Contact-Evoked Changes in EMG Activity During Human Grasp

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Collins, D. F., B. Knight, and A. Prochazka. Contact-evoked changes in EMG activity during human grasp. *J. Neurophysiol.* 81: 2215–2225, 1999. 2215 Cutaneous receptors in the digits discharge bursts of activity on contact with an object during human grasp. In this study, we investigated the contribution of this sensory activity to the responses of muscles involved in the task. Twelve subjects performed a standardized precision grasp task without the aid of vision. Electromyographic (EMG) responses in trials when the object was present were compared with those in which the object, and hence the associated afferent responses, were unexpectedly absent. Significant differences in EMG amplitude occurred in the interval 50–100 ms after contact in all subjects and in 33/46 of the muscles sampled. The differences emerged as early as 34 ms after contact and comprised as much as a fourfold change in EMG from 50 to 100 ms after contact with the object. Typically, EMG responses were larger when the object was present (OP), though there were cases, particularly in the thenar muscles, in which the responses increased when the object was absent (OA). Local anesthesia of the thumb and index finger attenuated contact-evoked EMG activity in at least one muscle in all four subjects tested. In one subject, contact-evoked responses were abolished completely during the anesthesia in all four muscles sampled. The results indicate that the sensory activity signaling contact plays a key role in regulating EMG activity during human grasp. Much of this feedback action is attributable to cutaneous receptors in the digits and probably involves both spinal and supraspinal pathways.

INTRODUCTION

The neural control of hand movements has received increasing attention in recent years, in particular the role of sensory feedback in shaping motor patterns. Human microneurography has provided much information on the nature of the feedback signals from muscle and skin receptors of the hand (Al-Falahe et al. 1990; Burke et al. 1988; Edin and Abbs 1991; Hulliger et al. 1979; Johansson and Westling 1991; Vallbo et al. 1979). However, the role of these signals in controlling the onset of grasp remains to be clarified. It has been shown in numerous experiments that sensory feedback is critical in adapting grip forces to sudden slips of an object held between the index finger and thumb (Johansson and Westling 1984, 1987; Johansson et al. 1992) and while lifting objects with different weights and frictional characteristics (Johansson and Westling 1984). In the present study, we investigated the contribution of the afferent signals evoked by contact with the grasped object to the modulation of electromyographic (EMG) activity controlling the grasp.

It is well known that immediately after the digits contact an object during human grasp, grip forces (normal to the object surface) develop in parallel with load forces (tangential to object surface) until sufficient force is developed to lift the object (Westling and Johansson 1984). Skin receptors in the digits signal contact (Johansson and Westling 1991; Westling and Johansson 1987) and blocking these signals by local anesthesia delays the development of appropriate lifting forces (Johansson and Westling 1984). Although there are no corresponding human data on muscle afferents, muscle spindles in the cat are known to respond sensitively to ground contact in gait (Prochazka and Gorassini 1998).

A significant portion of the EMG in early stance arises from afferent input evoked by foot contact (Gorassini et al. 1994). This was shown by comparing EMG activity in normal step cycles with those when ground support and thus sensory activity signaling foot contact were absent. In the present study, we used a similar approach to study the role of the sensory contact signal during human grasp. Subjects were instructed to grasp, lift, and replace an object without the aid of vision. Mean EMG activity from these trials was compared with activity from trials in which the object and therefore the contact signal were unexpectedly absent. We posited that the afferent barrage evoked by contact with the object would initiate increases in EMG activity in the muscles involved in the grasping task. Preliminary data were published in abstract form (Collins and Prochazka 1996a).

METHODS

Twelve informed volunteers 9 male, 3 female, aged 22–51, with no history of neurological or skeletomotor disease, participated in the experiments, which were conducted in accordance with the declaration of Helsinki and the approval of the University of Alberta Hospitals Ethical Committee. Eight of the subjects were naive to the research hypothesis and experimental protocol. Two subjects participated in two experimental sessions.

Experimental protocol

Ten subjects participated in the initial experiments. During all trials subjects were seated comfortably at a table and were blindfolded or seated behind a screen to prevent vision of the test object. All movements were made with the right hand. Before each trial, the right arm and hand rested on the table with index finger and thumb extended in a standard posture determined by adjustable guideposts on either side of the object (see Fig. 1*A*). The guidepost positions were adjusted for the comfort of each subject at the beginning of each session. Subjects were instructed to grasp the object between thumb and index finger using a pinch (precision) grip, lift it to a height of ~5 cm, replace it back on the table, and return the fingers to the starting position (object present trials: OP). All movements were self-paced. Before data collection, subjects were allowed sufficient practice to become familiar with the grasping task. Rest periods were incorpo-

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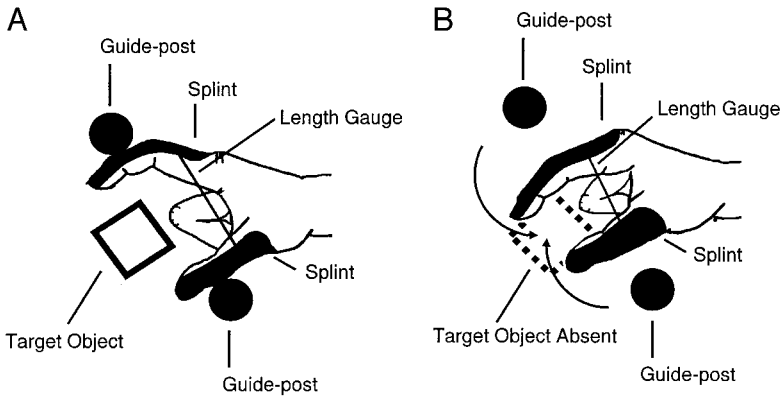

FIG. 1. Diagram of the experimental paradigm. *A*: standardized starting position of the hand. Before each trial the digits were extended to adjustable guideposts. *B*: example of a trial in which the target object was unexpectedly absent (object absent: OA).

rated to avoid fatigue. Each session comprised one to three blocks of 40–64 trials. After most trials, the experimenter replaced the object to the exact starting position. Randomly interspersed throughout each block were trials (20–33%) in which the object was not replaced, i.e., it was unexpectedly absent when the subject attempted to grasp it (object absent trials: OA, see Fig. 1*B*). To avoid prediction, the experimenter exaggerated the activity of replacing the object and made similar actions and noises when the object was not replaced. All subjects confirmed that they could not predict whether the object would be present or not. Mean EMG activity in OP trials, in which we assume sensory contact signals were present, was compared with mean activity in OA trials where these signals were presumably absent.

