Postural adjustments associated with different unloadings of the forearm: effects of proprioceptive and cutaneous afferent deprivation

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Abstract: Postural adjustments to imposed (passive) and voluntary (active) unloading conditions of the forearm were studied in normal subjects and a deafferented patient. The latency of the postural behaviour (deactivation of the biceps supporting the weight) was linearly related to the displacement amplitude of the unloaded forearm, independent of the unloading conditions. The postural behaviour consisted of an anticipatory postural adjustment (APA) occurring prior to active unloading (in both normals and the patient) and conversely in an unloading reflex response following passive unloading (only in the normals). In both the deafferented and the normal subjects, the amplitudes of the displacement during active unloadings were much smaller (3 x) than in the passive conditions and an APA was present in both the deafferented and the normal subjects. The APA could not be triggered by some types of active movement and was absent when the movement was not directly producing the unloading. The EMG latencies of the APA and of the contralateral muscles used to unload were tightly coupled. However, the latency would sometimes be decoupled, particularly when a temporal delay was introduced between the active movement and the unloading in normal subjects. In contrast to the normal subjects, who were able to adapt quickly to an unusual unloading condition (produced by voluntary knee flexion), the deafferented patient did not show an APA in this task. It was concluded that, although the APA is of central origin, it cannot be generated only on the basis of internal timing cues and must rely on afferent information for its generation during unfamiliar unloading conditions.

Key words: posture, bimanual coordination, deafferentation, open-loop control.

Résumé: On a examiné les ajustements posturaux en réponse à des conditions de délestages volontaires (actifs) et imposés (passifs) de l’avant-bras chez des sujets normaux et chez une patiente déafférentée. Une relation linéaire a été observée entre la latence du comportement postural (désactivation du biceps supportant le poids) et l’amplitude du déplacement de l’avant-bras délesté, et ce dans toutes les conditions de délestages. Le comportement postural consistait en un ajustement postural anticipé (APA) précédant le délestage actif (tandis que les sujets normaux que chez le patient) et, inversement, en une réponse réflexe de délestage suivant le délestage passif (seulement chez les sujets normaux). En plus de montrer un APA, les deux types de sujets ont démontré des amplitudes de déplacement plus faibles durant les délestages actifs (3x) que durant les délestages passifs. Certains types de mouvements actifs n’ont pu déclencher l’APA; celui-ci ne s’est pas non plus manifesté lorsque le mouvement n’était pas directement associé au délestage. Les latences EMG de l’APA et des muscles controlatéraux utilisés pour le délestage étaient étroitement couplées. Toutefois, elles ont parfois été découpées, particulièrement lorsque, chez les sujets normaux, un délai a été introduit entre le mouvement actif et le délestage. Contrairement à ces sujets, qui pouvaient s’adapter rapidement à une condition de délestage inhabituelle (produite par une flexion volontaire du genou), le sujet déafférenté n’a pas montré d’APA dans cette tâche. On a conclu que, même

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s'il est d'origine centrale, l'APA ne peut être produit seulement sur la base de repères chronologiques internes, et que son apparition, durant des conditions de décharges inhabituelles, doit reposer sur une information afférente.

Mots clés : posture, coordination bimanuelle, déafferentation, contrôle en boucle ouverte.

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Introduction

Adequate motor coordination implies not only controlling a movement itself but also compensating for the perturbing effects that the movement may otherwise produce on posture and equilibrium. Contrary to the postural reactions that follow and occur in response to an unexpected perturbation, anticipatory postural adjustments (APA) occur prior to the onset of movement to prevent a disturbance to posture or equilibrium (review by Massion 1992). The present experiments were performed to improve our understanding of the physiological mechanisms of the APA.

