significant differences between the means of the EMG response areas, head angular acceleration, and ankle torque of normals and patients with bilateral peripheral vestibular deficit – (Bonferroni *t*-test)

Variable	Eyes open				Eyes closed				Units
	Norms mean	(sd)	Bilats mean	(sd)	Norms mean	(sd)	Bilats mean	(sd)	
TA SL EMG	1.85	(1.63)	0.96	(1.26)	2.67	(2.70)	*0.33	(1.23)	μv.s
TA ML EMG	19.56	(6.23)	*9.83	(4.41)	21.07	(7.35)	*7.17	(5.71)	μv.s
TA LL EMG	18.35	(8.67)	*8.63	(2.62)	12.50	(10.27)	*5.00	(2.96)	μv.s
SOL SL EMG	2.09	(1.92)	2.19	(0.97)	1.66	(1.57)	1.82	(1.55)	μv.s
SOL ML EMG	1.80	(1.04)	*1.11	(1.29)	1.90	(1.19)	*0.45	(0.87)	μv.s
SOL LL EMG	1.56	(1.25)	*0.43	(0.25)	0.98	(1.43)	–0.03	(0.51)	μv.s
Forward sway									
Torque SL	0.90	(2.79)	0.57	(1.18)	2.84	(2.89)	*0.90	(1.89)	Nm*100/kg body weight
Torque ML	14.00	(3.36)	*7.24	(1.75)	14.28	(3.69)	*5.96	(2.41)	ditto
Torque LL	7.57	(3.78)	6.62	(0.76)	5.16	(3.03)	4.16	(1.61)	ditto

* Significant difference between NORMS and BILATS for eyes open or eyes closed at $p < 0.05$

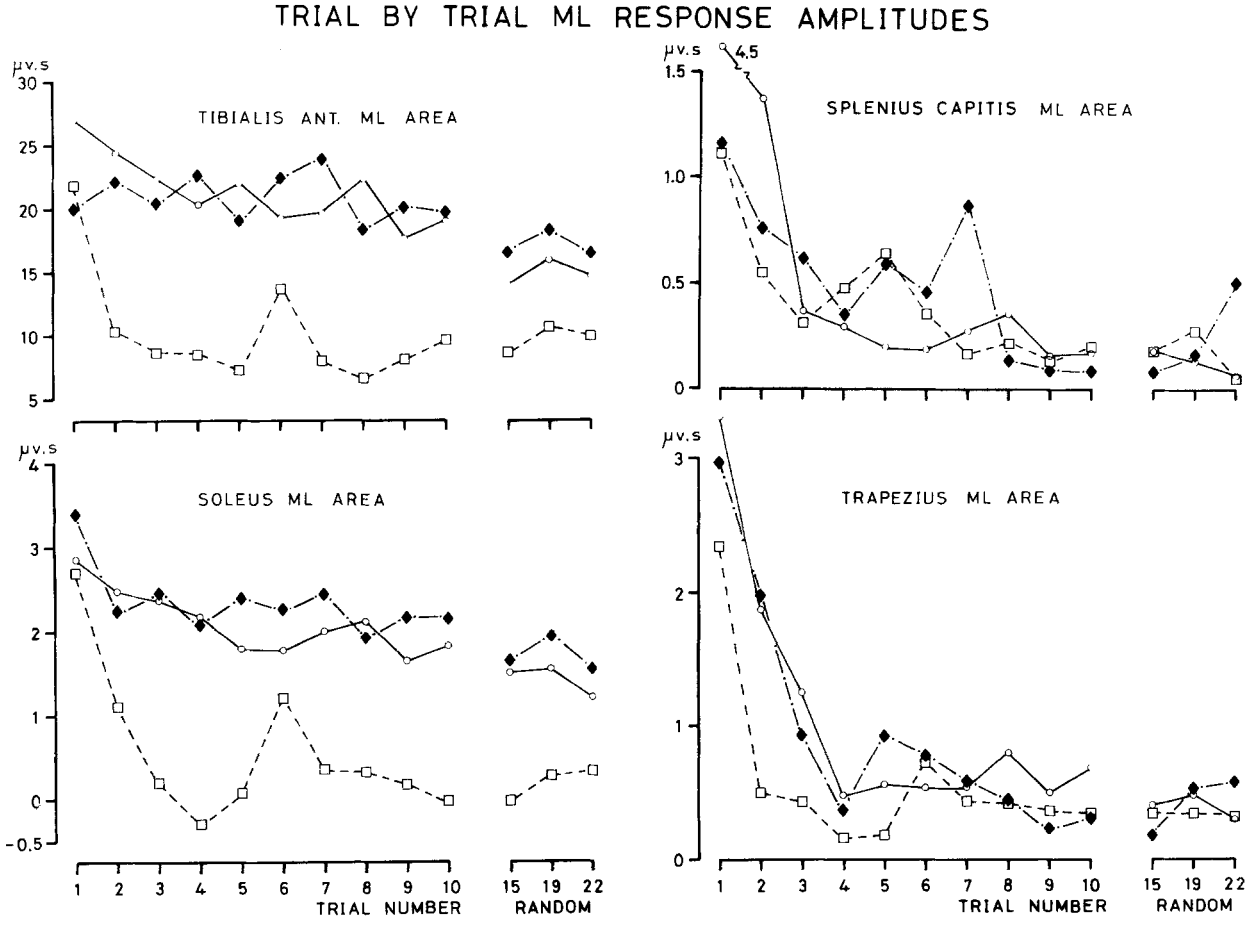


Fig. 3. Average activity under the EMG burst of medium latency TA, SOL, SPLEN CAP and TRAP for each trial of platform rotation. Data for the NORMS EO1 (open circles), EC1 (filled diamonds), and BILATS EO1 (open squares) groups is plotted. Trials one through ten were consecutive dorsiflexing rotations. The last three trials (15, 19, and 22) were dorsi-flexion rotations randomly presented among platform plantar flexing rotations. Note the early drop in the activity of all three groups, and the greater difference between the normal and patient data in the ankle muscles. Also note maintained response amplitudes during the random rotation trials. The absolute levels of the response areas (but not the differences between trials) are greater in this figure compared to those of Tables 2 and 3 since zero EMG activity was used as the reference level here (see Methods)

