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## Effects of galvanic mastoid stimulation in seated human subjects

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**Ghanim Z, Lamy JC, Lackmy A, Achache V, Roche N, Pénicaud A, Meunier S, Katz R.** Effects of galvanic mastoid stimulation in seated human subjects *J Appl Physiol* 106: 893–903, 2009. First published December 18, 2008; doi:10.1152/jappphysiol.90594.2008.—The vestibular responses evoked by transmastoid galvanic stimulation (GS) in the rectified soleus electromyogram (EMG) in freely standing human subjects disappear when seated. However, a GS-induced facilitation of the soleus monosynaptic (H and tendon jerk) reflex has been described in few experiments in subjects lying prone or seated. This study addresses the issue of whether this reflex facilitation while seated is of vestibulospinal origin. GS-induced responses in the soleus (modulation of the rectified ongoing EMG and of the monosynaptic reflexes) were compared in the same normal subjects while freely standing and sitting with back and head support. The polarity-dependent biphasic responses in the free-standing position were replaced by a non-polarity-dependent twofold facilitation while seated. The effects of GS were hardly detectable in the rectified ongoing voluntary EMG activity, weak for the H reflex, but large and constant for the tendon jerk. They were subject to habituation. Anesthesia of the skin beneath the GS electrodes markedly reduced the reflex facilitation, while a similar, although weaker, facilitation of the tendon jerk was observed when GS was replaced with purely cutaneous stimulation, a tap to the tendon of the sternomastoid muscle, or an auditory click. The stimulation polarity independence of the GS-induced reflex facilitation argues strongly against a vestibular response. However, the vestibular afferent volley, insufficient to produce a vestibular reflex response while seated, could summate with the GS-induced tactile or proprioceptive volley to produce a startle-like response responsible for the reflex facilitation.

vestibular afferents; sensory afferents; H and T reflexes; startle pattern; galvanic stimulation; humans

GALVANIC MASTOID STIMULATION (GS) has been used for over a century to probe the human vestibular system (see Ref. 15). The technique is simple and consists of a small long-duration rectangular current step applied through electrodes placed on the mastoid processes: generally the anode is placed on one mastoid process and the cathode on the other, but monopolar stimulation with a stimulating electrode behind one ear and the other at a distance induces similar effects (27). In a recent review, Fitzpatrick and Day (15) consider that the complex bodily response in freely standing human subjects, at least in its early stages, “reveals the operation of the balance system to a pure vestibular perturbation.” This view is supported by the fact that in various animal species, including primates, GS has

been proved to modulate the spontaneous firing of vestibular afferents: their frequency is increased on the cathodal side and decreased on the anodal side (see Refs. 7, 16, 18). Recent experiments performed in humans (8, 26) also support the hypothesis that GS causes a change in vestibular afferent firing.

In the freely standing human subject (i.e., a subject standing without support), previous studies have consistently shown that GS produces a head movement signal that has a potent effect on whole body motor control and that it causes reflex responses to appear in the electromyogram (EMG) of trunk and limb muscles (for review, see Ref. 15). Biphasic EMG responses are observed soon after current step stimulation: e.g., in the soleus after ipsilateral anodal stimulation, there is a facilitation (at ~55–65 ms) followed by an inhibition (at ~110–120 ms), with reversal (facilitation followed by inhibition) when the polarity is inverted. In the lower limb muscles, Fitzpatrick and Day (15) stressed that GS-induced EMG responses usually appeared if the muscle is engaged in the balance task. In their study of vestibular actions on back and hindlimb muscles during postural tasks in humans, Ali et al. (1) showed that in sitting position, responses are detectable in back muscles but not in lower limb muscles. In neck muscles, Watson et al. (28) and Colebatch and Rothwell (6) have described responses to galvanic vestibular stimuli while these muscles were not engaged in a balance task. In 1997, Day et al. (9), studying human body segment tilts (head, torso, and pelvis) induced by GS, indicated that tilts are still present, although strongly reduced, when subjects are seated on a stool. Furthermore, results of the rare experiments performed in the soleus EMG of nonstanding subjects (i.e., in lying or sitting subjects) are incongruent. Indeed, in subjects lying prone, Kennedy and Inglis (17) found a weak inhibition of the H reflex with anodal GS stimulation, at 100-ms interstimulus interval (ISI), which reversed to facilitation with cathodal stimulation. In seated subjects maintaining a voluntary soleus contraction, Fitzpatrick et al. (14) did not notice any GS-induced modulation of the ongoing EMG. However, Delwaide and Delbecq (12) found a marginal facilitation of the H reflex in seated subjects contrasting with a significant facilitation of the tendon jerk, which was uninfluenced by stimulation polarity. The question thus remains whether in the ankle muscles of seated subjects, vestibular activation resulted in changes at the level of soleus motoneurons. One aim of the present study was to compare, in seated subjects, the full time course of the GS-induced modulation of the soleus H and tendon jerk (T) reflexes and that of a soleus ongoing voluntarily EMG activity. Because

Address for reprint requests and other correspondence: R. Katz, UMRS 731 INSERM, Univ Paris 06, Médecine Physique et Réadaptation, Hôpital de la Salpêtrière, 75651 Paris cedex 13, France (e-mail: rose.katz@upmc.fr).

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we found that GS-induced changes in the monosynaptic reflexes in the sitting position were insensitive to stimulus polarity, we came to ask where these changes came from and whether stimulation of afferents other than the vestibular ones could contribute to them. In this respect, as a second step, we studied the effects of other afferents possibly activated by the GS: 1) cutaneous afferents, because the GS induced a local sensation under the electrodes; 2) neck muscle afferents in the sternomastoid muscle (SMM) which, given the upper insertion of the muscle on the mastoid process, could have been activated by spread of GS; and 3) an auditory signal because the latency of the effects was comparable to that of the startle reflex.

## METHODS

### General Experimental Procedure

The experiments were carried out on 28 healthy volunteers (age 22–58 yr, 19 women). All subjects gave written informed consent. The experimental protocol was approved by the ethics committee (Comité Consultatif de Protection des Personnes dans la Recherche Biomédicale Paris-Pitié-Salpêtrière) and conformed to the guidelines in the Declaration of Helsinki. In most experiments, the subjects were seated in an armchair with the back of the seat tilted slightly backward, the head straight and supported by a headrest, and the upper limbs supported by armrests. The limb carrying the soleus electrodes (the right leg) was fixed loosely with the hip semiflexed ( $120^\circ$ ), the knee slightly flexed ( $160^\circ$ ), and the ankle at  $110^\circ$ , while the foot was supported on a rest. The left leg was at rest with the foot resting on the floor. EMG activity was recorded in the right soleus by surface electrodes (silver plates, 0.8-cm diameter, 1.5 cm apart) secured to the skin over the muscle belly.

### Conditioning Stimuli

**GS.** A constant current isolated stimulator delivered the GS (electrical rectangular pulses of 300-ms duration, intensity varying from 2 to 4.5 mA among the subjects) through half-ball electrodes (2.5-cm diameter) placed over the mastoid processes and secured with tape. Two kinds of GS were used: 1) bimastoid stimulation: cathode on the right mastoid process and anode on the left mastoid process (see the sketch in Fig. 1) and vice versa and 2) monopolar mastoid stimulation with one electrode on the right mastoid and the other 10 cm below on the lateral part of the base of the neck (see the sketches in Fig. 2, A and D). Because GS in our experimental paradigm (see below) does not induce any body sway in the sitting position, we first verified that GS produced the expected body sway and soleus EMG responses in the free-standing position.