For the first five subjects the test object was a can (5 cm diameter, 750 g); for subsequent subjects, it was a rectangular block of stainless steel (3.8 × 3.8 × 12 cm high, 500 g). Two thin, thermally molded splints were form-fitted to the dorsal aspect of the right index finger and thumb to reduce movements at the interphalangeal joints (see Fig. 1). Grip aperture was monitored with a length gauge mounted between the metacarpophalangeal and proximal interphalangeal joints on the index-finger and thumb splints (see Fig. 1). This gauge was either a mercury-in-rubber length gauge or a strain gauge attached to a thin silastic tube (1-mm diam).

Detecting contact

The moment of digit contact with the object was monitored in two ways. In the first five subjects, thin strips of flexible, self-adhesive, conductive material (~5 × 40 mm) were wrapped around the distal portion of the index finger and thumb that first made contact with the metal object. On contact, each digit closed a separate battery circuit and the resulting signals were recorded. In subsequent subjects, two accelerometers (Analogue Electronics, 5 *g* range) mounted on the dorsal aspect of the distal part of each splint replaced the conductive strips. The accelerometer signals were band-pass filtered (0.1–30 Hz).

The contact signals were analyzed off-line to determine the time of first contact in OP trials. The data then were realigned such that first contact was at *time 0*. To realign OA traces to the moment that contact would have occurred had the object been present, a trigger pulse was obtained from the grip aperture signal that corresponded to close proximity to the object. The mean delay from this trigger pulse to first contact in OP trials was calculated for each subject and used to align OA trials in the same subject. The mean error in estimating the ''real'' time of contact from the aperture signal in OP trials was 2.9 ms ± 0.6 (SD) ms, and we assume that a similar error applied in OA trials. This error is small in relation to the time resolution of latency measurements.

In the first nine subjects, the duration of the lift of the object from the table was also recorded. The moments of lift-off and touch-down of the object were obtained by monitoring electrical contact between metal surfaces on the object and the table.

For each trial, data were stored ≥150 ms before (to a maximum of 1 s) and 250 ms after (maximum 3 s) this trigger signal. All data were digitized at 500 Hz (Cambridge Electronic Design 1401 A/D interface using Sigavg 6.0 software) and stored on a personal computer.

EMG recording

Surface EMG activity was recorded using self-adhesive, silver/silver-chloride electrodes (2.2 × 3.4 cm, Jason Electrotrace). Pairs of electrodes were placed over the bellies of four of the following muscle groups; first dorsal interosseus (FDI), flexor pollicis brevis/abductor pollicis brevis (thenar), flexor carpi radialis (FCR), flexor digitorum (FD), extensor carpi radialis (ECR), or extensor carpi ulnaris (ECU). For the intrinsic muscles (FDI and thenar), the electrodes were trimmed to ~1.5 cm in diameter. EMG signals were amplified 1,000–3,000 times, high-pass filtered (10 Hz), full-wave rectified, low-pass filtered (300 Hz), and digitized at 500 Hz (see following text).

Digital anesthesia

After initial experiments in 10 subjects, the experimental protocol was repeated before and during digital anesthesia of the right index finger and thumb. Four subjects participated in these experiments, two of whom had participated in the initial experiments. Four blocks of grasping trials were collected (64 trials/block, 25% OA condition), two before anesthesia and two during the anesthesia. After the first two blocks of trials, carbonated lidocaine hydrochloride (Xylocaine, Astra Pharma, product 173) was injected transcutaneously immediately distal to the metacarpophalangeal joints of the index finger and thumb by an anesthesiologist. Anesthesia was assessed in three ways: subjective reports during data collection, standardized tactile perception tests using Semmes-Weinstein monofilaments (Bell-Krotoski and Tomancik 1987), and comparison of the amplitude of electrically evoked cutaneous reflexes (see following text). Before anesthesia, cutaneous sensibility in all subjects was in the normal range as assessed by monofilament testing (subjects could perceive readily the force applied with the 2.83 monofilament, ~0. 8 mN). The extent of anesthesia was considered sufficient when subjects could not detect palpation of the digits by the experimenter and only the largest of the Semmes-Weinstein monofilaments (6.65 monofilament, ~2.8 N force) could be detected. Subjects often could not detect this monofilament as it indented the skin but could do so as it was removed. If this extent of anesthesia was not achieved 20–30 min postinjection, additional Xylocaine was administered (total amount 2–4 ml/digit). In two subjects, the anesthesia remained complete for the duration of the experiment. The other two subjects (*S10B* and *S11* in Table 2) reported the return of some cutaneous sensibility during the second block of postinjection trials. Monofilament and reflex tests confirmed this. For these subjects, only data from the first block of postinjection trials were included for analysis.

Cutaneous reflexes were evoked by electrical stimulation (3–5

pulses, 300 Hz) of the glabrous skin at the tip of the right index finger and thumb at three times perceptual threshold. Three blocks of 100 trials were collected while subjects maintained a moderate pinch grip force. The first block preceded anesthesia, the second and third occurred during anesthesia, just before and just after the two blocks of grasp trials respectively.

Reaction time

The minimal voluntary reaction time to an innocuous electrical stimulus applied to the skin of the contralateral index fingertip was measured in eight subjects. A warning stimulus (1-ms current pulse, 1.4 times perceptual threshold) was followed ~1 s later by an identical stimulus to which subjects were instructed to respond as quickly as possible with a precision-grip movement. Contralateral stimulation was used to avoid simple reflex responses to the stimulation. Twenty to 40 trials were collected for each subject. To minimize subject anticipation, no response signal was presented in 20% of the trials. Background EMG levels in FDI were computed over the 100 ms preceding the GO stimulus. Reaction time was defined as the time at which FDI EMG exceeded background by 2 SD for ≥25 ms.

Statistical analysis

The latency from *time 0* (first contact with the object in OP trials) at which the mean EMG activity in OP trials became significantly different from that in OA trials was identified for each subject as follows. EMG signals from OP and OA trials were separately averaged over 8-ms bins from *time 0* to 98 ms. Student's *t*-tests (or Mann-Whitney *U* tests when data were not normally distributed) were used to detect statistically significant differences between corresponding OP and OA bins. The magnitude of the contact-evoked responses was expressed as the OP/OA ratio. This was calculated by dividing the mean EMG in the interval 50–100 ms after contact in OP trials by the corresponding EMG in OA trials. Student's *t*-tests (or Mann-Whitney *U* tests when data were not normally distributed) were used to detect statistically significant differences in mean EMG activity between OP and OA trials over this interval. Data were normalized to the corresponding mean over the 100 ms interval prior to contact in the OP trials. All descriptive statistics are given as the mean ± 1 SE except where indicated. For all tests statistical significance was accepted when $P < 0.05$.