Table 3. Significant habituation between the first 3 and second 7 trials of the mean EMG responses areas, head angular acceleration, and ankle torque in the eyes open (EO1) condition – (Bonferroni *t*-test)

Variable	Norms				Bilats				Units
	First 3		Second 7		First 3		Second 7		
	mean	(sd)	mean	(sd)	mean	(sd)	mean	(sd)	
TA SL EMG	2.04	(2.03)	1.67	(1.26)	0.75	(1.40)	1.17	(1.14)	μv.s
TA ML EMG	21.74	(7.22)	**17.37	(5.37)	11.25	(5.08)	8.42	(3.83)	μv.s
TA LL EMG	21.44	(9.05)	**15.26	(8.48)	10.33	(2.87)	*6.92	(2.43)	μv.s
SOL SL EMG	2.24	(1.92)	1.93	(1.91)	2.86	(1.51)	**1.53	(0.43)	μv.s
SOL ML EMG	2.17	(1.18)	** 1.43	(0.90)	1.58	(1.63)	**0.65	(0.95)	μv.s
SOL LL EMG	1.86	(1.27)	** 1.25	(1.24)	0.66	(0.31)	0.20	(0.19)	μv.s
Forward sway									
Torque SL	0.57	(2.46)	1.23	(3.12)	−0.14	(1.32)	**1.23	(0.99)	Nm*100/kgbodyweight
Torque ML	14.57	(3.41)	13.43	(3.31)	8.09	(0.99)	**6.39	(2.51)	ditto
Torque LL	8.75	(4.30)	** 6.43	(3.26)	7.99	(0.43)	5.30	(1.09)	ditto
Trap ML EMG	1.61	(1.18)	** 0.53	(0.46)	1.10	(0.69)	**0.31	(0.25)	μv.s.
Trap LL EMG	1.44	(1.35)	** 0.21	(0.48)	0.35	(0.33)	0.08	(0.18)	μv.s
Splen CAP ML	1.04	(1.44)	** 0.21	(0.24)	0.63	(0.82)	*0.37	(0.53)	μv.s
Splen CAP LL	1.92	(2.79)	** 0.18	(0.26)	0.26	(0.19)	0.26	(0.41)	μv.s
Head ACCEL	194	(93)	**65	(66)	171	(61)	+107	(132)	deg/s^2

** Significant habituation for either normal subjects (Norms) or patients with bilateral peripheral vestibular deficit (Bilats) at $p < 0.05$
* $p < 0.10$
+ Significant habituation only in the eyes closed condition at $p < 0.05$

SL and ML responses of TA were somewhat delayed, on average, when compared to the normal data, as was the ML onset of the TRAP muscle (see Table 1). Consequently, acceleration and torque responses demonstrated similar delays. Second, except for the myotatic, SL response in SOL, the amplitudes for all of the ankle muscle EMG and torque traces were less than those of the normal population from the first set of trials (EO1). Aspects of both these distinctions between the normal and clinical populations have been noted previously (Allum and Pfaltz 1985). Thirdly the peak in head angular acceleration at 266 ms was delayed some 50 ms, with respect to normals.

Mean responses of the NORMS and BILATS were compared statistically to further clarify the role of the peripheral vestibular inputs on the recorded ankle muscle responses, and ankle torque. The NORMS EO1 group was compared to BILATS EO1 (eyes open, tested first), and NORMS EC2 (eyes closed, tested second) compared to BILATS EC2. As seen in Table 2, both the ML and LL responses of the TA muscle are significantly smaller in the BILATS with eyes open and closed. SL TA significantly decreased in the BILATS with eyes closed. The functional result of these differences is seen in the significantly lower ML torque exerted on the platform with eyes open and closed and the lower SL torque with eyes closed. At ML, the SOL responses of the BILATS were also significantly smaller. Except for TRAP LL, the neck EMG responses were not significantly reduced, at the $p < 0.05$ level, when compared to the NORMS. Here though, the response profile of TRAP was markedly different, including a SL response not observed in normals, and a delayed ML response (compare TRAP records in Figs. 1 and 2). The ML TRAP responses were also smaller on average in the patients but only at the $p < 0.1$ level of significance.

Response adaptation for normal and vestibularly deficient subjects

Evident in the original data of each subject presented in Figs. 1 and 2 is a decrease in the amplitude and/or oscillation frequency of each trace between the first three and next seven trials. The initial response size and subsequent final amplitude in each EMG trace differs between the normals and the patients as documented above but both show adaptation in ankle and neck muscle responses. To examine the similar adaptation more carefully, the mean area under the EMG response of each ankle and neck

muscle was calculated for each group and plotted for each trial. The areas for the three trials of dorsiflexion during random rotations were also included in this data analysis. Some general conclusions can be drawn from the data which is shown in Fig. 3. For both NORMS and BILATS there definitely is a decrease in response size from the first trial, that levels off by trial 3 or 4. Only the initial size of the muscle responses of the normal EC1 group tends to be less than that of the normal EO1 group. However, the ankle muscle responses of BILATS are much lower, the neck muscle responses marginally lower, than those of EO1 and EC1 normals. From earlier reports (Nashner 1976), when the platform direction was changed to plantar flexion, resensitization of the reflex circuits responsible for ankle muscle responses, and a return of the initial response amplitude in ankle muscles would have been expected for subsequent dorsi-flexion rotations. Our tests with randomized plantar and dorsi-flexion rotations produced continued decreases or a leveling off of dorsi-flexion responses with respect to the last 7 trials of successive dorsi-flexion (see Fig. 3).