**Effects of habituation.** To test the effects of habituation, the amount of facilitation was calculated for the first 5 conditioned and the last 5 conditioned reflexes in each run of 40 reflexes for 40- and 100-ms ISIs. Three runs were recorded for each ISI, and the time interval between each run was  $\sim 1$  min.

**Cutaneous stimulation.** The electrode, originally placed on the right mastoid process in the unipolar GS configuration, was lowered a few centimeters to the lateral part of the neck, to induce a similar cutaneous sensation, but without the illusion of movement (all other characteristics of the electrical stimulation being the same; see the sketch in Fig. 5C).

**Cutaneous anesthesia.** In these experiments, the skin beneath the electrodes was anesthetized with an anesthetic gel (RIVADIS) to suppress the cutaneous sensation. Skin anesthesia lasted for  $\sim 45$  min. As a result, with unipolar GS, the cutaneous sensation of pricking disappeared while the illusion of movement remained the same.

**Mechanical percussion of the SMM.** The electrical stimulation was replaced by a mechanical tap (same electromagnetic hammer as that used to evoke the soleus tendon jerk, see below) applied to the tendon of the SMM to activate muscle spindle afferents (see the sketch in Fig. 5E).

**Auditory stimulus.** In this series of experiments, the GS was replaced by an auditory stimulus produced by a loudspeaker situated around 60 cm from the right ear (see sketch in Fig. 5G). The loudspeaker was driven by a square electrical pulse. In the first series of experiments, we used a click of 3-ms duration ( $\sim 90$  dB), comparable to that used by one of the authors in a previous study devoted to the postural reaction to sudden body displacement (2). In the second series of experiments, we used a square of 30-ms tone burst at 700 Hz (comparable to the auditory stimulus most often used to study the auditory startle response; see for example, Refs. 13, 29).

### Reflex Experiments

We compared the time courses of the effects of the conditioning stimuli on the soleus H reflex and the soleus tendon jerk. To that end, we varied the ISI between conditioning and test stimuli between 0 and 300 ms (using 10- to 20-ms steps) among blocks of stimulations (Fig. 1, B and C). Twenty control test reflexes and 20 test reflexes preceded by conditioning stimulation were randomly intermixed in each block. Thus the time between two conditioning stimuli was unpredictable for the subjects and varied from 3 to 15 s. Test reflexes were triggered every 3 s (see Fig. 1, B and C). By convention, the timing of the test stimulus is referred to that of the onset of the conditioning stimulus (Fig. 1, B and C).

Test reflexes were evoked in the right soleus. Soleus H reflexes were evoked by stimulation of the posterior tibial nerve (1-ms rectangular pulses) through a monopolar stimulating electrode (active cathode in the popliteal fossa, anode on the anterior aspect of the knee). Soleus T reflexes were evoked by a mechanical tap applied to the Achilles tendon by an electromagnetic hammer (model 4809, Bruel and Kjaer, Naerum, Denmark) which produced a rapid-transient stretch. The intensity of the percussion was graded using a power amplifier, and special care was taken to ensure that the hammer struck the tendon in the same position throughout the experiment. Nonrectified reflex responses were assessed peak to peak. The size of the control reflexes, H and T, was adjusted so that, in each subject, the two reflexes had approximately the same amplitude (6–38% of the maximum motor response, according to the subjects).

### Modulation of the Rectified Ongoing EMG Activity

The subjects were asked to perform a slight tonic isometric contraction 10–15% of the maximum voluntary contraction of the right soleus. The rectified voluntary ongoing EMG activity of the soleus was displayed on an oscilloscope so that the subjects could maintain a constant contraction level throughout the experiment. Ongoing EMG activity was filtered (100 Hz to 1 kHz), full-wave rectified, and digitized using a sampling rate of 1–2 kHz during the 300 ms following the onset of GS (Fig. 1D). A total of 100 trials, 50 unconditioned trials (i.e., trials in which the background soleus EMG activity was assessed) and 50 conditioned trials, were regularly alternated every 1 s in each sequence. The data recorded over 5 sequences were averaged to produce a single run containing 250 conditioned responses. The rectified background EMG was measured in the corresponding unconditioned trials and then integrated over 300 ms to provide a fixed measure of baseline rectified EMG within the sequence (mean rectified unconditioned EMG). The difference between the grand average of conditioned values (in a single subject) and the baseline rectified EMG was expressed as a percentage of this baseline. Data so obtained in single subjects were averaged to provide the grand average of the population.

### Statistical Analysis

Statistical analyses were performed using Statview 5. The mean and SE were calculated online for each individual experiment. Statistical analysis was carried out at individual and group levels. At the individual level, the comparison was done for each tested ISI, between test (20 values) and conditioned (20 values) reflex sizes using a paired *t*-test. For