The APA can be observed in the legs or the arms. For example when a subject is standing, a rapid movement of the arm that challenges equilibrium (i.e., changes the centre of gravity) will be preceded by an anticipatory change in the muscles of the back and of the legs (Belenkiy et al. 1967; Bouisset and Zattara 1981; Cordo and Nashner 1982; Friedli et al. 1984; Horak et al. 1984; Lee et al. 1987). In these reports, the onset of EMG changes in the postural muscles often preceded that of the EMG in the arm's prime mover. However, both behavioural and biomechanical conditions have been shown to affect the recruitment time of the postural muscles, so that the postural leg and focal arm muscles can be recruited at the same time (Lee et al. 1987). Similarly, APA can occur in the arm during bimanual tasks. When a subject actively uses his hand to remove a weight supported by the other hand, the unloaded limb shows very little displacement compared with passive unloading performed by the experimenter. Anticipatory deactivation of the muscles supporting the weight is part of the postural compensation preventing the undesirable movement of the unloaded limb (Hugon et al. 1982). In this case, unloading is done with the subject seated so that equilibrium is not threatened. Nevertheless, an APA is present in order to stabilize the position (i.e., posture) of the unloaded arm. A tight coupling between the latencies of activation of the muscles lifting the weight and the deactivation of the postural muscles has been observed (Hugon et al. 1982; Dufossé et al. 1985). The initiation of the postural adjustment before movement onset was taken as evidence that the APA depends on feedforward postural control, relying only on the output of a central command and not on the feedback input from peripheral afferents. The central origin of the APA has been supported by the presence of this APA in a deafferented woman deprived of proprioceptive and cutaneous afferents in both arms, the trunk, and neck (Forget and Lamarre 1990). It has also been suggested that the acquisition of this APA depends on a central command rather than on the resulting movement parameters (Paulignan et al. 1989).

In the present experiments, we studied muscle activity and forearm displacement amplitudes during different passive and active unloading conditions of the arm in normal subjects and in the deafferented woman. The goal was to study the APA when different motor commands produced the unloading.

The motor command was varied by changing the muscles used to remove a constant load. The main objectives were to study the short-term effects on postural adjustments (consistency of the response across a limited number of trials for each condition) and to compare the normal subjects’ response with that of the deafferented patient to determine whether proprioceptive and cutaneous afferents contribute to these effects.

Methods

Different unloading conditions of a 1-kg weight supported by the right forearm were studied in five normal subjects (two women and three men 24 — 33 years old) and a deafferented woman (37 years old). Informed consent was obtained from all subjects prior to beginning the experiment. A clinical description of the patient can be found elsewhere (Cooke et al. 1985; Forget and Lamarre 1987). The patient suffered a permanent and specific loss of the large sensory myelinated fibres following an episode of sensory polyneuropathy. This resulted in a loss of all tendon reflexes, light and crude touch, vibration, kinesesthesia, and position senses in her four limbs, trunk, and neck. She can feel strong deep pressure, and the sense of pain and temperature seem to be intact. The condition involved strictly her peripheral sensory nervous system, and the motor pathways were not affected. Motor nerve conduction velocities and needle EMG investigation of the muscles of the arms were normal. No sensory potential could be recorded in the hands or the feet and no cortical response could be evoked by electrical stimulation of the peripheral nerves of either arm. A sural nerve biopsy revealed a loss of the large A-β myelinated fibres (Fig. 1). The density of her fibres (2496/mm²) was roughly 40 % of what is commonly reported in normal subjects (Charron et al. 1980; Ochoa and Mair 1969). The fibres larger than 9 µm were absent and those above 6.5 µm represented only 2% of the total number of fibres (n = 1601), compared with a value of approximately 40% in normal subjects. Small myelinated A-δ fibres were still present, and their number and distribution resembled what is usually observed in normal subjects.

Experimental setup

The deafferented and the control subjects were seated on an adjustable chair with the right shoulder in a neutral position and the elbow flexed to 90°. Figure 2 illustrates the experimental setup. The unloaded right arm was the dominant side for all subjects. The right forearm was in a neutral position (0° supination—pronation), and shoulder extension was prevented by a cushion to which the upper arm was secured with Velcro straps. The 1-kg weight was suspended from a mechanical arm attached to the right wrist by a rigid articulated rod and a strap. Angular displacement of the elbow was monitored by a precision potentiometer situated in parallel and directly under the forearm to detect displacement of the mechanical arm. A handle, hooked to a
Fig. 1. Sural nerve biopsies of (A) the deafferented patient and (B, inset) a normal 33-year-old subject. Histograms of the number of myelinated fibres as a function of their diameter. The two biopsies were made in the same laboratory following the technique described by Charron et al. (1980). N, total number of fibres counted. Notice that the fibre distribution of the patient is unimodal instead of the normal bimodal one.

Fig. 2. The experimental setup to produce unloading of a 1-kg weight supported by the right forearm. Different unloadings could be accomplished by changing the pulley system. EMG monitoring of biceps brachii (B); anconeus (A); and quadriceps (Q).