The change in muscle response size as trials progressed was examined within each group by performing a MANOVA on the means of the first three and next seven trials. Except for SL EMG in normals, responses decreased significantly over trial presentations as indicated in Table 3 for the neck and ankle muscles of NORMS EO1 and BILATS EO1 groups. The greatest reductions in the ML and LL responses for the normals appeared during the first set of trials, be it with eyes open or closed first (EO1 or EC1). Vision though had no influence on the reductions since there was no significant difference between the reductions of the normal EO1 and EC1 groups. Eventually the ankle and neck muscle areas diminished to a constant lower level since there are virtually no significant changes across trials in the EC2 (eyes closed, tested 2nd) NORM and BILAT groups except for LL TRAP for the normals. The attenuation of the muscle responses of NORMS is first reflected biomechanically in the diminished LL response of ankle torque (225–300 ms) and the backwards pitch of head angular acceleration, peaking at 266 ms (Table 3).

Table 3 also presents significant adaptation of EMG bursts in the BILATS EO1 that is not in agreement with earlier reports of a lack of adaptation for this patient population (Black et al. 1983; Nashner et al. 1982). Due to the smaller number of subjects and larger standard deviations in the patient group, significance for multiple comparisons was not as easily obtained for the TA and SPLEN CAP muscles of this group at the 5% level. Averaged data, however, suggested that attenuation was taking place in these muscles in addition to the 5% significance changes in SOL and TRAP (examine the means between the first three and second 7 trials in Table 3). Multiple comparisons performed at an experiment-wise error rate of 10% revealed some additional significant differences in TA and SPLEN CAP. Thus, though not as highly significant as the normals, all of the muscles responses of BILATS EO1 demonstrate adaptation. The influence of diminishing ankle muscle activity also appears in the ankle torque responses of the BILATS. Unlike the NORMS however, the decreases begin at medium latencies of torque (150–225 ms), while SL foreward sway torque response amplitudes increased from the first three to last seven trials corresponding to the attenuation of SL, SOL EMG in BILATS.

As stated earlier, there were no significant differences between the normal subjects tested with eyes open or eyes closed first. Vision did, however, exert significant effects over time. LL torque in the EC2 groups was less than in the EO1 groups ($F(1, 11) = 34.36$, $p < 0.0001$), and surprisingly, SL torque was *larger* for both NORMS and BILATS with eyes closed in the second presentation of trials than in the first presentation of trials with eyes open ($F(1, 11) = 6.30$, $p < 0.03$).

Muscle coactivation patterns at the ankle joint

Two criteria were considered necessary to justify calling the overlapping action of antagonist muscles at a joint coactivation. The time between the onset of the muscle responses must be non significant, and the size of the response of agonist and antagonist must alter concurrently in a single direction. Naturally, we eliminated the possibility of crosstalk between electrodes on leg muscles by having the subjects produce isolated contractions of each muscle while sitting, and checking that the antagonist recording was silent.

Returning to Table 1, it can be seen that SOL and TA muscles satisfy the first criterion of temporal concurrence. The antagonist muscles at the ankle joint exhibited responses within one standard deviation of each other. The same muscle pair were tested for meeting the second criterion by plotting the mean area of response under the EMG curve of the two muscles with eyes open and closed for the first three and next seven trials of each subject (Figs. 4–6). Pearson product moment correlation coefficients were then derived. The areas under the ML bursts of TA and SOL that act to restabilize the body following the dorsiflexing ramp stimulus are plotted in the lower half of Fig. 4. The antagonist muscles at the

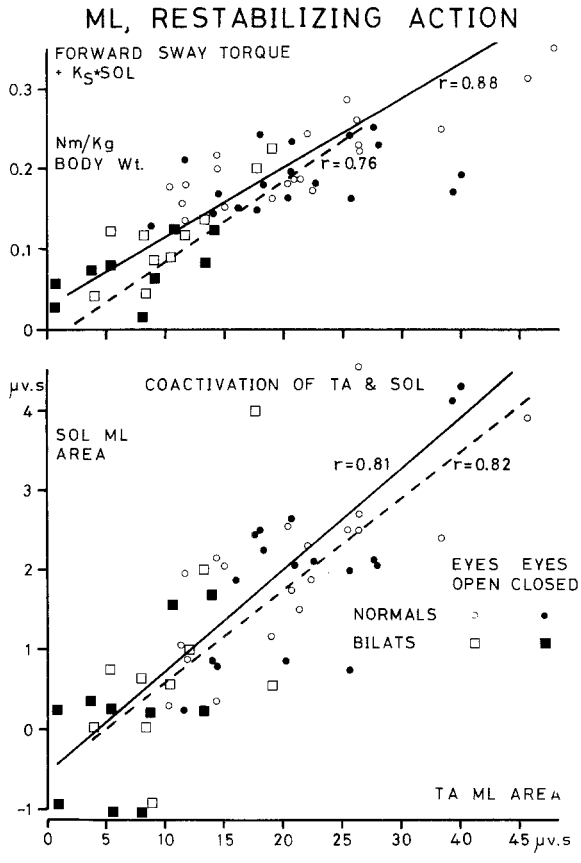


Fig. 4. Coupled activity of SOL and TA and their joint influence on ankle torque at medium latencies. *Lower half of figure:* the average areas under the ML burst of TA activity (x axis) of each subjects' 1st 3 and 2nd 7 trials of dorsiflexion rotations are plotted against the average areas under the ML EMG burst of SOL. Open and filled circles represent the data of the NORMS EO1 and EC2 groups respectively. The linear regression lines have been drawn for the combined EO1 normal and patient data (solid line) and the combined EC2 data (broken line). The respective correlation coefficient of the combined group data for EO and EC is printed next to each line. *Upper half of figure*: linear regression of the average area under the EMG burst of TA (x axis) on the average area of ankle torque after controlling for SOL ML area (the term, K_S * SOL) (y axis) for each subjects' 1st 3 and 2nd 7 trials at ML. Individual ankle torque values have been divided by the subject's body weight; then the average ML activity of SOL multiplied by its bivariate regression coefficient is added to the score (Forward Sway Torque + K_S * SOL). Data for normals and patients is again combined and the correlation coefficients are printed nect to the regression lines for the EO (solid line) and EC (broken line) conditions. Other symbols as above