RESULTS

General movement characteristics

Subjects grasped, lifted, and replaced the test object at their own pace. Mean EMG activity and grip aperture in *subject S7* during 93 OP trials are shown in Fig. 2, thick lines (see also Table 1). The dashed line in this and all subsequent figures represents the time of first contact of one or other digit with the object (see METHODS). For this subject, the whole task (from movement onset to return of the digits to the approximate starting position) took 2.0 ± 0.1 s. Across the first nine subjects, this duration averaged 2.0 ± 0.2 s. Sweep durations were <2 s in the remaining three subjects. The task was divided into four temporal phases. *1*) Movement onset to first digit contact with the object (vertical line in figures) averaged 153 ± 20 ms across the nine subjects. The thumb and index finger usually contacted the object asynchronously, the mean difference in contact times being ~20 ms. *2*) First contact to object lift-off averaged 269 ± 45 ms. To check whether the experimental constraints affected the kinematics of the task, we separately filmed five unencumbered subjects who were instructed to grasp and lift the rectangular test object at their own pace. Frame-by-frame analysis showed that movement onset to first digit contact was 230 ± 55 ms and first contact to object lift-off was 365 ± 150 ms, indicating that the experimental data were representative of unconstrained grasp and lift. *3*) The duration from lift-off to replacement of the object on the table averaged 1.0 ± 0.1 s. *4*) Time from replacement of the object to return of the digits to the starting position averaged 0.6 ± 0.1 s.

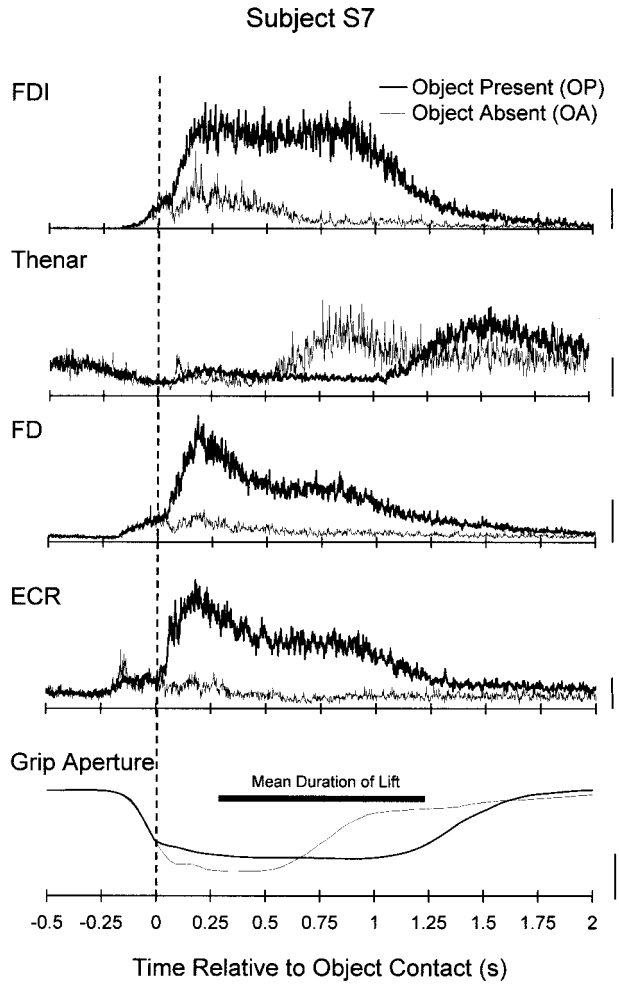

FIG. 2. Mean rectified electromyogram (EMG) in 4 muscles: *subject S7* comparing object-present trials (OP, $n = 93$) with OA trials ($n = 33$). Moment of 1st contact with the object in OP trials is shown by the vertical dashed line. Horizontal solid line over the grip aperture trace shows the average length of time the target object was lifted off the table. Calibration bars = 50 μV for EMG data and 2 cm for grip aperture.

Mean data from 33 OA trials for *subject S7* are shown by the thin lines in Fig. 2. The data were aligned to *time 0* as defined in METHODS. In most trials in which the target was absent the digits continued moving, though often decelerating, until they touched each other ~75 ms after contact would have been made. In three subjects, in ~20% of OA trials, the digits rapidly reextended, then flexed again ~100 ms after contact would have occurred as though in search of the object.

Reaction time

The minimal latency for a volitional response in FDI to electrical stimulation of the contralateral fingertip was inves-

TABLE 1. *Summary of contact-evoked changes in EMG activity for individual subjects*

	FDI		Thenar		Finger Flexion		Wrist Extension	
Subject	Latency, ms	OP/OA	Latency, ms	OP/OA	Latency, ms	OP/OA	Latency, ms	OP/OA
S1	44	4.0†	NS	0.8 NS			74	1.4 NS
S2	34	2.1†	74	0.6*	44	1.5†	34	1.7†
S3	34	1.4 NS			44	1.9*	44	1.5*
S4	84	1.0 NS	64	0.4†	74	1.2*	54	1.9†
S5	NS	1.2 NS	44	1.8*	84	0.9*	64	1.4*
S6a	34	1.5†	84	0.8†	34	1.3†	54	2.6†
S7	54	2.4†	54	0.8 NS	44	3.4†	44	3.6†
S8	54	0.8*	NS	0.9 NS			NS	1.2 NS
S9	64	0.6*	NS	1.2 NS	74	1.3*	34	1.2 NS
S10a	74	2.0†			54	2.8†	54	2.5†
S11	34	2.1†	74	0.8 NS	54	2.3†	54	2.2†
S12	74	0.6†	74	0.7†	54	1.1 NS	44	1.7†
Mean	54	1.6	67	0.9	56	1.8	50	1.9

Latencies refer to the center of the bin in which mean electromyographic (EMG) activity during object present (OP) trials became significantly different than that in corresponding object absent (OA) trials. Magnitudes are expressed as OP/OA ratios, i.e., mean EMG from 50 to 100 ms after *time 0* in OP trials divided by that in OA trials. Statistical significance: * $P < 0.05$, † $P < 0.0001$, NS, no significant difference.

tigated in eight subjects, 20 trials per subject. Catch trials (warning but no stimulus) were included to detect anticipation. Mean reaction time across all trials was 190 ± 14 ms (range 136–236). Mean minimal reaction time, calculated from the fastest reaction obtained in each subject was 113 ± 5 ms (range 92–140). From this we conclude that any responses inferred from OP-OA differences in the range 50–100 ms after contact are too early to be entirely voluntary and by exclusion are therefore largely if not completely reflexive.