Unloading conditions
Six different unloading conditions (two passive and four active) were used with each of the normal subjects and the deafferented patient. In the two passive (imposed) conditions, the experimenter produced the unloading by raising the handle attached to the triangle above the subject's wrist (condition P1) or by switching off the electromagnet (condition P2). In the four active (voluntary) conditions the subject unloaded, in response to an auditory stimulus (50 ms, 800 Hz, 35 dB), his right forearm with a movement of his contralateral limb. No emphasis was given to react as soon as possible to the auditory stimulus. The instruction was to hold the position of the right arm and remove the weight at the sound of the beep. The unloadings were produced by raising the handle, using a combination of flexion at the left elbow and shoulder, when the handle was attached to the triangle above the right wrist (condition A1); by pulling the handle downwards (as shown in Fig. 2) using an extension movement of the left elbow (condition A2); by left knee flexion with the rope attached to the left ankle (condition A3); or by switching off the electromagnet with an abduction of the left index finger (condition A4). Switching off the magnet, either actively or passively, produced a more abrupt unloading. This resulted in a higher peak acceleration of the unloaded forearm than the manual unloading. Series of 10 consecutive trials were recorded for each unloading condition. All series and unloading conditions were done in a single recording session for each subject. The experiments were performed without visual feedback for all subjects. In the deafferented subject, trials with visual feedback were also recorded in conditions P1, A1, and A2.

After the active unloading conditions, an additional experiment was performed in three normal subjects to study the effects of introducing a temporal delay, to document the strength of the coupling between the motor commands to each arm. A supplementary delay of approximately 100-150 ms between active movement and unloading was produced by making a 15-cm loop in the rope above the handle before the subject actively unloaded his forearm as in condition A1 above (using flexion of the contralateral arm). Thus the subject had to displace his arm 15 cm before the force would be used to unload the contralateral forearm. This condition differed from the previous conditions whereby the cord was tight and thus the weight was lifted at the beginning of the voluntary active movement. This additional experiment could not be performed reliably with the deafferented patient, because she could not concentrate on both arms simultaneously to precisely hold the positions. Thus the left arm would start drifting when vision was removed, thereby changing the size of the loop. In the previous unloading
Fig. 3. Elbow angular displacements (±1 SD) during the passive and active unloading conditions in the normal subjects (●) and the patient (○). ***p < 0.001; **p < 0.01. FLEX. L.E., flexion of left elbow; EXT. L.E., extension of left elbow; FLEX. L.K., flexion of left knee.

conditions, the tightness exerted on the cord prevented the left arm from drifting for at least a few seconds.

Data acquisition and analysis
EMG activity was recorded (bandpass filtered, 10-1000 Hz) with Ag-AgCl surface electrodes at the biceps brachii and anconeus muscles on both sides starting 500 ms before and ending 1500 ms after the unloading. The left quadriceps and first dorsal interosseous (FDI) muscles were also recorded during unloading conditions with the leg (A3) and electromagnet (A4), respectively. The raw EMG activity was rectified and integrated by a circuit with a voltage reset threshold where each reset gave a pulse. The frequency of the pulses was thus proportional to the energy of the signal and an envelope was derived from the frequency histogram. The pulse intervals generated by the EMG activity were measured with a resolution of 1 ms and the angular displacement signal was digitized at 200 Hz. The beginning of the angular displacement of the right elbow corresponded to the moment of unloading (less than 10 ms between these events). The latency between the change in EMG activity and the beginning of the displacement was studied for each muscle recorded.

Results
The amplitude of the elbow rotation (right forearm displacement) following the six different unloading conditions is presented in Fig. 3 for the normal subjects and patient. Typical raw EMG activity of the right and left biceps and anconeus muscles are also shown for a normal subject (Fig. 4) and the patient (Fig. 5). The EMG latencies for the left voluntary muscles producing the unloading and the right arm postural muscles are illustrated for the six conditions in Fig. 6. Notice that for the patient, and in contrast to the normal subjects, there is no value reported for biceps inhibition on the right unloaded side in some conditions. This was because there were either no unloading reflexes (P1, P2, A4) or no APA (A3). Table 1 summarizes the average displacement values, EMG latencies (±1 SD), and direction of EMG changes (excitation or inhibition) obtained on both sides for all experimental conditions in the normal subjects and the patient. In the deafferented patient, trials with visual feedback (P1, A1, and A2) gave similar results as the ones without vision. The following sections will deal only with the trials executed without vision in both the deafferented and normal subjects.