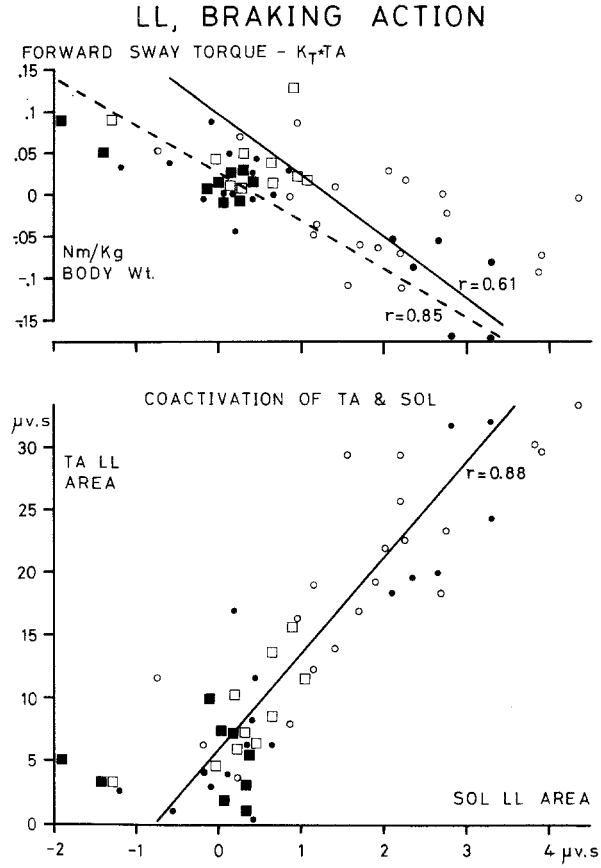


Fig. 5. Coupled activity of SOL and TA and their joint influence on ankle torque at long latencies. The layout is identical to Fig. 4 as are the symbols. *Lower half of figure:* average SOL LL area (x axis) plotted against average TA LL area (y axis). Only a single linear regression line is drawn because the correlation coefficients for EO and EC were identical. Normal and patient data have again been combined to produce the linear regression. *Upper half of figure:* linear regression of SOL (x axis) on ankle torque after controlling for TA area (the term, – K_T * TA) (y axis). As in Fig. 4, individual ankle torque values were divided by the subject's body weight. The average activity of TA is multiplied by its regression coefficient and subtracted from the values of torque (Forward Sway Torque – K_T * TA). Data for regression lines and correlation coefficients were combined for normals and patients with EO and EC

ankle joint exhibited a strongly correlated direction in the magnitude of activity at this latency for combined NORMS and BILATS data both with eyes open ($r = 0.81$, $p < 0.005$) and eyes closed ($r = 0.82$, $p < 0.005$). Muscle responses of the BILATS contribute to this correlation of coactivation, but their EMG bursts were conspicuously smaller than those of the normals (see Fig. 4). Highly correlated coactivation is also seen in the combined NORMS and BILATS LL Muscle responses at the ankle (Fig. 5, $r = 0.88$ for EO; $r = 0.85$ for EC, $p < 0.005$). In the LL period however, the range of the BILATS eyes open responses overlapped that of normals with eyes closed (see lower half of Fig. 5).

The correlation between ML TA and TRAP was also investigated to test the hypothesis that the temporal coincidence in their onsets was indicative of a single coactivation command at both joints. Neither the NORMS EO1 nor EC2 data with the respective BILATS EO1 and BILATS EC2 data demonstrated a significant coactivation correlation ($r = 0.50$ for

TRAP and TA) between the neck and ankle muscles. This finding does not support a single coactivation command for these neck and ankle muscles. Interestingly we have not been able to demonstrate a highly significant vestibulo-spinal input to neck muscles (see above) during the stabilisation task, in contrast to the strong vestibulo-spinal influence on leg muscles documented in Table 2.

Influence of coactivation and adaptation on biomechanical measurements

Interrelationships between ankle torque and ankle muscle EMG activity were more closely examined through multiple linear regressions. Actual torque output at the ankle was divided by the weight of the subject to remove variability due to subject size. When these data were plotted against the EMG activity of either muscle at the ankle, weak correlations (r = 0.1 to 0.6) between torque and muscle output appeared. However, when the coactivated action of both muscles of the ankle joint was taken into account in a multiple regression, high and significant correlations ($p < 0.0005$) appeared between ankle torque and muscle activity.

The upper part of Figs. 4–6 illustrate the bivariate regression of the two ankle muscles on torque at the ankle for the three muscle response latencies. The mean scores of each subject for the first three and next seven trials are plotted for both the NORMS and BILATS with EO1 and EC2. The plot in the lower part of Fig. 6 indicates the lack of a correlational relationship between TA and SOL EMG areas at their respective short latency values. Even though there were separate latencies of onset, individual reflex origins, and different directions of functional effectiveness (in other words that SOL was expected to pull the destabilized body further backwards), the ankle antagonist muscles jointly influenced the forward SL ankle torque (65–150 ms). SOL activity was more predictive of the resultant small, almost constant torque values (see Fig. 6 and torque over this interval in Figs. 1 and 2). The explanation for this result rests in the finding of Allum and Mauritz (1984) that the SOL myotatic reflex compensates for intrinsic muscle stiffness while linearizing the force output of the muscle. SOL SL activity must then act in conjunction with TA SL responses to regulate torque output at the ankle joint at a constant level rather than further destabilizing the body.