Peripheral afferent contributions to EMG activity

The main focus of this study was to investigate the contribution of sensory input to EMG responses in human grasp. Differences in EMG activity in OP and OA trials were attributed to the presence or absence of contact-evoked sensory signals, as illustrated for *subject S7* in Fig. 2. These data are replotted in Fig. 3 on an expanded time scale. Mean raw data and mean binned data are shown on the left and right sides, respectively. Note that the binned data were calculated for the 0- to 98-ms interval from the corresponding data on the left side (i.e., data between the vertical dashed lines). The left side of Fig. 3 shows clear differences in EMG in OP versus OA trials within 100 ms after *time 0*. Student's *t*-test comparisons of corresponding bins indicated that these discrepancies became significantly different 54, 54, 44, and 44 ms after contact for FDI, thenar, FD, and ECR, respectively. Table 1 summarizes the results of statistical tests for each subject. Subjects are listed in the order in which they participated in the experiments and are referred to throughout this paper by the subject code as indicated in Tables 1 and 2. Summarized are the latencies and magnitudes of the discrepancies in EMG activity between OP and OA trials as defined in METHODS. Within 100 ms of *time 0*, significant differences were found in at least one bin in all subjects and in 39 of the 46 muscles sampled (see OP/OA columns, Table 1, FCR data not shown). The shortest latency at which these differences appeared was 34 ms after contact, which was seen in seven muscles (4 subjects). The OP/OA ratios were calculated from the mean EMG activity levels over the interval 50–100 ms after *time 0*, which we argue above encompasses a period during which responses are too early to be voluntary. More EMG activity was recorded in OP trials than in OA trials over this interval in 33 of the 46 muscles sampled (OP/OA ratio >1). Statistical significance was reached in 24 of these cases. In 12 of the 46 muscles sampled, the EMG responses in OP trials were smaller than those in OA trials (OP/OA ratio <1). This was significant in eight cases. Overall, the OP/OA ratio ranged from 0.4 (*S4*, thenar) to 4.0 (*S1*, FDI).

Ensemble data averaged across all 12 subjects are shown in Fig. 4. As in Fig. 3, the *left side* shows data in the interval 100 ms before to 200 ms after *time 0* and the *right side* shows the same set of data averaged into 8-ms bins over the interval 0–98 ms after *time 0*. The EMG profiles in OP and OA trials began diverging between 40 and 60 ms after *time 0* in all four muscle groups. However, statistical significance in individual bins was not reached until the 50–58 ms bin in FDI and thenar muscles (asterisks). This reflects the large intersubject variability observed in these muscles. In general, OP trials showed more activity than OA trials beginning ~44–54 ms after contact and lasting throughout the data collection period. However, there were exceptions, as shown in the detailed description of each muscle group that follows.

FDI. In 11/12 subjects, there were significant OP-OA EMG differences between pairs of bins in the first 100 ms (Table 1). The mean latency of such differences was 54 ± 6 ms (range 34–84 ms; note that a latency of 34 ms was seen in 4 subjects). Nine of the subjects showed significant OP-OA differences when this was tested over the period 50–100 ms (Table 1). The OP/OA ratio over this interval ranged from 0.6 to 4 (mean 1.6). In six subjects the ratio exceeded unity (e.g., *subject S7* shown in Figs. 2 and 3, *top*), and in three subjects the ratio was less than unity, i.e., there was significantly less FDI activity in OP than in OA trials. In *subjects S9* and *S12*, the reduction was quite large (OP/OA ratio = 0.6). These reductions were consistent with the EMG response in FDI evoked by electrical stimulation of the digits during static grasp (Fig. 5).

THENAR MUSCLES. In 7/10 subjects, there were significant OP-OA EMG differences in individual bins in the first 100 ms. The mean latency of such bins was 63 ± 5 ms (range 44–84 ms). Five of the subjects showed significant OP-OA differ-

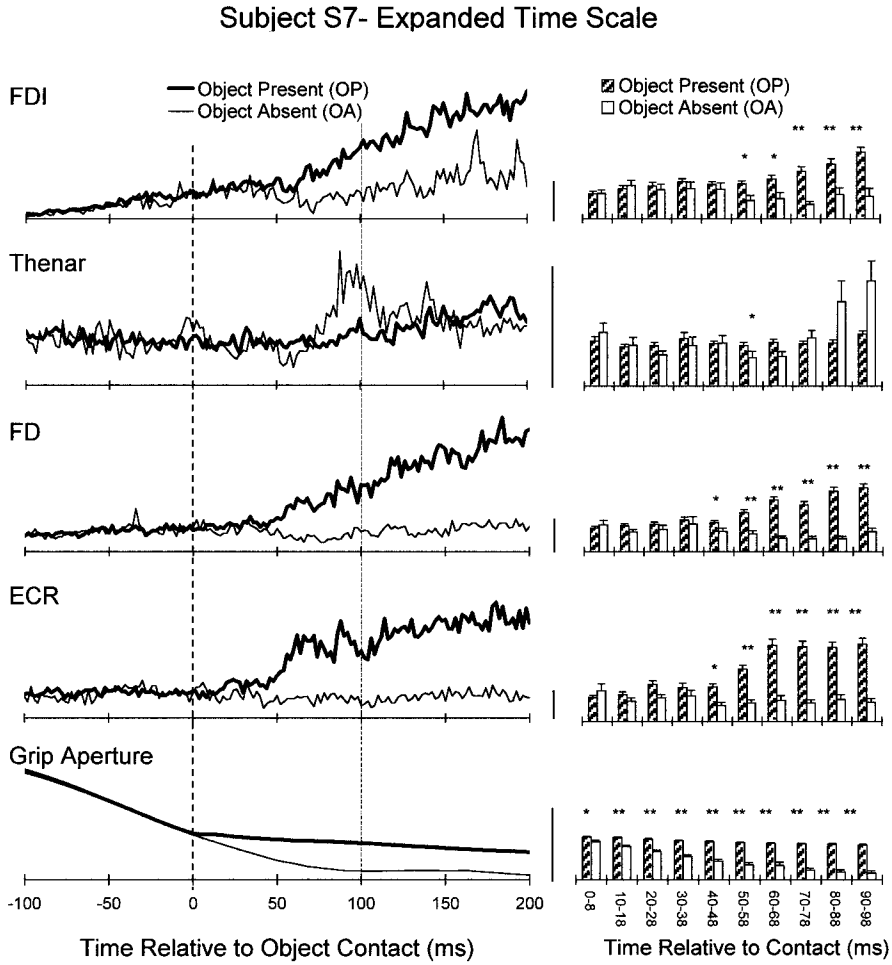

FIG. 3. Portion of the data in Fig. 2 shown on an expanded time scale. *Left*: mean rectified EMG shown from 100 ms before to 200 ms after 1st contact (*time 0*: thick vertical dashed line). *Right*: mean EMG data in 8-ms bins from corresponding data over the interval between the 2 vertical dashed lines in left of figure (0–98 ms). Calibration bars = 25 μV for EMG data and 2 cm for grip aperture. Statistical significance: * $P < 0.05$, ** $P < 0.0001$.