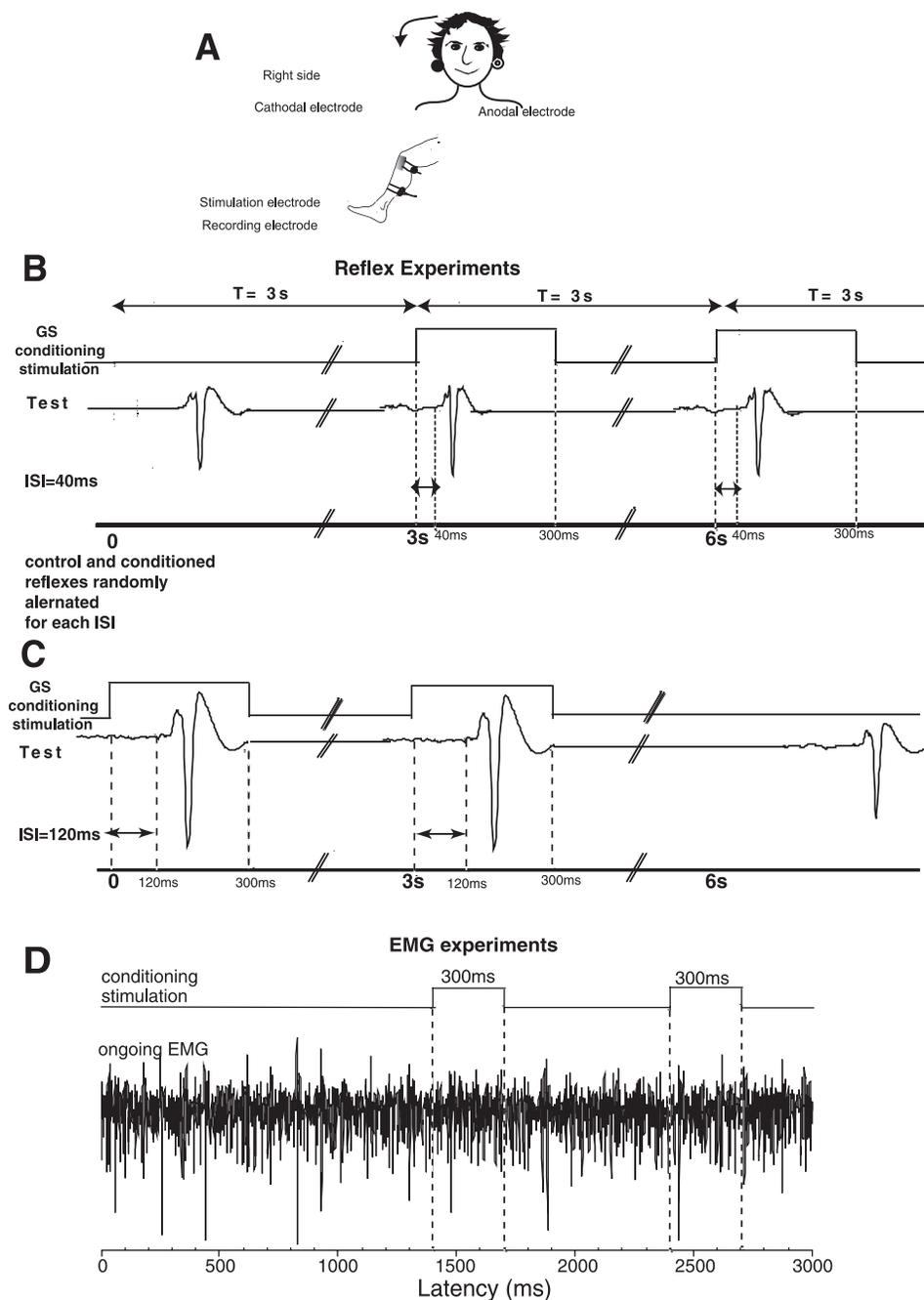


Fig. 1. General experimental setup. *A*: the drawing represents the galvanic mastoid stimulation (GS) electrode position on the right and left mastoid processes, the right posterior tibial nerve stimulation at knee level, and the recording electrodes on the right soleus muscle. The arrow indicates the direction of the illusory head movement in sitting position; *B*: in reflex experiments, the test reflexes are evoked every 3 s ( $T = 3$  s). Unconditioned (control) reflexes and reflexes conditioned by GS are randomly alternated. The time interval between the GS trigger and that of the test reflex is 40 ms (horizontal arrows). *C*: time interval between the GS trigger and that of the test reflex is 120 ms (horizontal arrows). *B*, examples of H reflexes are presented; in *C*, examples of tendon jerk (T) reflexes are represented. *D* in rectified EMG experiments, the GS is delivered at 1-s intervals during weak tonic contraction of the soleus muscle. ISI, interstimulus interval; EMG, electromyogram.

the group level analysis, because some subjects participated several times in the protocol, all the observations for a given subject were averaged. To find out whether soleus H- and T-reflex sizes were significantly modulated by conditioning stimuli over time in the whole group of subjects, values of the conditioned reflex, normalized with respect to baseline H- or T-reflex sizes, were entered in a repeated-measures ANOVA analysis. The values of the reflex at all the tested ISIs (0, 20, 30, 40, 50, 70, 100, 120, 140, 160, 180, 200, 220, 240, 260, 280, and 300 ms) formed the repeats. When there was a significant effect of "ISI," a post hoc analysis using a Fisher test was done to compare pairwise the values obtained at the different ISIs. Asterisks drawn on the figures indicate statistically significant differences of the tested parameter between the corresponding ISI and the 0-ms ISI.

The effect of anesthetizing the skin beneath the electrode delivering GS stimulation was tested by a two-level repeated-measures ANOVA

with the values of the T reflex at the different ISIs either with or without cutaneous anesthesia forming the repeats.

## RESULTS

### *Checking the Efficiency of GS in Eliciting Responses of Vestibulospinal Origin in Standing Position*

As stated in METHODS, to ensure that GS used in the present experiments was able to elicit vestibular responses, we first verified that it produced the expected body sway and soleus EMG responses in the free-standing position: the subjects stood erect, feet together in the frontal plane, eyes closed, and the head looking over the left shoulder. Then, monopolar GS stimulation was applied (see METHODS and Fig. 2 *A* and *D*). When