At medium latencies, TA exerted the greater influence on ankle torque when the activity of SOL was taken into account (see Fig. 4). The action of the TA in this circumstance was clearly to increase

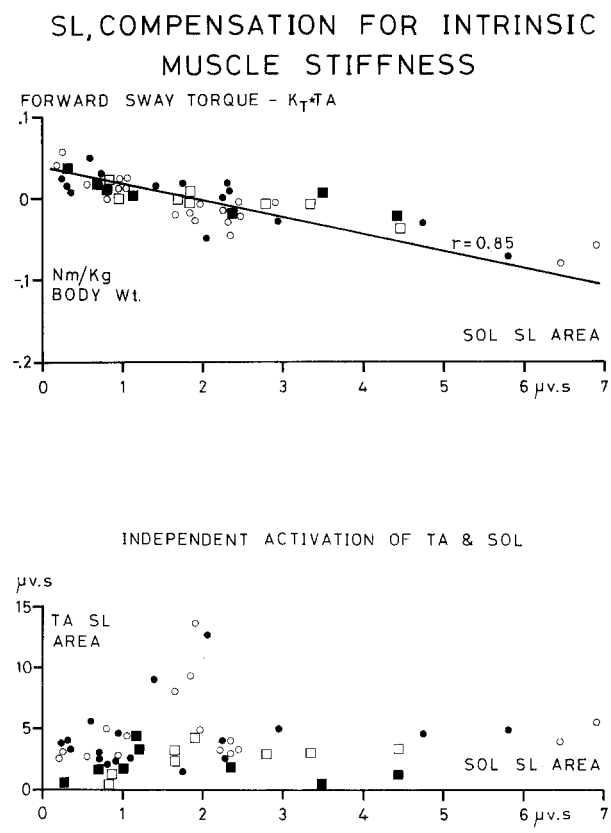


Fig. 6. Independent activation of SOL and TA and their joint influence on ankle torque at short latencies. The layout and symbols are identical to Fig. 3. *Lower half of figure:* average SOL SL area (x axis) plotted against the average SL area for TA (y axis) for each subject. A linear regression was statistically insignificant for this widely scattered data. *Upper half of figure*: linear regression of SOL (x axis) on the ankle torque after controlling for TA area (y axis). As in Fig. 5, ankle torque values are divided by body weight and then TA activity multiplied by its regression coefficient is subtracted (Torque – K_T * TA). A single regression line combining the normal and patient data in the EO condition has been drawn, and the multiple correlation coefficient printed next to it

forward sway torque about the ankle joint to counteract the rearward thrust from the platform. Longer latency torque was best predicted by LL SOL EMG when controlling for LL activity in TA. The difference between the EO1 and EC2 groups is greatest at this latency, with eyes closed producing a stronger correlation than eyes open (see Fig. 5). In this case, the more influential SOL action produced a negative sloping regression line suggesting a braking torque action on the forward sway produced by the ML activity of TA.

A linear regression data analysis was used in evaluating the influence of neck muscle activation on the backward pitch of head angular acceleration that peaked at 266 ms and led to a 7 Hz oscillation in