ences when this was tested over the period 50–100 ms, and in four cases, this represented a reduction of EMG in OP trials. An individual example is shown in the left of Fig. 7 (*subject S10B*: OP/OA ratio 0.7; Table 1; in *subject S5,* the OP/OA = 1.8). In five subjects, there were no significant OP-OA differences in thenar activity over the 50- to 100-ms interval. The mean OP/OA ratio across all subjects was 0.9. We argue later that the thenar responses are consistent their functional role in the task.

FCR. Data from FCR were collected in two subjects. One showed a significant OP-OA difference 64 ms after *time 0* in binned data, but the OP/OA ratio computed over the 50- to 100-ms interval was 1.1 in both subjects and the difference was not statistically significant.

FINGER FLEXORS. Data were recorded from FD (8 subjects) and FPL (2 subjects). All 10 subjects showed significant OP-OA EMG differences in individual bins in the first 100 ms, the mean latency of which was 56 ± 5 ms (range 44–84 ms: Table 1). 8/10 subjects showed significantly more OP than OA activity over the period 50–100 ms (OP/OA 1.2–3.4), and 1 subject showed significantly less (OP/OA = 0.9). Examples of significant responses from individual subjects are shown in Figs. 3 and 7 (FD). Mean OP/OA ratio across all subjects was 1.8.

WRIST EXTENSORS. Eleven of 12 subjects showed significant OP-OA EMG differences in individual bins in the first 100 ms. The mean latency of such bins was 54 ± 4 ms (range 34–74 ms). All subjects showed more OP than OA activity over the period 50–100 ms, and this reached significance in nine cases (OP/OA range 1.4–3.6). Individual responses are shown in Figs. 3 and 7. Mean OP/OA ratio across all subjects was 1.9.

Digital anesthesia

The OP-OA experiments were repeated before and during anesthesia of the index finger and thumb in four subjects. This procedure eliminated all but a slight cutaneous sensibility in the affected digits (see METHODS). Responses evoked by electrical stimulation of the index finger and thumb at three times perceptual threshold, averaged across all subjects ($n = 4$), are shown in Fig. 5. These data were recorded before the anesthesia and also during anesthesia immediately before the first block of postinjection grasp trials. The anesthesia abolished cutaneous reflex responses in all subjects. In two subjects (*S10B* and *S11*) some cutaneous sensibility returned during the second block of postinjection trials (see METHODS). These data are not detailed here; however, the EMG responses were intermediate between those in control and fully anesthetized trials.

Digital anesthesia impaired all subjects' performance of the task. In all sessions the object occasionally slipped or dropped to the table, often without the subject being immediately aware of it. The number of slips and drops declined as data collection

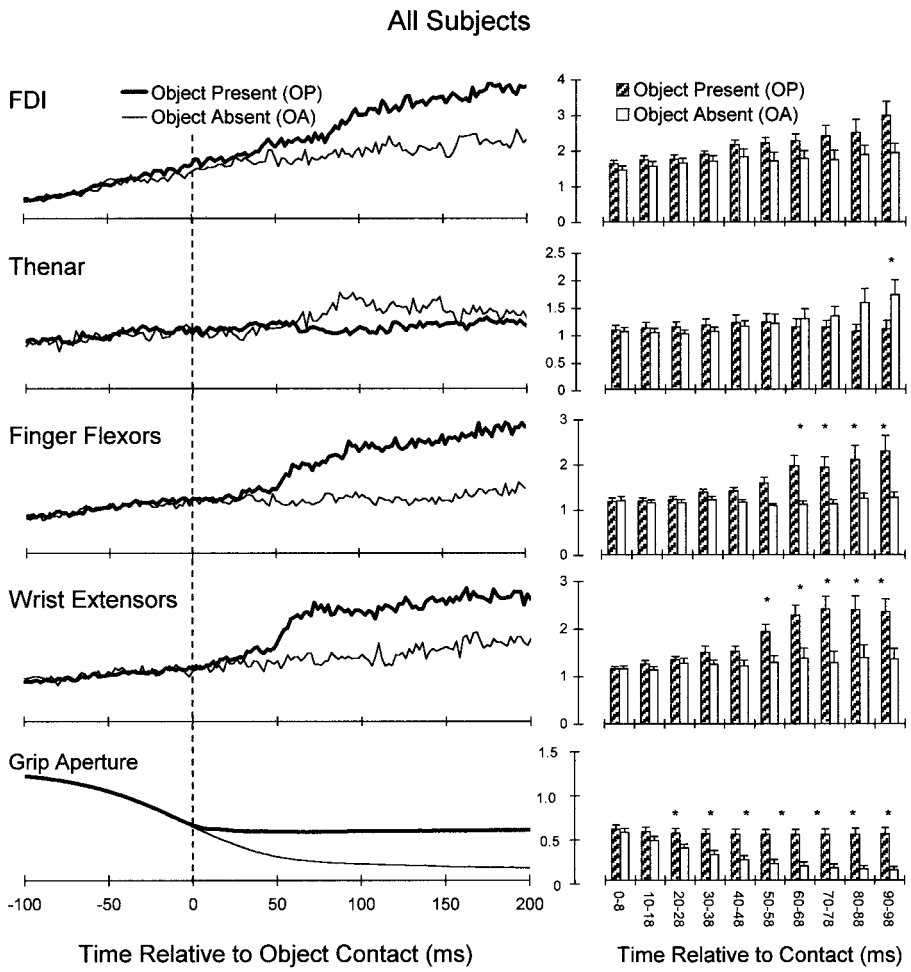

FIG. 4. Mean data across all 12 subjects. Data for each subject were normalized to the corresponding mean during the 100 ms before contact in the OP trials. *Left*: mean rectified EMG data from 100 ms before to 200 ms after *time 0*. *Right*: mean EMG in 8-ms bins from 0 to 98 ms. Statistical significance: * $P < 0.05$.

proceeded. In general, EMG amplitudes during OP trials over the whole grasp movement were similar in pre- and postinjection trials. The fact that the object slipped in such cases may have been due to reduced sweating as a result of autonomic nerve blockade. In some cases there was more EMG activity during digital anesthesia, suggesting an adaptive strategy.