Passive unloading
When the experimenter lifted the weight, all normal subjects showed a large amplitude (16° ± 2°) elbow flexion. An unloading reflex, characterized by an inhibition of the right biceps activity at 39 ± 18 ms, followed the removal of the weight (Fig. 4, P1). A burst of EMG activity was also observed at 72 ± 23 ms in the stretched anconeus. In the deafferented patient, most postural holdings were accomplished by increased activity in the antagonist muscles (cocontraction), which was not present in the normal subjects (compare Figs. 5 and 4, before unloading). Unloadings in the patient produced displacements with amplitudes (16° ± 4°) similar to the ones observed in the normal subjects, but without an unloading reflex in the biceps nor a stretch reflex in the
Fig. 5. EMG activity and elbow angular displacement during different unloadings of the right forearm in the deafferented patient. Each series of traces represents muscle activity, elbow displacement, and the same unloading conditions as in Fig. 4. Biceps inhibition is absent in the leg drop condition (D). Abbreviations as in Fig. 4.

anconeus (Fig. 5, P1). Increased EMG activity was often observed at longer latencies in the biceps (236 ± 58 ms) and in the anconeus (171 ± 29 ms). This appeared to be a voluntary response to the unloading since the patient mentioned she was aware of unloadings through "a feeling in her head." We surmise that the unloading produced a perturbation that could be detected by her intact vestibular system. When the experimenter produced the unloading by deactivating the electromagnet (P2), the averaged displacement amplitudes for normal subjects (26° ± 8°) were significantly ($p < 0.01$) larger than in the preceding passive condition. The peak acceleration produced in condition P2 (4692°/s²) was also significantly greater ($p < 0.001$) than in condition P1 (2734°/s²). Furthermore, the ratio of the acceleration (4692/2734 = 1.72) between these two passive conditions was closer to their corresponding amplitude ratio (26/16 = 1.63). Thus, the increased acceleration was accompanied by a comparable increase in the displacement amplitude. The EMG activity was also similar to that observed in condition P1, although the latency of the unloading reflex (53 ± 14 ms) had a tendency to increase in the normal subjects. The deafferented patient showed a similar displacement amplitude (23° ± 9°) (see Fig. 3). While there was still no unloading reflex, there was increased activity in both the right elbow flexor and extensor at approximately 200 ms (Table 1) following the removal of the weight.

Active unloading

Using the contralateral arm

Contrary to passive unloading, the active lifting of the weight (conditions A1 and A2) was always preceded by a change in the EMG activity of the right postural muscles, which resulted in a small amplitude displacement (5° to 7°) of the unloaded forearm. There were no differences in the displacement amplitude obtained when unloading was done by contralateral elbow flexion (A1) or extension (A2), nor between the values of the normal subjects and the patient (Fig. 3). Voluntary activation of the left agonist muscles used for unloading started 30—150 ms prior to the beginning of right elbow displacement (means of 73 ± 18 and 88 ± 29 ms for conditions A1 and A2, respectively, in the normal subjects). At approximately the same time, biceps activity was inhibited in the unloaded arm. On average, this inhibition of the right postural muscle was produced less than 10 ms after the activation of the contralateral muscles used to unload. However, the postural inhibition would sometimes precede the contralateral activation by as much as 30—40 ms (as illustrated in Fig. 4, A2). In most trials the right anconeus was also activated 20 ms after biceps inhibition. While this early activation of the elbow extensor on the postural side was present in the patient
prior to unloading, it did not show a distinct burst as observed in the normal subjects (as seen by comparing Figs. 4 and 5).

Using the contralateral leg

In the unloading condition $A_1$, produced by flexing the contralateral knee, the results were different in the normal subjects and in the patient. In the normal subjects, the average displacement was small ($6^\circ \pm 5^\circ$) and similar to the two preceding conditions using the contralateral arm, although more variable. In the normal subjects, left knee flexion was produced by an inhibition of the quadriceps muscle, which occurred much earlier ($166 \pm 34$ ms) than the postural adjustment and also significantly sooner ($p < 0.001$) than the activation of the left arm muscles used in the other unloading conditions (Fig. 6). In the unloaded arm, the postural adjustment (i.e., biceps inhibition) occurred at an average latency ($63 \pm 64$ ms) similar to the latency observed during unloading with the contralateral arm; however, it was more variable, occurring too late in the first few trials to suppress a large amplitude movement. In the patient, there was no APA. The amplitude of the displacements was large ($17^\circ \pm 5^\circ$), similar to the passive unloading, with no change in the right biceps activity prior to unloading.