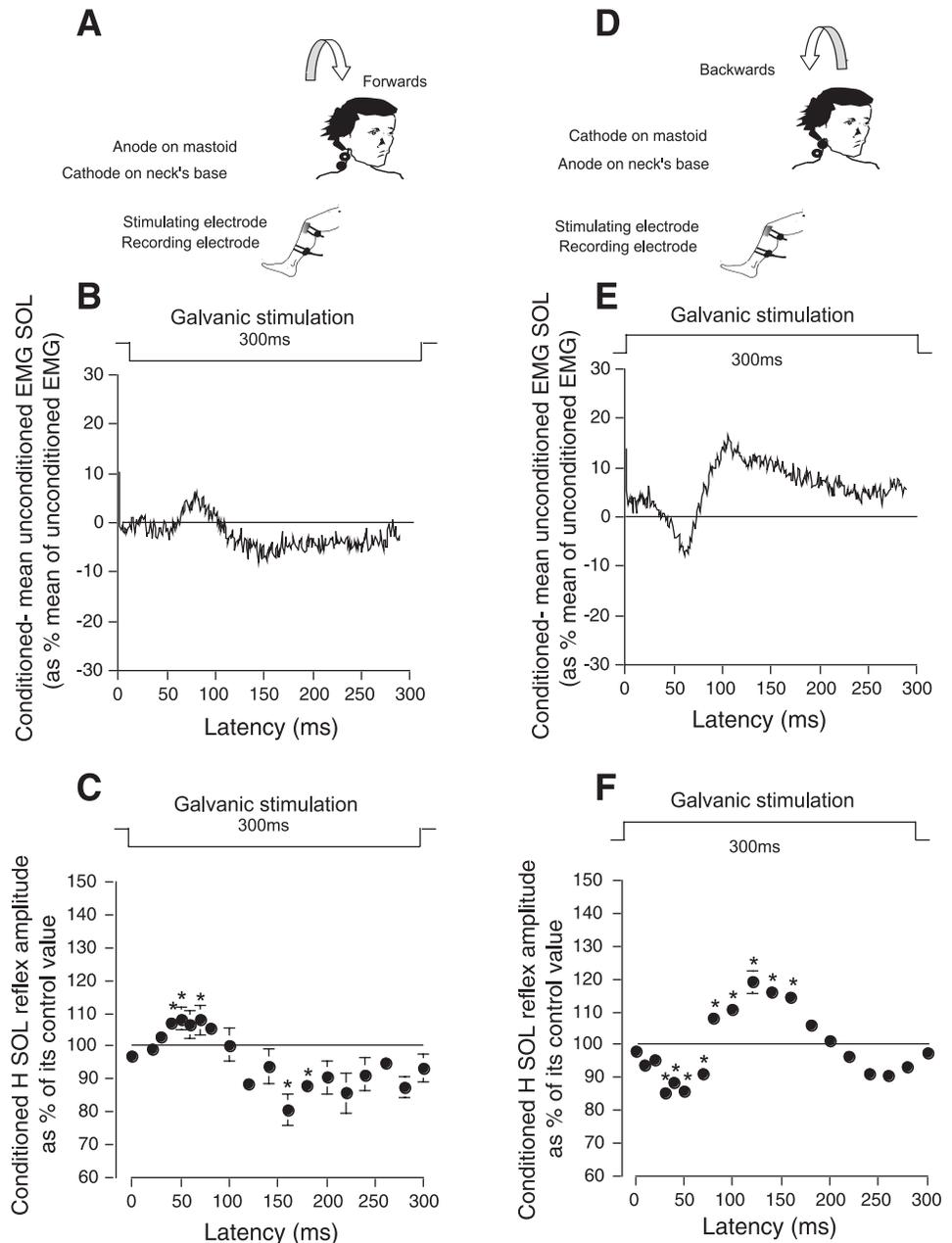


Fig. 2. Effects of the reversal of GS polarity in the standing position. *A*, *B*, and *C*: monopolar mastoid stimulation with the anode placed on the mastoid process and GS eliciting a forward sway. *D*, *E*, and *F*: monopolar mastoid stimulation with the cathode placed on the mastoid process and GS eliciting a backward sway. *B* and *E*: EMG experiments. Time course of the changes in rectified ongoing EMG activity induced by GS. *x*-Axis: latency from GS onset. *y*-Axis: difference between conditioned and mean unconditioned EMG expressed as a percentage of the mean unconditioned EMG. *B*: grand average of 21 experiments performed on 10 subjects with the anode on the mastoid (5,250 trials). *E*: grand average of 33 experiments performed on 12 subjects with the cathode on the mastoid (8,250 trials). *C* and *F*: reflex experiments. Time course of the GS-induced changes in H reflex amplitude. *x*-Axis: latency from GS onset. *y*-Axis: conditioned H-reflex amplitude as a percentage of its control value. Vertical bars around the symbols represent 1 SE. \*Statistically significant post hoc Fisher test changes in H-reflex amplitude,  $P < 0.05$ . *C*: 18 experiments were performed on 8 subjects with the anode on the mastoid. *F*: 48 experiments were performed on 9 subjects with the cathode on the mastoid. Sol, soleus.

the anode was placed over the mastoid (Fig. 2A), the subjects swayed forward. In the reverse situation, i.e., with the cathode placed on the mastoid, the subjects swayed backward (Fig. 2D). Changes in the rectified ongoing EMG activity and in the H-reflex amplitude were investigated.

GS produced biphasic changes in the rectified ongoing soleus EMG activity, which were inverted when the polarity was reversed (Fig. 2, *B* and *E*). With the anode on the right mastoid process (Fig. 2*B*, grand average, 21 experiments, 10 subjects), the effect was a short-latency facilitation followed by a subsequent inhibition. With the cathode on the right mastoid process (Fig. 2*E*, grand average, 33 experiments, 12 subjects), the effects were reversed: a short-latency inhibition followed by a subsequent facilitation as described in a previous report (see Ref. 14).

GS-induced biphasic changes in the soleus H reflex paralleled those seen in the rectified ongoing soleus EMG activity (as the

latency of the H reflex,  $\sim 35$  ms, has to be added to reach the latency for the effects measured in the EMG; see Ref. 23). When the anode was placed over the mastoid process (Fig. 2*C*, grand average, 18 experiments, 8 subjects), GS produced an early facilitation (between 30 and 100 ms) of the H reflex followed by an inhibition. Both effects were statistically significant (ANOVA, effect of ISI:  $F = 7.6$ ,  $P < 0.0001$ ). When the polarity of GS was reversed (Fig. 2*F*, 48 experiments, 9 subjects), GS produced an early inhibition, (between 10 and 80 ms) followed by a facilitation peaking at 120 ms which both reached statistical significance (ANOVA, effect of ISI:  $F = 45$ ,  $P < .0001$ ).

#### Effects of GS in the Sitting Position

In the sitting position with back rest and head rest, the perceptual effects and the changes in soleus motoneuron

excitability differed substantially from those observed in the standing position without support. No body or head sway was observed, but subjects described an illusory tilt of the head toward the right shoulder, when the cathode was placed on the right mastoid, whether 1) the position of the head was facing or rotated to the left shoulder, and 2) the position of the anode was on the left mastoid or on the right lateral part of the base of the neck, a few centimeters below the cathode. It is also worth noting that there was a decrease in these perceptual effects over time. The importance of this habituation varied among subjects, but it was often necessary to

increase the intensity of GS every 10–15 min to maintain the illusory tilt.

When seated, GS-induced changes in soleus motoneuron excitability are absent or hardly discernible in both rectified ongoing EMG activity and H reflex amplitude, but clear cut in the T-reflex amplitude (cf. the introduction and RESULTS below). Therefore, as a first step, the effects of the same GS stimulation used in standing position (right monopolar GS stimulation, head rotated to the left shoulder) were studied in the H reflex of the two subjects in which GS-induced changes in the soleus H reflex were clear cut (Fig. 3A).