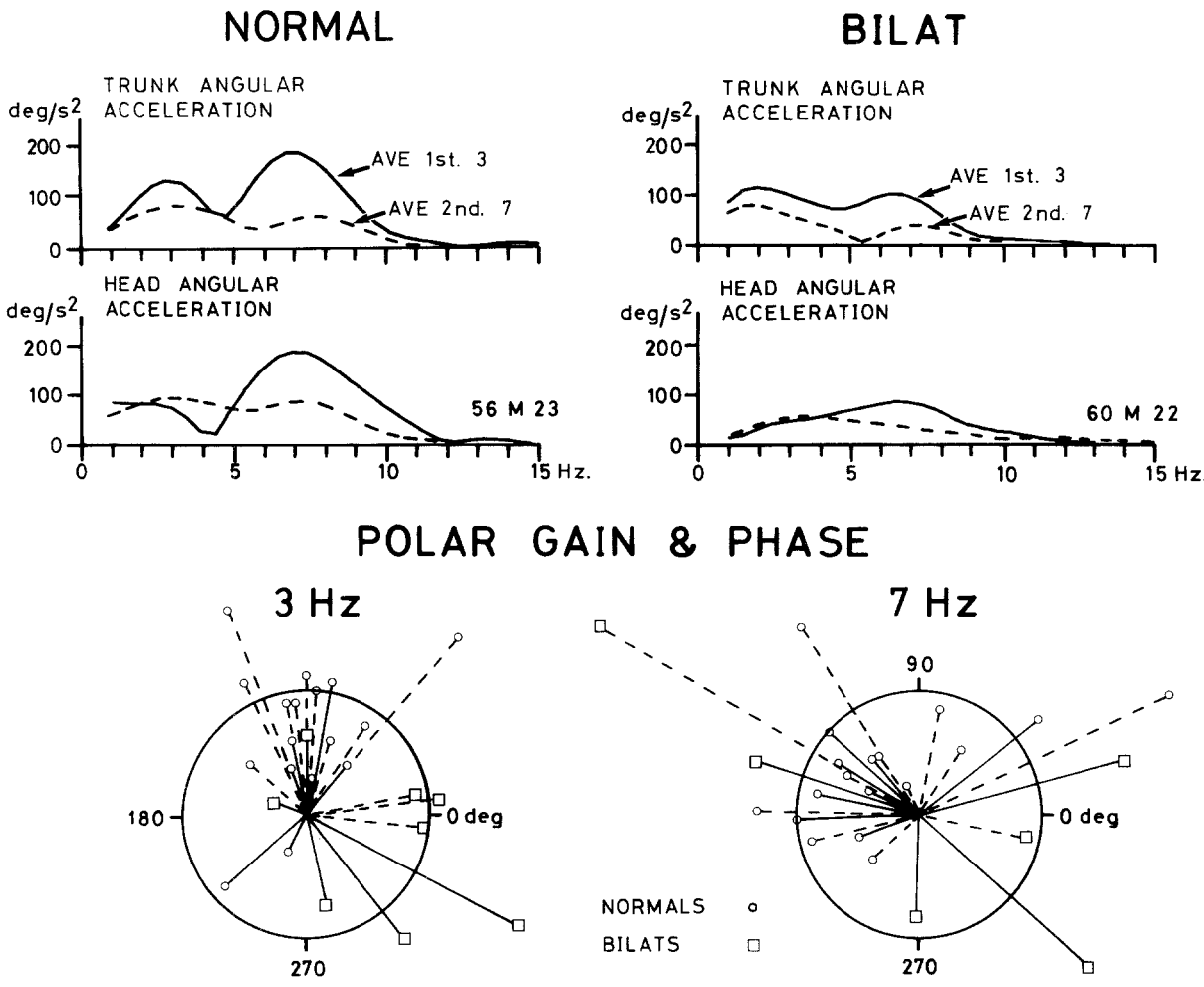


Fig. 7. Fourier power spectrums and polar plots of high and low frequency phase and gain. *Upper half of figure*: eyes open average head and body angular acceleration spectra for the first three and second seven trials of a 23 year old normal and 22 year old patient with a bilateral peripheral vestibular deficit. Note that the high frequency, ca 7 Hz, peak in the angular acceleration spectre of the patient is far less pronounced and even absent for the head in the second set of trials. *Lower half of figure:* Polar gain and phase plots of head acceleration with respect to body acceleration for the average of the first three and second seven trials of each subject at low and high frequencies of oscillation. Data are plotted for the EO1 (open circles) and BILATS EO1 (open squares) groups. These data symbols are connected to the origin by a full line for first three trials, and by a broken line for the second seven trials. Gain is represented by the distance of each symbol from the origin, phase by its placement on the 360 degree coordinate system. The circle represents unity gain. Around 90 deg phase for 3 Hz values and 180 deg phase for 7 Hz values, some of the normal data points have not been plotted to improve clarity. BILATS often displayed no 7 Hz peak in the 2nd 7 trial averages

head angular acceleration traces. Unlike the ankle torque responses, the regression was much stronger when the BILATS EO1 group was not included ($r = 0.61$, $p < 0.005$). The delayed peak of head angular acceleration (320 ms) in BILATS may have caused the poor correlation with TRAP neck muscle activity when the patient data was included in the regression. The amplitude of neck muscle activity did not, however, appear to be highly dependent on the availability of vestibular inputs (see Table 2).

Stabilization when defined by the size of head oscillations, varies considerably for BILATS (see Figs. 6 and 7, Allum and Pfaltz 1985). In order to examine the extent of the variability in head and trunk oscillations, a Fourier analysis was performed on the angular acceleration patterns of the trunk and head. Figure 7 provides examples of the normal and patient power spectra generated by the Fourier analysis of head and body angular acceleration in each group. General characteristics include the presence of a low frequency peak at ca 3 Hz, and a higher frequency peak at ca 7 Hz. Clearly reflected in the normal spectra is the decrease in amplitude, particularly marked at 7 Hz, that was observed across trials in the original acceleration traces (Fig. 1). It is of some interest that the BILATS tended not to have

the high frequency response, or to produce it at a lower mean frequency than that of the NORMS (6.1 Hz vs 7.6 Hz).

Gain and phase values of head angular acceleration with respect to body angular acceleration are illustrated for the high (ca 7 Hz) and low (ca 3 Hz) frequency oscillations of the EO1 group of normals and patients in Fig. 7. Each EO1 NORM and BILAT gain value is plotted on a circular coordinate system with phase represented by the angle, and gain the distance from the origin. Gains were close to 1.0 for 3 and 7 Hz. Accompanying the decrease in acceleration amplitudes is an increase in gain for the normals (0.74 to 1.05) and the BILATS (0.92 to 1.08) at the low frequency of oscillation. Evident at 3 Hz for the EO1 normal group is a 90 degree phase relationship between peak angular acceleration of the head and body. The BILATS tended to demonstrate a 0 degree phase in their later trials. At high frequencies of oscillation, normal phase relationships between head and body shifted toward a pattern that was 180 degrees out of phase. The majority of the BILATS maintained their phase between 0 and –90 degrees. The patient population thus seems to engage a system whereby the head is driven more directly with the body while the NORMS exhibit a counterbalancing action of the head and body pitch rotations in the sagittal plane. This finding correlates with the well known clinical observation that this patient group holds the head stiff in relation to the body.

Discussion

The purpose of this study was to determine the functional effect of two processes hypothesized as underlying the organization of sway stabilizing reactions in normals and patients with bilateral peripheral vestibular deficit. These processes, response adaptation (Nashner 1976) and the coupling of opposing muscle forces at the ankle joint (Allum and Büdingen 1979), appear to be of central rather than vestibular or visual origin since both processes were present in the vestibularly deficient patients examined here and in normals tested first with eyes closed rather than with eyes open. The examination of the influence of functionally absent vestibular inputs on sway stabilization in this study further clarified the role of these inputs during sway stabilization. Our evidence suggests that although both the patients' neck and ankle muscles respond as a result of induced destabilization, it is the ankle muscle responses that are significantly weaker as a result of the vestibular deficit and therefore are not as effective in producing the necessary forward torque about the ankle joint that would restabilize the body.

The results of this present study provide evidence to support the hypothesis of vestibulogenic coactivation of sway stabilizing responses (Allum 1983; Allum and Pfaltz 1985). Observations on the interplay of the opposing muscle forces at the ankle joint, and their effect on ankle torque, permit several conclusions about the sway stabilizing process. This study has statistically supported and extended the results of a prior investigation by Allum and Pfaltz (1985) indicating that the absence (as tested in standard clinical tests) of peripheral vestibular system signals will produce a much reduced stabilizing response in the ankle muscles followed by a reduced ankle torque. The markedly reduced restabilizing function of the ML TA and coactivated SOL muscle responses resulted in an increasing tendency of the patients to fall backwards as eyes open trials progressed and by the necessity for the examiner to be ready to prevent a fall during eyes closed trials. The early excitation of semicircular canal afferents to pitch angular accelerations, ca 20 ms following onset of platform rotations, would supply the necessary vestibulospinal signals for the sway stabilizing responses in the ankle (Allum and Pfaltz 1985). The EMG and ankle torque responses of the vestibular deficit patients recorded in this study were reduced to, on average, 30% of normal at medium latencies with the eyes closed (Table 2). The onset times for these EMG responses in the leg muscles were marginally delayed some 6 ms (Table 1). While this evidence is necessary to establish a vestibulogenic origin for the complete ML response it is not sufficient evidence. Sufficient evidence would include an absence of a response and/or a fundamental change in its latency when the vestibular system is deficient. On the basis of our current data we cannot exclude the possibility that the ML response is triggered or gated by proprioceptive afferent signals elicited by muscle stretch (in the lower leg or neck muscles) and then the response amplitude is considerably modulated by vestibulospinal signals. It is also possible that the remaining 30% of the response is proprioceptively generated. Clearly, further experiments are required to determine the afferent origin of the non-vestibular component of the ML response.