Using the switch

When the subjects deactivated the electromagnet by actively closing the switch with a abduction movement of their left index finger (condition $A_2$), the amplitudes of the forearm displacement were large for the normal subjects ($20^\circ \pm 6^\circ$) and the deafferented patient ($27^\circ \pm 5^\circ$). Although this represents a significant difference ($p < 0.01$) between the patient and the normal subjects, these values were not significantly different than the ones obtained for each subject during passive unloading ($P_2$), when the experimenter deactivated the magnet. These large displacements were not accompanied by an APA despite a voluntary activation of the FDI about 110 ms prior to the unloading in the active condition. The behaviour and latency of the biceps activity on the postural side were similar to those seen when unloading occurred as a result of the experimenter deactivating the magnet.

Introducing a temporal delay

The introduction of a loop in the rope resulted in an additional 147-ms delay, between the activation of the left biceps used to lift the weight and the onset of unloading ($220 \pm 24$ ms), compared with the latency of the analogous condition ($A_1$) without a loop ($73 \pm 18$ ms). All three normal

Table 1. Angular displacements and EMG latencies during different unloadings of the right forearm.

<table>
<thead>
<tr>
<th></th>
<th>Angular displacement, right elbow (°)</th>
<th>EMG latency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left unloading muscles (ms)</td>
<td>Right unloaded biceps (ms)</td>
</tr>
<tr>
<td>Passive (by experimenter)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$P_1$, direct lift</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>$16 \pm 2$</td>
<td>$+39 \pm 18$</td>
</tr>
<tr>
<td>Deafferented, no vision</td>
<td>$16 \pm 4$</td>
<td>$+236 \pm 58$ E</td>
</tr>
<tr>
<td>Deafferented, vision</td>
<td>$21 \pm 2$</td>
<td>$+240 \pm 68$ E</td>
</tr>
<tr>
<td>$P_2$, switch</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>$26 \pm 8$</td>
<td>$+53 \pm 14$</td>
</tr>
<tr>
<td>Deafferented, no vision</td>
<td>$23 \pm 9$</td>
<td>$+208 \pm 25$ E</td>
</tr>
<tr>
<td>Active (by subject)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$A_1$, flexion left elbow</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>$5 \pm 2$</td>
<td>$-73 \pm 18$ E</td>
</tr>
<tr>
<td>Deafferented, no vision</td>
<td>$5 \pm 3$</td>
<td>$-72 \pm 18$ E</td>
</tr>
<tr>
<td>Deafferented, vision</td>
<td>$6 \pm 4$</td>
<td>$-75 \pm 12$ E</td>
</tr>
<tr>
<td>$A_2$, extension left elbow</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>$6 \pm 2$</td>
<td>$-220 \pm 24$ E</td>
</tr>
<tr>
<td>Deafferented, no vision</td>
<td>$5 \pm 3$</td>
<td>$-88 \pm 29$ E</td>
</tr>
<tr>
<td>Deafferented, vision</td>
<td>$7 \pm 6$</td>
<td>$-81 \pm 15$ E</td>
</tr>
<tr>
<td>$A_3$, left knee flexion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>$4 \pm 5$</td>
<td>$-90 \pm 16$ E</td>
</tr>
<tr>
<td>Deafferented, no vision</td>
<td>$17 \pm 5$</td>
<td>$-166 \pm 34$ I</td>
</tr>
<tr>
<td>$A_4$, switch (abduct index)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>$20 \pm 6$</td>
<td>$-116 \pm 75$ E</td>
</tr>
<tr>
<td>Deafferented, no vision</td>
<td>$27 \pm 5$</td>
<td>$-106 \pm 52$ E</td>
</tr>
</tbody>
</table>

Note: E, excitation; I, inhibition.
Fig. 7. EMG activity and elbow angular displacement when a delay is introduced between the activation of the muscle used to unload (left biceps) and the right unloaded forearm in a normal subject. A rigid coupling between the EMG changes on both sides resulted in a premature right biceps inhibition, which produces an elbow extension (downward displacement) before unloading. After the load was removed, the elbow started flexing and a second inhibition, consisting in an unloading reflex, was observed 25 ms later in the right biceps.