Mean data across the four subjects, before and during digital anesthesia, are shown on the *left* and *right* sides of Fig. 6, respectively. Anesthesia reduced mean OP-OA differences in FD and ECR but surprisingly caused slight increases in these differences in FDI and the thenar muscles. Table 2 summarizes the effects for individual subjects. With normal sensibility significant OP/OA differences were found over the interval 50–100 ms after *time 0* in 12/16 muscles sampled. Anesthesia abolished or reduced these differences in nine of these muscles. Across all four subjects, OP/OA ratios in the intervals 50–75 ms and 76–100 ms after *time 0* were compared before and during anesthesia (Fig. 6). Significance at the 95% level was reached in FDI and FD but not in thenar and ECR muscles.

The effect of digital anesthesia on EMG activity is shown for *subject S10B* in Fig. 7. Anesthesia completely abolished all significant OP-OA differences over the 50- to 100-ms interval (see also Table 2). It is therefore interesting that during anesthesia this subject had relatively few slips or drops of the object compared with the other subjects. In the other subjects, significant OP/OA differences were present during anesthesia in the 50- to 100-ms interval in 9/12 muscles. In two cases (*S6B*, FDI and thenar), these differences emerged in muscles that showed no significant differences with normal sensibility. With some exceptions, the OP-OA differences during anesthesia were qualitatively similar to, though smaller than, those seen with normal sensibility. Details of the effects of anesthesia on individual muscle groups follow.

FDI. Mean FDI responses across the four subjects before and during digital anesthesia are shown in Fig. 6, *top*. These data suggest a lack of any OP-OA difference with normal sensibility and less OP than OA activity from 60 to 150 ms during anesthesia. However, before anesthesia, *subjects S10B* and *S11* had OP/OA ratios of 2.0 and 2.1, respectively, whereas in *subject S12*, OP/OA = 0.6 (Table 2). This explains why the preanesthesia FDI profiles in Fig. 6 do not show the clear increase in OP trials seen in Fig. 4 (all 12 subjects). Across all four subjects the OP/OA ratio was 1.4 before anesthesia and 0.8 during anesthesia. To test whether the change was statistically significant, in each subject, before and during digital anesthesia, we computed the OP/OA EMG ratios over 50- to 75- and 76- to 100-ms intervals after *time 0* on the assumption that responses in these intervals are independent. This showed that the reduction in OP/OA ratio in FDI during anesthesia was significant (Student's *t*-test, $n = 8$, $P = 0.04$).

THENAR. Mean OP-OA differences in thenar EMG in the four subjects were not changed during anesthesia (Fig. 6). With normal sensibility *subjects S10B* and *S12* showed significantly

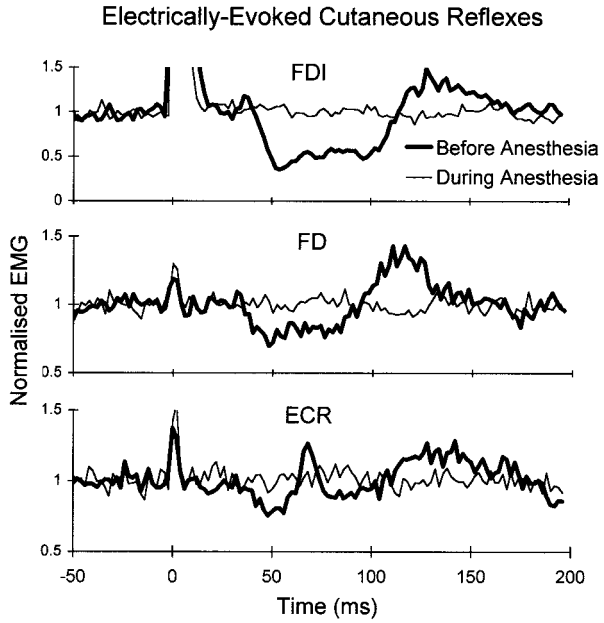

FIG. 5. Electrically evoked cutaneous reflexes before and during digital anesthesia. Data for each subject were normalized to the corresponding mean during the prestimulus 100 ms. Averaged responses in 3 muscles to stimuli delivered at *time 0* ($n = 100$). Deflections in first 10 ms are stimulus artifacts.

less thenar EMG activity during OP trials (Table 2). In *subject S10B*, this difference was reduced by anesthesia (Fig. 7), but in *subject S12*, it was augmented. One of the two subjects who showed no significant OP-OA difference before the anesthesia had significantly less OP activity during the anesthesia. Across all subjects the mean OP/OA ratio was 0.8 before anesthesia and 0.7 during anesthesia. The reduction, tested as in FDI, was not significant (Mann Whitney U test, $n = 8$, $P = 0.7$).

FD. Mean responses in FD across the four subjects show a large OP-OA difference before the anesthesia that was reduced markedly during anesthesia in all subjects (Fig. 6, Table 2). Across all subjects the OP/OA ratio was 1.8 before anesthesia and 1.3 during anesthesia, a statistically significant reduction (Student's t-test, $n = 8$, $P = 0.04$).

ECR. As in FD, ECR also showed evidence of large contact-evoked responses before the anesthesia that were reduced during anesthesia (Fig. 6). With normal sensibility, all subjects showed significantly more ECR activity in OP than in OA trials (see Table 2). Digital anesthesia reduced the OP/OA ratio in three subjects but increased it from 2.2 to 2.8 in *subject S11*. Across all subjects the OP/OA ratio was 2.0 before anesthesia and 1.7 during anesthesia, but because of the increase in *subject S11*, overall, the difference was not statistically significant (Student's t-test, $n = 8$, $P = 0.20$).