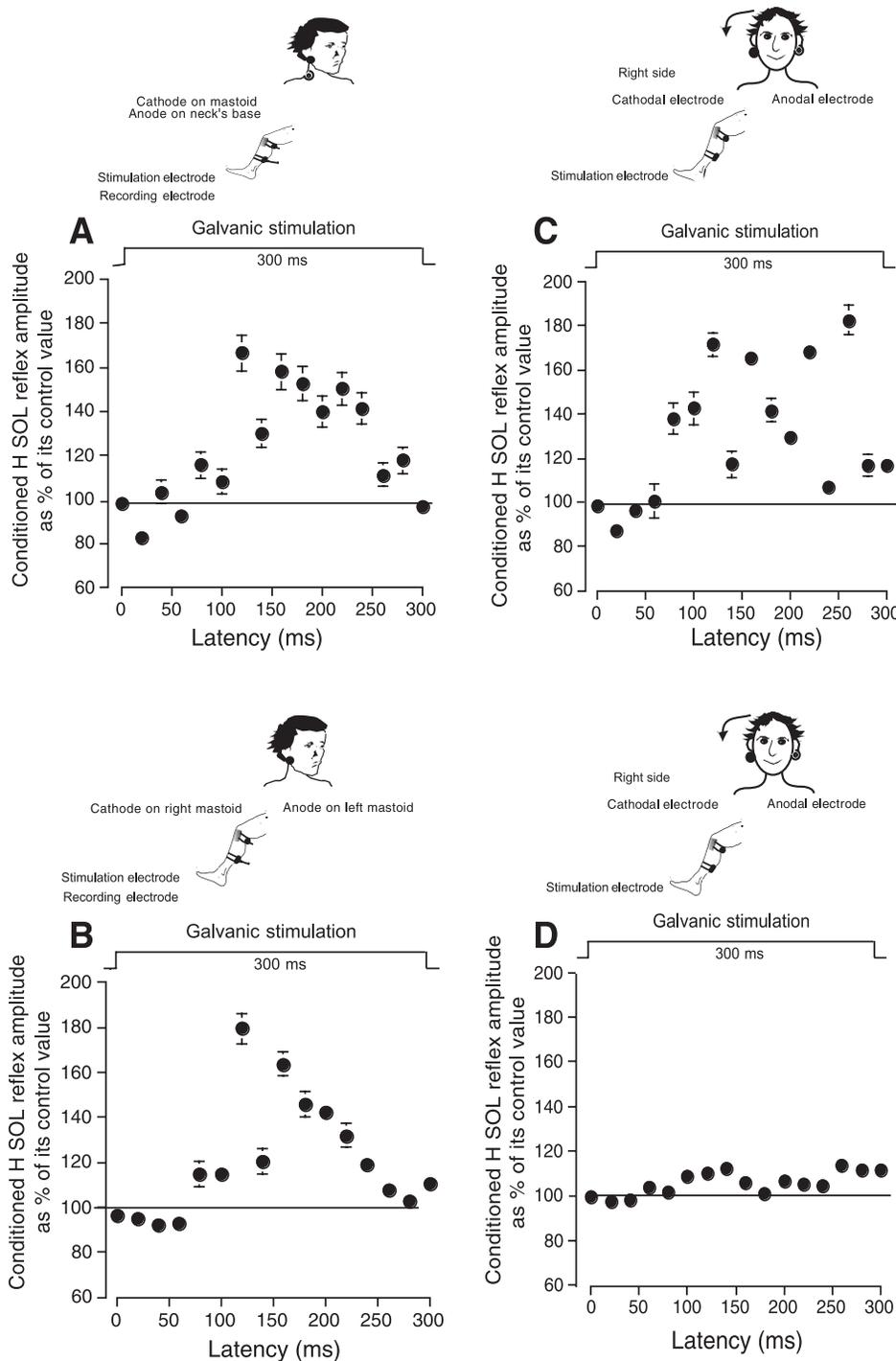


Fig. 3. Effects of GS polarity and head position on GS-induced changes in the soleus H reflex in the seated position. *A*, *B*, and *C*: effects of GS stimulation on H-reflex amplitude in 1 of the 2 subjects for whom changes were significant (see text). *A*: monopolar, cathode on the right mastoid, head rotated over the left shoulder. *B* and *C*: bimastoid GS stimulation (cathode on the right mastoid, anode on the left mastoid); in *B* with head rotated over the left shoulder and in *C* with head facing. *D*: grand average of 10 experiments performed in 7 subjects with bimastoid GS (cathode on the right mastoid, anode on the left mastoid) head facing. y-Axis: amplitude of the conditioned H reflex expressed as a percentage of its control value. y-Axis: latency from onset of galvanic stimulation. *A*, *B*, and *C*: each symbol represents the mean of 20 reflexes. *D*: each symbol represents the mean of 200 reflexes. Vertical bars around the symbol represent 1 SE.

Taking into account that bimaistoid GS stimulation induced larger effects than monopolar stimulation (26), the same experiments were repeated with bimaistoid GS stimulation (Fig. 3B). In the second step, experiments were performed with soleus T reflexes, in which different influences were tested to determine the origin of the GS-induced changes in soleus motoneuron excitability: 1) change in head position; 2) change in stimulus polarity; and 3) effects of cutaneous, proprioceptive, and auditory stimuli.

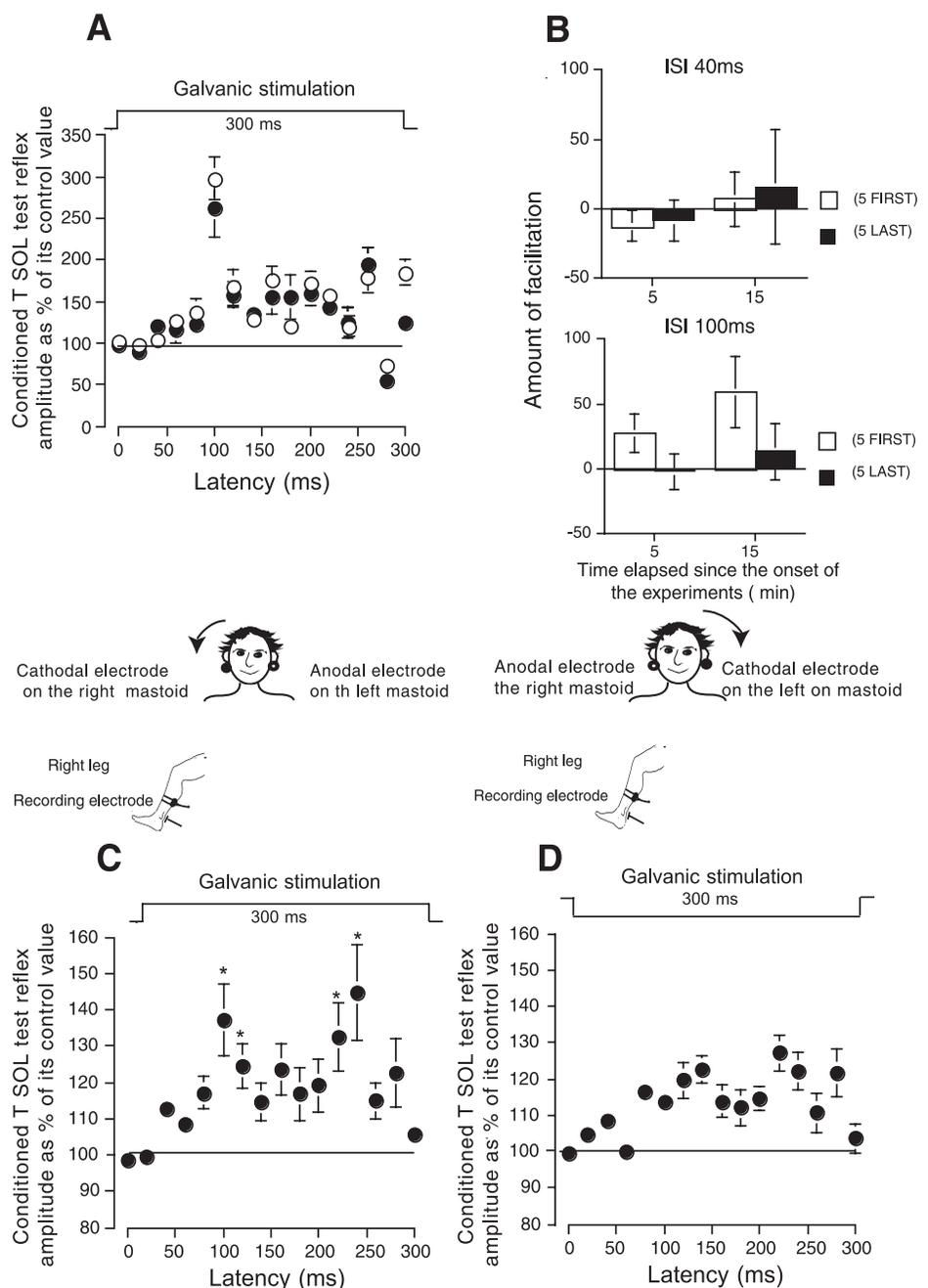
**GS-induced changes in the soleus H reflex.** The effects of GS stimulation on H-reflex amplitude are illustrated in Fig. 3. They differ considerably from those obtained in the standing position because the biphasic modulation (Fig. 2) was replaced by a weak and nonsignificant facilitation (ANOVA, effect of ISI:  $F = 1.6$ ,  $P > 0.07$ ; Fig. 3A, grand average, 10

experiments, 7 subjects). Figure 3, A–C, obtained in one of the two subjects in whom this facilitation was clearcut, shows that the effect was roughly similar whether GS stimulation was monopolar (Fig. 3A) or transmastoid GS stimulation (Fig. 3, B and C) and whether the head was rotated to the left shoulder (Fig. 3B) or facing forward (Fig. 3C).