**ACTIVE FLEXION WITH DELAY**

**LEFT BICEPS**

**RIGHT BICEPS**

**RIGHT ELBOW DISPLACEMENT**

In the first four trials of unloading by flexing the contralateral leg (A3, +), biceps inhibition occurred slightly after or slightly before the displacement. This resulted in relatively large flexion amplitudes of about 10°. In the trials that followed, biceps latency and displacement were similar to conditions A1 and A2. Finally, in the condition where a supplementary delay was introduced (A4), biceps inhibition was premature in 7 of 10 trials, which resulted in a small (1°-3°) elbow extension.

**Discussion**

**EMG activity and forearm displacement during passive unloading**

Passive unloading by the experimenter produced a large amplitude flexion accompanied by a rapid shortening of the muscles supporting the weight and a stretch of their antagonists. In the normal subjects, the concomitant decrease in the right biceps EMG (approximately 40 ms following the unloading) corresponds to the unloading reflex reported by Hansen and Hoffmann (1922) and studied by others (reviewed by Struppler et al. 1973). This decrease in activity is likely to be caused by a decrease or a pause in the shortening of the muscle spindles (Angel et al. 1965). Conversely, flexion of the elbow produced a long latency stretch reflex (72 ± 23 ms) in the anconeus. The relatively long latency could be due to a less rapid stretch, and thus larger latencies than those usually reported in studies imposing stretch with a torque motor. The absence of a short latency response is also common in such stretching conditions compared with the ones produced by a tendon tap.
In the two passive unloading conditions where the experimenters removed the weight either directly (P1) or via the electromagnet (P2), the amplitude of the forearm displacement was larger in the latter. This difference could be explained by the more abrupt unloading that occurs with the deactivation of the magnet, which then produces a larger acceleration of the forearm and thus a larger displacement amplitude. Similarly, Lum et al. (1992) reported an increase in the unloading rate (change in force over time) resulted in an increased acceleration and displacement amplitude.

It is possible that the reflex response contributed to the attenuation of the arm displacement lasting approximately 250 ms. It has been reported that an increase in the gain of the unloading reflex, after repetitive unloading, was accompanied by a reduced mechanical effect of perturbation (Paulignan et al. 1989). On the other hand, the deafferented patient showed displacement amplitudes similar to that of the normal subjects in the absence of reflex responses during passive unloading. This could be interpreted in two ways. Either the contribution of the reflex responses in minimizing the displacement was normally relatively small or the patient used other compensatory mechanisms. Two motor strategies could contribute to limit the amplitude of her arm displacement. First, she may have used a cocontraction strategy to increase joint stiffness. Cocontraction is a common feature of deafferented patients (Rothwell et al. 1982; Sanes et al. 1985; Forget and Lamarre 1987) and it was observed under the present experimental conditions. Joint stiffness has been reported to be low in this deafferented subject despite the use of a cocontraction strategy (Levin et al. 1995). This suggests that a small increase in joint stiffness might not be the only factor limiting the displacement. However, the contribution of the unloading reflex in normals and the increased stiffness in the deafferented subject are difficult to evaluate without an extensive biomechanical analysis. Secondly, she may have used a voluntary reaction strategy. The patient reportedly felt the perturbation (presumably through vestibular input) and responded with increased EMG activity observed in the elbow extensors and flexors, at an average latency of 175-200 ms, which is compatible with voluntary reaction time. If we add 30-40 ms for the excitation-contraction coupling, then the torque created by this voluntary activity could contribute to the deceleration before the end of the 250-ms displacement. Although the time of passive unloading was unpredictable, this was not the case for the magnitude and direction of the unloading. This predictability could have facilitated the use of these two strategies (cocontraction and a rapid reaction time) to dampen the effects of the perturbation.

When comparing passive and active unloading conditions, the results indicate that reflex activity alone certainly was not as efficient as an APA in reducing the perturbation. Paulignan et al. (1989) showed that an unloading reflex with a high gain could, at most, decrease the amplitude of a perturbation by approximately 25-30%, compared with the same unloading condition when no reflex was present. This value is considerably less (3x) than the average amplitude decrease produced by an APA associated with an active unloading. Indeed, in the present experiment and in other similar ones (Paulignan et al. 1989; Dufossé et al. 1985; Hugon et al. 1982), a further decrease, about 70-75%, was found with an active and direct unloading where an APA was produced, compared with passive unloading accompanied by reflex activity only.