An overlapping ML onset of the opposing ankle muscle responses has been recorded in this study and significance testing has revealed no differences between their latencies. The fact that this overlap takes place within a single standard deviation implies a coincident activation. Studies on voluntary limb movements (Fel'dman 1966; Gielen and Zuylen

1986), oculomotor control (Fel'dman 1981) and head-eye positioning tasks (Bizzi et al. 1982) have claimed that the CNS specifies postures as changing ratios between the levels of activation in the muscles around a joint. Coactivation commands will simultaneously shift the activation levels in agonists and antagonists. Coactivation produces a functional stability by decreasing oscillation and overshoot through the control of the total stiffness of opposing muscle forces (Fel'dman 1966; Fel'dman 1981). The strong positive correlations between the ML and LL responses of the antagonist muscles at the ankle in this task supports the coupling hypothesis of antagonists for the same functional goal; a hypothesis strongly supported by the bivariate linear regressions indicating the influential role of the opposing muscles on ankle torque (Figs. 4 and 5).

Surprisingly, although the major, ML, stabilizing responses occur at approximately the same latency, neck and ankle muscles appear not to be coactivated in the same way that opposing ankle muscles are coactivated. A separate pathway for control of the head and neck during pitch sway stabilization must be considered. The afferent origin of the neck muscle responses noted during postural restabilization (Allum and Pfaltz 1985; Woollacott and Keshner 1984) has not been clearly identified. The role of the vestibulocollic and cervicocollic reflexes in head stabilization has up to now only been investigated during rotational stimulation. These studies have indicated that head stabilization in the cat was dominated by short latency vestibular and neck reflexes (Peterson et al. 1985). Humans however tend to rely more heavily on longer latency, voluntary motor commands and the passive mechanics of the head-neck system (Kasai and Zee 1978; Viviani and Berthoz 1975; Wereley et al. 1983). The delay in the neck muscle responses required to occur coincidently with the medium latency TA response is suggestive then of some central coordination of muscle activation at both body segments.

The central mechanism that has an overriding influence on both the muscle and mechanical forces is adaptation. Previous studies had demonstrated that the size of the EMG response in a stretched gastrocnemius muscle would diminish over dorsi-flexion trials; the authors of these studies argued firstly that the action of this muscle inappropriately caused destabilization, and secondly that long-loop (to supra-spinal systems and back to the stretched muscle) proprioceptively triggered (by stretch to soleus and gastrocnemius) responses ascended the body in a distal to proximal latency order (Nashner 1976; Nashner et al. 1979; Nashner et al. 1982). It is difficult to reconcile these conclusions with our experimental data. If the opposing muscles at the ankle joint are coactivated, then the output of each is a necessary contribution to the final stabilization of sway. Thus, a reduction in the response of any one muscle at the ankle joint would require reduction in the opposing muscle forces, or a destabilization would take place in the opposite direction. That is, the subjects would fall too far forward if only triceps surae muscles adapted.

The arguments of Nashner and coworkers are clearly weakened by the lack of directional specificity in the present findings. Response attenuation appeared not only in the stretched SOL muscle, but also in the released antagonist muscle TA. Furthermore the muscle that influenced torque most depended on the response segment. For example, the bivariate regression analysis demonstrated that the greatest influence of SOL on ankle torque takes place at short and long latencies, and not at the medium latencies previously predicted (Nashner 1976; Nashner et al. 1979). It is the TA that acts as a prime mover in order to impose a forward stabilizing pitch on the body at medium latencies. Body angular acceleration traces in Figs. 1 and 2 support this result with an initial pulse that was consistently manifested in an anterior direction. The earlier influence of SOL on ankle torque, at latencies indicative of a myotatic reflex, causes, as shown by Allum and Mauritz (1984), a linearizing influence of the reflex on the intrinsic visco-elastic forces about the ankle joint. The late, LL, greater influence of SOL presumably brakes the body in the new upright position. Continuation of the attenuation in the trials with random rotations of the platform for all subjects questions the notion that changes in long loop stretch reflexes underlie adaptation. On the basis of Nashner's early work (1976) one would have expected a resensitization of these reflex gains after several trials with plantar flexion rotations. We were not able to observe this resensitization (see Fig. 3).

Consistency of the functional relationship between ankle muscles and ankle torque throughout the experimental period indicates that these muscles are prime movers during sway stabilization. Thus requesting the subjects to lean back slightly, in order to increase TA background EMG activity and thereby more effectively monitor TA reflex responses (Allum 1983), appears not to shift the position of the primary sway stabilization axis away from the ankle to the hip joints. Recent results (Horak and Nashner 1986) indicate that ankle muscle responses for the "hip strategy" are considerably reduced in comparison to those of the "ankle strategy". In our tests, however, as the subjects lean back, shifting the centre of foot pressure towards the

heels, the "ankle strategy" is presumably still used, since TA and SOL responses become larger (Allum 1983).

Another argument against adaptation being due to a change in the gain of ascending ankle stretch reflexes is that it is not a local effect restricted to leg muscles but also occurs in neck muscles. As the latencies of the neck muscle responses are similar to those of leg muscles, it is rather difficult to reconcile these muscle onsets with an ascending command (see Woollacott and Keshner 1984, for complimentary evidence from responses to linear anterior-posterior support surface movements).