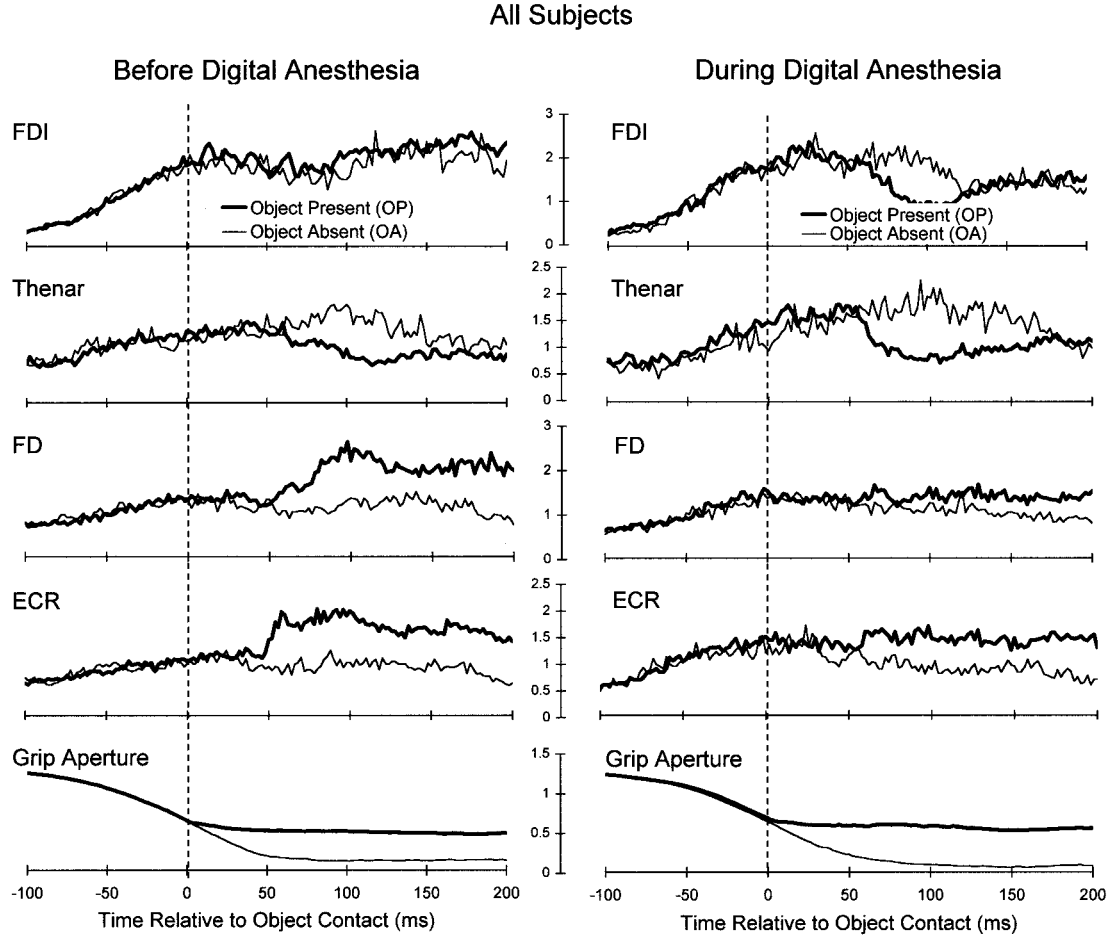

FIG. 6. Mean rectified EMG data across 4 subjects before anesthesia (*left*) and during anesthesia (*right*). Data for each subject were normalized to the mean activity during the 100 ms before contact in the OP trials.

TABLE 2. *Effect of digital anesthesia on contact-evoked changes in EMG*

	Muscle Group (OP/OA ratio)			
Subject	FDI	Thenar	FD	ECR
S10b				
Before	2.0*	0.7*	1.4*	2.4*
During	0.8 NS	0.8 NS	1.2 NS	1.2 NS
S6b				
Before	1.0 NS	0.9 NS	2.3*	1.6*
During	0.6*	0.6*	1.6*	1.4*
S11				
Before	2.1*	0.8 NS	2.3*	2.2*
During	1.1 NS	1.0 NS	1.3†	2.8*
S12				
Before	0.6*	0.7*	1.1 NS	1.7*
During	0.5*	0.4*	0.9 NS	1.3*
Mean				
Before	1.4	0.8	1.8	2.0
During	0.8	0.7	1.3	1.7

Magnitudes are expressed as OP/OA ratios, i.e., mean EMG from 50 to 100 ms after *time 0* in OP trials divided by that in OA trials. FDI, first dorsal interosseus; thenar, flexor pollicis brevis/abductor pollicis brevis; FD, flexor digitorum; ECR, extensor carpiradialis. Statistical significance: * $P < 0.0001$, † $P < 0.05$, NS, no significant difference.

DISCUSSION

In this study, we investigated how sensory feedback from the hand helps to shape motor output during human grasp. Specifically, we investigated how the burst of afferent activity known to be evoked when the fingertips contact an object (Johansson and Westling 1991; Westling and Johansson 1987) contributes to the EMG activity in muscles involved in the task. Data from trials in which subjects grasped an object were compared with trials in which the object and, therefore the contact-evoked sensory bursts, unexpectedly were absent. The results clearly show contact-evoked changes in EMG activity emerging shortly after contact (*time 0*, Figs. 2–4). In individual subjects, significant differences in OP and OA EMG profiles were apparent 34 ms after first contact of the index finger or thumb with the object in 7/46 muscles (see Table 1). These contact-evoked changes were often quite large. In one subject (*S1* in Table 1), four times more mean activity was recorded in FDI from 50–100 ms after *time 0* in OP trials than in corresponding OA trials (OP/OA ratio = 4, see Table 1). Responses in the intrinsic hand muscles (FDI and thenar) were variable across subjects: whereas 6/12 subjects had significantly more FDI EMG activity in OP than in OA trials, 3 subjects showed less such activity, contrary to our hypothesis. Reduced responses in the OP condition also were seen in the thenar muscles in 8/12 subjects, though the discrepancy was significant in only four cases. The inconsistency of responses in intrinsic muscles is discussed in *Functional implications*. Responses in the extrinsic muscles (finger flexors and wrist extensors) were more consistent across subjects. Significantly more EMG activity was recorded in OP than OA trials in 17/22 extrinsic muscles sampled. In only one case (*subject S5*, finger flexors) was significantly less activity recorded in OP trials.

Qualitatively, the contact-dependent components of EMG activity in the present study were similar to those described in experiments in cats in which extensor EMG in the load-bearing phase of the step cycle was compared with and without ground support (Gorassini et al. 1994). Along similar lines, Johansson and Westling (1988a) recorded EMG responses when a ball was dropped into a hand-held receptacle, causing it to displace between the subject's thumb and fingers. The responses were absent when the ball unexpectedly was prevented from landing in the receptacle.