#### GS-Induced Changes in the Soleus T Reflex

In contrast with the inconsistent H-reflex facilitation (Fig. 3D), GS produced a large and robust facilitation of the tendon jerk amplitude in seated subjects. Figure 4C shows the results observed with the cathode on the right mastoid. GS induced a twofold significant facilitation of the T-reflex amplitude with a first phase between 50 and 160 ms fol-

Fig. 4. Time course of bimaistoid GS effects on the right soleus, in the sitting position. A, C, and D: the plots are arranged as in Fig. 2, C and F. A: effects of GS on soleus T reflex, cathode on the right mastoid in one experiment. ○, Head rotated to the left shoulder; ●, head facing. Each symbol is the mean of 20 reflexes; B: habituation effects. Open bars, amount of facilitation of the first 5 conditioned reflexes; filled bars, amount of facilitation of the last 5 conditioned reflexes in a series of 40 reflexes performed 5 and 15 min after the beginning of the experiment. Top graph: time interval between the onset of GS and T reflexes is 40 ms. Bottom graph: time interval between the onset of GS and T reflexes is 100 ms; y-Axis amount of facilitation:  $(T_{\text{conditioned}} - T_{\text{control}}) / T_{\text{control}}$ . C: changes in T-reflex amplitude following bimaistoid GS stimulation with the cathode on the right mastoid. Grand average from 10 experiments performed in 8 subjects. D: changes in T-reflex amplitude following bimaistoid GS (cathode on the left mastoid). Grand average from 8 experiments performed on 8 subjects. \*Statistically significant post hoc Fisher test,  $P < 0.05$ .



lowed by a second phase between 180 and 300 ms (ANOVA, effect of ISI,  $F = 2$ ,  $P < 0.02$ ; Fig. 4C, grand average, 10 experiments, 8 subjects). Figure 4A, obtained in one subject, shows that the effects obtained with head facing forward (filled circles) are similar to those obtained with the head rotated to the left shoulder (open circles). The fact that head position did not influence the GS-induced effects was confirmed in the seven experiments performed in seven subjects.

**Effect of GS polarity reversal.** Although reversing the stimulus polarity (i.e., with the cathode on the left mastoid) did result in an inversion of the illusory head movement, GS-induced changes in T-reflex amplitude remained facilitatory in both cases (Fig. 4, C and D). Thus GS polarity reversal induced a twofold significant facilitation with a first phase between 80–180 ms and followed by a second phase beginning at 200 ms (ANOVA, effect of ISI,  $F = 8.9$ ,  $P < 0.004$ ; Fig. 4D, grand average, 8 experiments, 8 subjects).

**Effects of habituation.** Figure 4B shows that GS-induced facilitation was more marked for the first (open squares) than for the last reflexes (filled squares) at the 100-ms ISI (bottom graph; ANOVA, effect of habituation,  $F = 4.9$ ,  $P < 0.05$ ), whereas there was no difference in the conditioned reflexes at the 40-ms ISI (top graph; ANOVA, effect of habituation,  $F = 0.29$ ,  $P = 0.59$ ).

#### *Effects of Stimulation of Non/vestibular Afferents*

**Effects of skin anesthesia beneath the electrodes.** Skin anesthesia caused the local pricking beneath the electrodes to disappear, while the head movement illusion was the same as with no anesthesia. Figure 5B (grand average, 12 experiments, 8 subjects) shows that, during the period of skin anesthesia, the two phases of the GS-induced facilitation of the T reflex were depressed (open circles) compared with the preanaesthesia results (filled circles) (ANOVA, effect of anesthesia,  $F = 5.8$ ,  $P < 0.03$ ).

**Effects of selective activation of cutaneous afferents.** Moving the electrode to slightly below the mastoid produced a purely cutaneous sensation with no head movement illusion (and without any muscle contraction or pain). Figure 5D (grand average, 13 experiments, 7 subjects) shows that this purely cutaneous stimulation produced a twofold facilitation of the soleus T reflex with a time course similar to that induced by GS. Although weaker than the GS-induced facilitation, (Fig. 5B, filled circles), the cutaneous-induced facilitation remained statistically significant (ANOVA, effect of ISI,  $F = 5.3$ ,  $P < 0.007$ ). Anesthesia of the skin beneath the electrodes resulted in a complete suppression of the cutaneous-induced facilitation (3 of 3 subjects tested).

**Effects of mechanical stimulation applied to the SMM tendon.** Mechanical stimulation of SMM tendon, activating both muscle spindles and cutaneous receptors, resulted in a twofold significant facilitation (ANOVA, effect of ISI,  $F = 3.3$ ,  $P < 0.01$ ; Fig. 5F, grand average, 6 experiments, 6 subjects), which was more marked than that produced by a purely cutaneous stimulus (Fig. 5D).

**Effect of auditory stimulation.** Figure 5H, obtained in 1 subject, shows that the effects produced by a 3-ms click (filled circles) and a 30 ms auditory stimulus (open circles) were similar and resembled to that of GS stimulation obtained in the

same subject (Fig. 3, A and C). Such a facilitation, beginning ~50 ms after the onset of the auditory stimulus, was found for both a 3-ms click (7 experiments, 7 subjects) and a 30-ms auditory stimulus (6 experiments, 5 subjects).

#### *Modulation of the Rectified Ongoing EMG Activity*

Results obtained in sitting position (Fig. 6) differed markedly from those observed in standing position (Fig. 2, B and E). Individual results obtained in different subjects showed that GS either did not modify the rectified ongoing EMG activity (Fig. 6, A, B, F, and G) or else induced only small variations (Fig. 6, C, D, H, and I). In any case, the same modulation was observed whatever the polarity of GS (compare *left* and *right* columns). Consequently, Fig. 6 E, J (grand average, 7 experiments, 4 subjects) shows that whatever the stimulus polarity, GS did not produce discernable change onto rectified ongoing EMG activity in sitting position.