The "adaptation ratio" calculated in our experiments (average of the second 7 divided by average of the first 3 responses) equalled 0.66 in normals and 0.41 in the patients for the ML SOL area. This measurement was influenced most by adaptation. The values for the normals are higher, i.e. showed less adaptation, than those recorded by Nashner and coworkers (Black et al. 1983; Nashner et al. 1982). Thus the question arises whether differences in stimulus conditions between the two sets of experiments could account for discrepancies between the results of the normals. (A previous report of a lack of adaptation (Diener et al. 1984b) has been clarified. Adaptation was not examined by Diener and coworkers since the first 3 responses were not recorded, merely used to acustom the subjects to the stimulus (Diener 1986, personal communication)). For his 1976 article Nashner employed a stimulus velocity of 6 deg/s, 1.8 deg in amplitude, and only 50% of the normal subjects showed adaptation in the triceps surae muscle response starting at 120 ms (Nashner 1976). Later work (Black et al. 1983; Nashner et al. 1982) used a stimulus velocity of 20 deg/s, amplitude 5 deg, for which responses started at 90 to 110 ms. Although it is not stated directly, their articles imply that all their normal subjects showed adaptation. The inference being, the faster the stimulus velocity the clearer the responses and the effect of adaptation. Our stimuli were slightly faster, 36 deg/s, but smaller in amplitude, 3 deg, than in more recent publications of the Nashner group. However, the basic pattern of the response in SOL and TA is not altered when stimulus velocity is changed from 20 to 40 deg/s (Allum and Pfaltz 1985; Diener et al. 1984a). Only the response latencies and amplitudes are earlier and larger respectively as support surface rotation velocity is increased.

The same effect, a shortening of latency and larger response amplitudes also occurs as the subjects' initial position prior to the rotation is changed from upright to the stance leaning slightly backward used in our experiments (Allum 1983; Diener et al. 1983). Unfortunately the Nashner group did not control for the subjects' initial position prior to each rotation. If not controlled, subjects have a tendency to lean more forward with each successive dorsiflexion rotation which tips their centre of gravity backward. The records of Diener et al. 1983, Fig. 3, indicate that then the triceps surae response would be increased and the effect of adaptation would be masked and therefore less than for a controlled stance. In summary, our changes in stimulus conditions were designed to produce more prominent earlier responses, so that if anything the effect of adaptation should have been more evident than in the work of the Nashner group, not less as we found. Finally it is important to note that we examined EMG areas over the complete response divided into 3, 75 ms intervals, not just the 75 ms interval starting at 90–110 ms as the Nashner group did. Thus if our increase in stimulus velocity (20 to 36 deg/s) but decrease in stimulus amplitude (5 to 3 deg) had effects on the major interval of adaptation we would have captured this effect.

All patients with bilateral peripheral vestibular deficit showed an intact adaptation system acting on all muscles tested, even with eyes closed. Despite the fact that their responses were already smaller than those of the normals from the loss of vestibular inputs, the patient group continued to exhibit further attenuation as trials progressed. An intact peripheral vestibular system is, therefore, not a prerequisite for the adaptation of sway stabilizing responses.

Our results appear to exclude the notion that a decrease in the gain of proprioceptively triggered long loop reflexes from ankle muscles causes adaptation. Visual influences were also excluded as the responses of normals tested first with eyes closed adapted similarly to responses recorded in the group of normals tested eyes open first. Nor is a direct vestibular influence on adaptation compatible with our results. The possibility remains that proprioceptive reflexes from the neck underlie adaptation, a possibility which is difficult to verify, but would fit with the greater adaptation in the neck than in ankle muscles. Currently the most parsimonious explanation for adaptation is that it is a central process receiving a number of afferent inputs without being wholly dependent on any one for its function. Such a process would have some similarities with vestibulo-ocular reflex *habituation*, and would be more appropriately so termed.

Collins (1973) described vestibular habituation as a central mechanism. There is a change in the original form of the response so that it is gradually modified to oppose the inappropriate components, and to develop those that compensate for the task

(Collins 1973; Young and Henn 1974). The effect of these compensatory modifications can be monitored via the biomechanical responses to support surface rotations. Firstly, the decreasing high frequency oscillations of the head combined with increasing gains of head to body angular acceleration at low frequencies suggests a decrease in the degrees of freedom of motion at the neck. The diminished contributions of the neck muscles to the acceleration patterns indicates a greater reliance on the visco-elastic properties of the head-neck system (Viviani and Berthoz 1975). Secondly, habituation did not show up in the ankle torque of the normal subjects until the long latency response time, even though the 25 ms delay and 75 ms window should have encompassed any changes resulting from reductions in the short and medium latency muscle responses. Diminishing muscle output was combined with diminishing amplitude and oscillation of head and body angular accelerations, with the probable result of minimizing the displacement of the body. It is therefore as important for the central coactivation command to reduce the level of force output from the separate muscle-spring units, in order to save energy, as it is to produce a balance between the opposing muscle forces in order to sustain the functional ankle torque necessary for stabilization.

We propose that the postural control system is organised to attain optimal levels of torque about the ankle joint with a minimum of energy expenditure. Hence, in *normals* the process of habituation does not alter the functional relationship between the coactivated components at the ankle joint despite the continuous change in individual muscle force output. Denying a visual input has little effect on the response levels and functional torque output since vestibulo-spinal input to the coactivation command centre is large enough to generate the necessary output levels for stability. In contrast, ankle torque responses of the patients habituated in an earlier part of the torque response, at medium latencies, because the coactivated opposing muscle force outputs when habituated could no longer maintain a functional balance at this latency. The much lower torque output was evidenced in a progressively deteriorating ability to regain balance once tilted by the support surface, that appeared following the fourth or fifth eyes open trial, and during all eyes closed trials. We conclude that the practical absence or major reduction of vestibulospinal signals in the patients results in a muscle output at the ankle joint insufficient to successfully restabilize the body once the modifications of habituation occur. The instability is even more profound if visual compensation for the vestibular deficit is absent.

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