**Anticipated postural adjustment with active unloading**

The presence of a normal APA in the deafferented subject supports the hypothesis that the motor command that drives this adjustment is of central origin and does not necessitate peripheral feedback (Forget and Lamarre 1990). This was also confirmed recently in another deafferented subject (patient AN in Cole and Sedgwick 1992), who had the opportunity to test with the unloading task using contralateral arm flexion (unpublished observations by N. Teasdale and R. Forget). The characteristics of the postural adjustment, its relationship with the displacement amplitude, and its behaviour during the different unloading conditions will be examined closely before discussing further the origin of the APA.

**Importance of the postural inhibition latency**

An early inhibition of the postural muscle supporting the weight and activation of its antagonist (150 ms prior to the beginning of the unloading perturbation) is associated with the reduced displacement observed during active unloading. The latency of this inhibition and its timing relationship with the contralateral muscles are key factors in understanding the size of the perturbation produced and how the nervous system planned to minimize it. The strong linear relationship (r = 0.92) between the latency of biceps inhibition and the amplitude of the displacement, independent of the unloading conditions, serves as strong evidence that the latency of the APA is a major factor in determining the size of the perturbation. This timing factor alone explains about 85% of the variance found in the forearm displacement amplitude. Other possible factors influencing the size of the perturbation are the intensity and duration of the activity of the muscles that produce the APA, since an increased unloading rate (from removing the weight by deactivating the electromagnet) resulted in an increased perturbation (also Lum et al. 1992).

**Coupling of bilateral activity**

The relationship found between the latency of biceps inhibition and the elbow displacement also revealed that a perfect compensation (0° displacement) would necessitate an inhibition occurring approximately 150 ms prior to the unloading (see Fig. 8). In our experiments biceps inhibition was tightly coupled with the latency of the contralateral muscle activity used to unload. It occurred between 60 and 120 ms prior to unloading, and the resulting displacement varied from 7° to 2°. We suggest that this strong coupling between bilateral muscle changes prevented a perfectly timed anticipatory compensation. It also implies that a perfect compensation would have necessitated either earlier EMG activity changes on both sides or a decoupling of the EMG latency difference between the two sides. Decoupling would signify that postural inhibition would have had to slightly precede (90-30 ms) contralateral activation if it remained between 60 and 120 ms. Evidence for a strong coupling between the motor commands in both limbs comes not only from the observation of similar latencies in the bilateral EMG changes during active conditions (A1 and A2) but also from its persistence in unloading conditions where coupling of...
latencies would be inappropriate. Indeed, when a 150-ms delay was purposely introduced between the muscle activation on one side and the unloading on the other side (by making a loop in the rope), the coupling persisted for most trials and resulted in a premature postural adjustment, causing elbow extension. Nevertheless, decoupling the two sides was sometimes observed in normal subjects in the last trials of the delayed unloading condition. In these cases, biceps inhibition was delayed in relation to the contralateral unloading muscles. The reverse situation, in which the postural inhibition preceded the activation of the unloading side, was also observed on a few occasions in the other active unloading condition using the contralateral arm (A₁ and A₂). These decouplings were observed in the last trials at the end of the series. It is probable that this behaviour might be observed more frequently after a larger number of trials, so that training would optimize compensation (i.e., result in less displacement). The small number of trials used in our experimental conditions were not sufficient to study long-term adjustments. In experiments where acquisition of APA has been studied during unloading tasks (Paulignan et al. 1989), 40-60 trials were necessary before the postural adjustment would stabilize.

Unusual unloading condition

When unloading was produced by flexing the contralateral leg, the deafferented subject did not show an APA and her displacement amplitudes were similar to those in the passive condition (P₁). The normal subjects did show an APA, but not on the first trial; some of them required three or four trials before an APA was observed. Thus it appears that proprioceptive and (or) cutaneous feedback are necessary for rapidly learning how to produce an APA during a very unusual unloading condition. It remains to be determined if peripheral feedback from the limbs is necessary for learning this anticipatory adjustment, or whether it could be developed by the deafferented patient by trial and error, or by using other feedback mechanisms (i.e., vision) during long-term training. The leg unloading condition also differed from the arm unloading conditions in that it involved a deactivation (i.e., quadriceps inhibition) of the unloading muscle activity instead of an activation. This condition was probably not as efficient for unloading as conditions using the arm since it involved a longer delay between the change in muscle activity in the quadriceps and the unloading produced by gravity acting on the leg. The introduction of this delay meant that a decoupling was needed between the timing of the deactivation of the quadriceps and of the postural biceps.