#### DISCUSSION

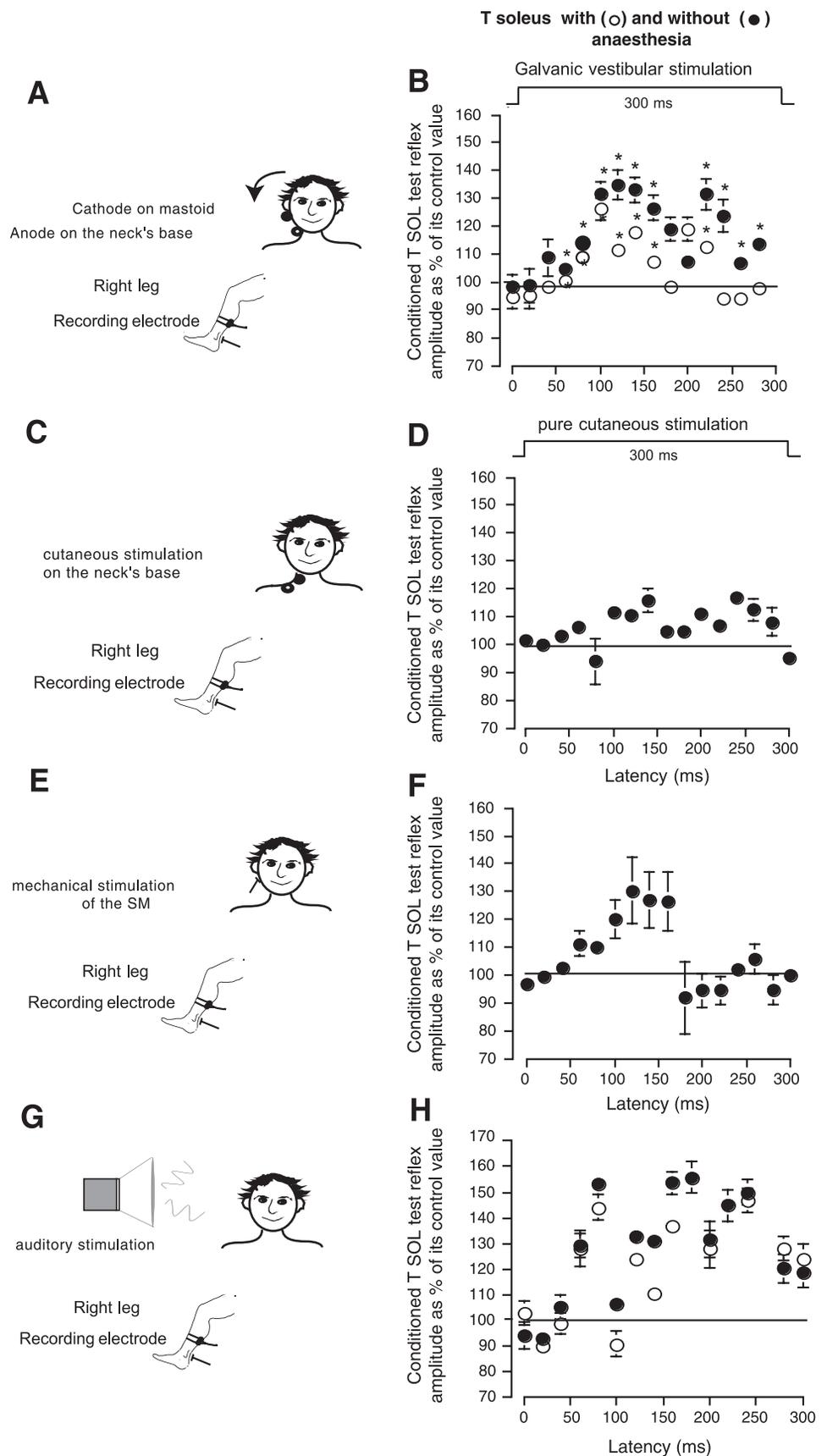
The present study shows that, when seated, GS produces a facilitation in the soleus, which was large for the tendon jerk, very weak for the H reflex, and hardly discernable in the rectified ongoing EMG. This effect was independent of GS polarity and subject to habituation. A similar facilitation was observed when GS was replaced by purely cutaneous stimuli, mechanical stimulation of the SMM tendon, or an auditory stimulus.

The disappearance of the GS-induced biphasic polarity-dependent modulation of the rectified ongoing soleus EMG while seated confirms previous observations (see Refs. 3, 14). Furthermore, the polarity independence of the GS-induced facilitation of the H and T reflexes argues strongly against a vestibulospinal reflex. However, effective stimulation of vestibular afferents was supported by the polarity-dependent illusion of head movement and the fact that, in freely standing subjects, typical biphasic polarity-dependent alterations of soleus motoneuron excitability were produced by the same GS stimulation (Fig. 2). In this respect, Kennedy and Inglis (17) reported a very small polarity-dependent change in the soleus H reflex amplitude in subjects lying in a prone position. This discrepancy with the present results may be explained by the fact that they employed two large electrodes with the reference electrode placed over the wrist, so that “with this configuration electrode the subjects could barely perceive the 4 mA GS in prone position.” The most parsimonious explanation is therefore that the potent cutaneous effect described with the present experimental protocol overwhelms the very weak vestibulospinal effect reported by Kennedy and Inglis (17).

#### *Afferent Pathway*

If the GS-induced vestibular afferent volley is insufficient to trigger a vestibulospinal response while seated, we show below that it can summate with cutaneous afferent volleys to produce a startle response. The role of cutaneous afferents in the production of the GS-induced reflex facilitation when seated was demonstrated by the depression of this facilitation after anesthesia of the skin beneath the electrodes evoking GS (Fig. 5B) and the finding that a purely cutaneous stimulation produced a similar, although weaker, twofold facilitation of the tendon jerk (Fig. 5D). A role of SMM

Fig. 5. Effects of monopolar GS, mechanical percussion, and auditory stimulation on soleus T reflexes. The plots are arranged as in Fig. 4, *C* and *D*. *A* and *B*: time course of the effects of monopolar GS (cathode on the right mastoid) on the right soleus T reflex, with (○) and without (●) skin anaesthesia beneath the electrodes. *C* and *D*: time course of the effects of cutaneous stimulation, on the right soleus T reflex. *E* and *F*: time course of the effects of a mechanical percussive stimulus on the right Sol T reflex, applied to the sternomastoid (SM) muscle. *G* and *H*: time courses of the effects of a 3 ms click (filled circles) and a 30-ms auditory stimulus (open circles) onto the right Sol T reflex, in the same subject as that in Fig. 3, *A* and *C*. *y*-Axis: amplitude of conditioned T soleus reflexes as a percentage of their control values. *x*-Axis: latency from onset of the conditioning stimulation. Vertical bars around the symbols represent 1 SE. *B*: grand average from 12 experiments performed on 8 subjects. Each symbol represents the mean of 240 reflexes. *D*: grand average from 13 experiments performed on 7 subjects. Each symbol represents the mean of 200 reflexes. *F*: grand average from 6 experiments performed on 6 subjects. Each symbol represents the mean of 120 reflexes. *H*: experiments performed in 1 subject (the same as in Fig. 3, *A* and *C*). Each symbol represents the mean of 20 reflexes. \*Statistically significant post hoc Fisher test,  $P < 0.05$ .



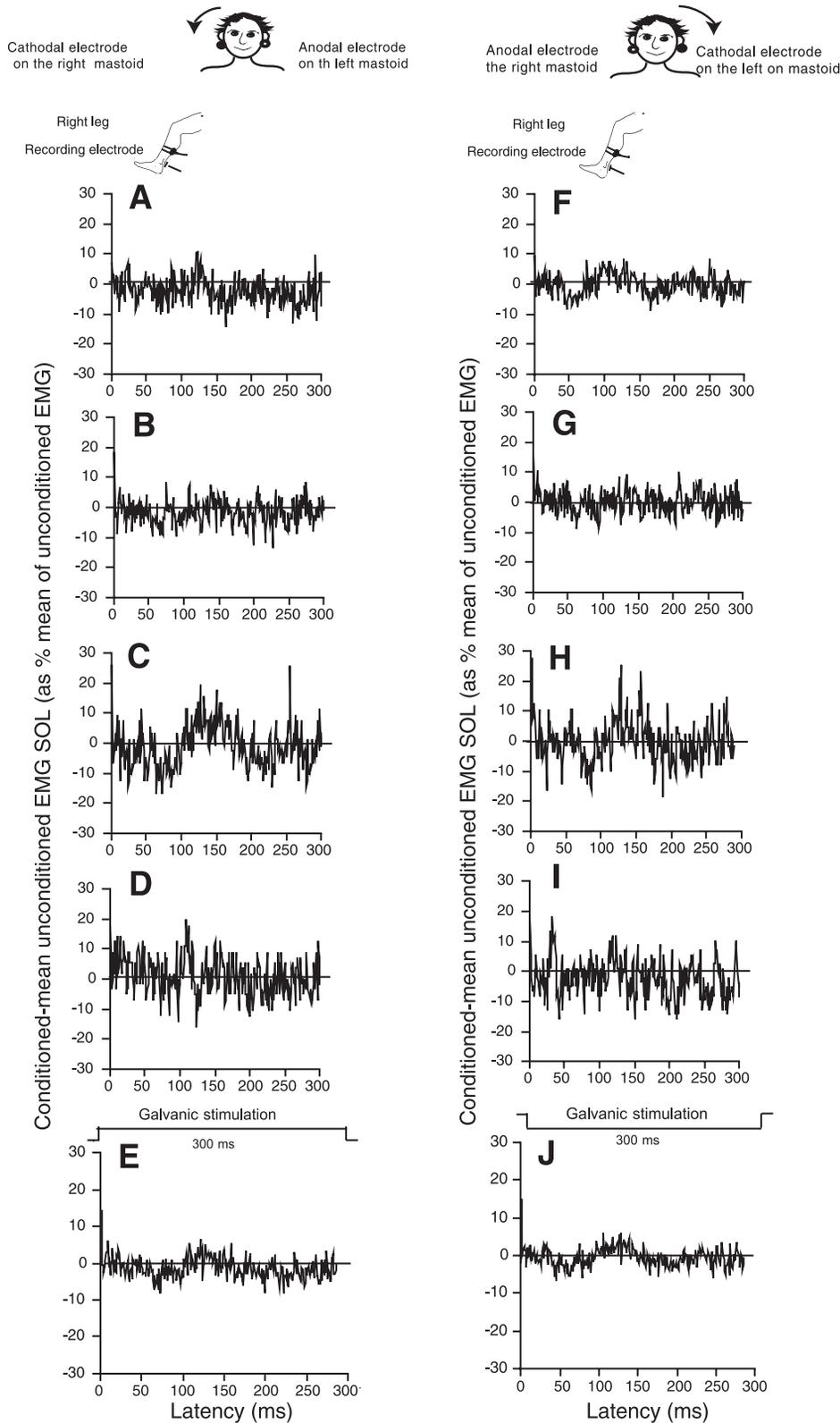


Fig. 6. Effects of bimastoid GS stimulation on rectified ongoing EMG soleus in the sitting position. *A, B, C, D, F, G, H, I*: individual data from 4 subjects. *E* and *J*: grand average of 7 experiments performed on 4 subjects. *A–E*: cathodal electrode on the right mastoid. (*F–J*) Anodal electrode on the right mastoid. Same coordinates as in Fig. 2, *B* and *E*.

muscle spindle afferent volleys activated by a spread of GS also seems possible, because the reflex facilitation following activation of both muscle spindles and cutaneous afferents by percussion of the SMM tendon (Fig. 5*F*) was more marked than that produced by a purely cutaneous stimulus.

*Possible Startle Response*

Stimulation of cutaneous afferents has been shown to produce a twofold facilitation of the monosynaptic reflex studied in distant pairs of antagonistic muscles (11). The

closer to the brain stem the stimulation (and/or the muscle tested), the earlier the latency of the facilitation. It has therefore been suggested that such a nonspecific activation of the motor nuclei in a rostrocaudal sequence reflects a startle response. This view is supported by the finding that the facilitation of the soleus H reflex after stimulation of the ophthalmic branch of the trigeminal nerve had the same latency as the facilitation produced by an auditory stimulus, attributable to a startle reaction (10). Indeed, both bursts in rectified ongoing soleus EMG and facilitation of the soleus H reflex have been described following an auditory stimulus (2, 12, 13, 29). Interestingly, Delwaide and Schepens (13), in their study of auditory startle reaction in lower limb muscles, have compared the latency of the facilitation of the soleus H reflex (~50 ms) and that of the EMG bursts (~100 ms). They concluded that taking into account the time for impulses to reach the spinal cord, the latency of the reflex facilitation corresponds to that of the EMG bursts. Therefore, because the GS-induced reflex facilitation while sitting occurs at the same 50 ms latency and is sensitive to habituation (like startle responses, see Ref. 25), we favor the hypothesis of a startle reaction mediated through a reticulospinal pathway. It has been shown in animals that trigeminal (tactile), auditory, and vestibular inputs can summate to reinforce startle responses (for review, see Ref. 30). Summation of GS-induced vestibular and cutaneous afferent volleys could thus account for the findings that, while sitting, the GS-induced reflex facilitation was stronger (Fig. 5B) than that evoked by purely cutaneous volleys (Fig. 5D) and that cutaneous anesthesia did not completely suppress the reflex facilitation (Fig. 5B). Similarly, the finding that the reflex facilitation was smaller after a purely cutaneous stimulus than after a tap to the SMM tendon, which activated both cutaneous and muscle spindle afferents, could be explained by a convergence of neck muscle afferents on reticulospinal neurones (19).

#### Target Spinal Circuit

It has often been implicitly accepted that a stronger facilitation of the T than of the H reflex, as observed in the present investigation, reflects an excitation of  $\gamma$ -motoneurons. However, given the conduction time through the  $\gamma$ -loop, a facilitation of  $\gamma$ -motoneurons by the conditioning stimulus should delay the facilitation of the tendon reflex by ~40 ms with respect to that of the H reflex. This does not fit the finding that facilitation of the two reflexes, H or T, occurred at approximately the same latency (Fig. 3C and Fig. 4A). Neither can the stronger facilitation of the T reflex be attributed, as proposed by Delwaide and Delbecq (12), to a decreased presynaptic inhibition of Ia terminals, because the afferent volley of the H reflex has been shown to be more sensitive to changes in presynaptic inhibition than that producing the tendon jerk (22). It has been suggested that Ib afferents curtail the electrically evoked Ia excitatory postsynaptic potential and that the H reflex can be altered by altering transmission across the Ib inhibitory interneurons, a situation not equally applicable to the tendon jerk (4, 20). Interestingly, an excitatory projection of reticulospinal tract fibres onto Ib interneurons has recently been described in the cat (5). This is in keeping with the

possible reticulospinal origin (see Ref. 30) of the startle response postulated above.

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