Origin of the APA

We previously showed the presence of the APA in the patient during active unloading with a contralateral elbow flexion (Forget and Lamarre 1990), thus supporting a central origin for this postural adjustment. The present results show that the APA is also present using contralateral elbow extension. This suggests that the central command responsible for the APA can be independent of the direction of the active movement and of the choice of the contralateral elbow muscles used to unload. The APA can even be produced by deactivating contralateral leg muscles (albeit, it must be learned using peripheral feedback), showing that it is not rigidly linked to motor commands from fixed muscular groups. However, it cannot be triggered generally by any type of motor command, since active switching of the electromagnet did not produce the APA. In this case, the distal muscle used to deactivate the mechanical system generated minimal force to close the switch and did not directly produce the unloading. It has been suggested that the intensity of the motor command is an important factor in eliciting a response (Dufossé et al. 1985) particularly during acquisition of the task (Paulignan et al. 1989). In addition, Paulignan et al. suggested that the use of a distal joint (first metacarpophalangeal), instead of a proximal one, was inappropriate for building up the coordination of the proximal contralateral postural muscles. The force of the triggering response does appear to be an important but not indispensable factor since the APA was learned in the leg flexion condition, where the motor command led to deactivation of all leg muscles. However, in this circumstance the motor command may not contain sufficient information, thus peripheral feedback might be required to learn when and how to produce the APA. Finally, in accordance with the results of Dufossé et al. (1985), who switched off an electromagnet to produce the unloading, knowing the precise moment at which unloading will take place (even when the subject controls the time of the unloading) is not sufficient information to generate an APA. Thus it appears that a motor command to the muscles that will be directly involved in the unloading is necessary to produce an APA. However, the degree of this involvement and the parameters necessary to plan the APA are still unclear.

Hugon et al. (1982) suggested that a "common command" is responsible for the EMG behaviour in the two arms. They based their hypothesis on the observation that the command for the APA and the active motor command that produce the unloading are coupled temporally. A hierarchical mode of control was suggested where the pathways controlling movement performance give off collaterals acting on the postural networks responsible for the APA (reviewed by Massion 1992). On the other hand, the decoupling of the two commands would support the notion control by parallel pathways (Horak et al. 1984; Lee et al. 1987). Tightly coupled EMG activity was normally observed in the present experiments. This coupling was also present in the deafferented patient, thus supporting the conclusion that coupling between the unloading and unloaded sides is mediated by descending pathways and not sensory afferents. Since decoupling was also observed in the normal subjects, both modes of control are probably available depending on the behavioural conditions of the task. However, decoupling was not documented in the absence of sensory feedback in the present experiments. Indeed, in the patient, there was no APA in the unusual unloading condition (knee flexion) and therefore no possibility to compare the EMG latencies on both sides. Furthermore, the experimental paradigm that introduced a temporal delay was not feasible. Future studies should address whether decoupling can be generated or learned without peripheral afferents.

Functional aspects of posture

A proper postural adjustment is needed for unloading of the hand during bimanual coordination. The anticipatory nature of this adjustment is a sine qua non condition for a stable system, its latency being linearly related to the size of the perturbation. Since it must be planned and executed before
the unloading, it should be, and it was, demonstrated that this feedforward command can be performed in the absence of peripheral feedback. However, a proper APA in a bimanual task is just part of a dynamic aspect of posture (i.e., postural adjustments) and it is not indicative of proper posture as a whole. Once the postural adjustment to the changing conditions is made, the new posture must be held for variable periods of time. The maintenance of static posture (i.e., postural holds) requires the maintenance of a constant EMG level. However, in all our conditions the arm of the patient started to drift after the postural adjustment. Since the inability to maintain a constant force output is one of the most constant findings in patients with sensory deficits (Rothwell et al. 1982; Forget and Lamarre 1985; Sanes et al. 1985), these two aspects of posture (dynamic and static) must be controlled in a very different manner, the former being in an open-loop mode, but the latter requiring closed-loop control via peripheral sensory feedback. The absence of postural reactions following perturbations and the difficulty to maintain a posture, despite proper APA with active movement, are severe incapacities that handicap a deafferented patient in activities of daily living.

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References