Sensory receptors

The obvious candidates for receptors mediating the contact-evoked responses are cutaneous receptors in the digit tips. These receptors are situated ideally to signal the moment of contact with a grasped object (Johansson 1996), and microneurographic studies have shown characteristic changes in their firing rates on contact (Johansson and Westling 1991; Westling and Johansson 1987). Their role in the rapid adaptations to slips of grasped objects has been well documented (Johansson and Westling 1984, 1987; Johansson et al. 1992). It also has been shown that these receptors encode the frictional characteristics of the object surface (Johansson and Westling 1984; Westling and Johansson 1984) and that this information is used to adjust fingertip forces independently at the digits (Burstedt et al. 1997; Edin et al. 1992). Previous studies have shown that removal of this feedback by digital anesthesia often delays the development of appropriate lifting forces (Johansson and Westling 1984) and can change movement kinematics throughout the reaching and grasping trajectory (Gentilucci et al. 1997). Our results show that these receptors play an important role in initiating short-latency contact-evoked EMG responses. Removal of feedback from the digits by anesthesia completely abolished OP-OA differences in one subject (see Fig. 7) and reduced such differences in the remaining three subjects (5 of 8 muscles). These changes in contact-evoked EMG activity likely underlie the delay in the development of appropriate lifting forces during digital anesthesia seen previously (Johansson and Westling 1984).

However, digital anesthesia did not eliminate all contact-dependent EMG responses in the present study. Significant responses were present in nine muscles after most cutaneous feedback from sites distal to the metacarpophalangeal joints of the index finger and thumb was removed. This suggests that receptors other than cutaneous receptors in the digits also can play a role. In three cases, OP-OA differences were augmented during skin anesthesia. Furthermore the receptor populations which mediate contact-dependent responses may vary both between and within subjects. The removal of all contact-evoked responses by skin anesthesia in *subject S10B* (Fig. 7) suggests that this subject relied primarily on cutaneous feedback from the digits to control grip force. In contrast, other subjects showed contact-driven responses during the skin anesthesia that must have originated from other afferent sources. In *subject S11*, digital anesthesia abolished OP-OA differences in FDI but augmented them in ECU (see Table 2). This indicates that different receptor populations may mediate responses in different muscles. With the full complement of receptors to choose from, the nervous system preferentially may use signals from skin receptors in the digits. When this feedback is unavailable, the nervous system may switch to alternate afferent sources. These could include cutaneous receptors remote from the digits that are known to be active during finger movements and are involved in adaptations dur-

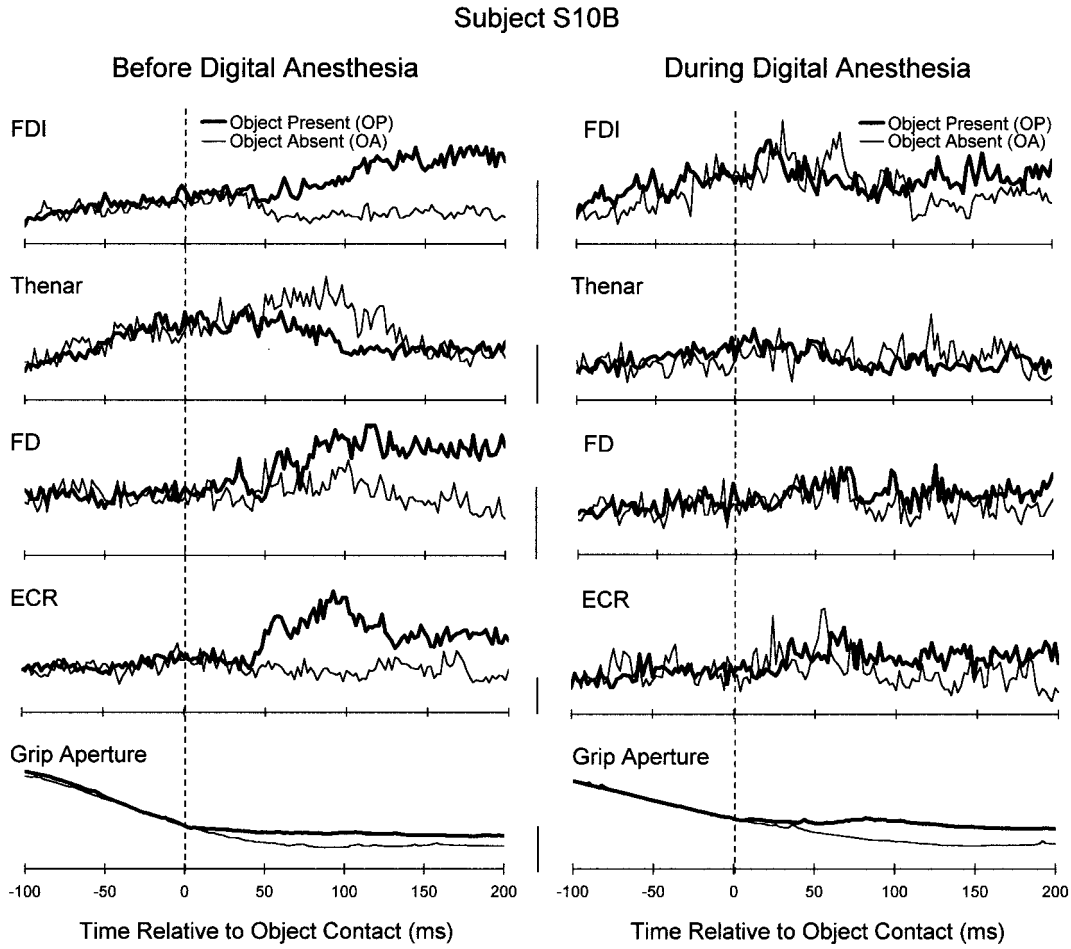

FIG. 7. Mean rectified EMG data from a single subject before (*left*) and after (*right*) digital anesthesia. Note the abolition of differences between OP and OA trials during anesthesia. Calibration bars = 25 μV for EMG data and 2 cm for grip aperture.

ing slips (Hager-Ross and Johansson 1996). Chronic recordings in cats (Prochazka and Gorassini 1998) suggest that muscle spindle receptors also may provide suitable contact-related signals. On the other hand, it seems unlikely that muscle forces would build up quickly enough after first contact to increase Golgi tendon organ firing rapidly enough to explain short-latency responses (particularly in light of the delay between thumb and finger contact). This detracts from the idea of Prochazka et al. (1997b) that positive force feedback mediated by tendon organs occurs during grasp, though a tendon organ contribution at longer latencies is not ruled out. Joint receptors probably play a minimal role as they are active primarily at the extreme ranges of joint rotation (Burgess and Clark 1969; Ferrell 1980, but note Edin 1990; Tracey 1979).

Neural pathways

In general, contact with the object generated changes in EMG activity starting 34–54 ms after *time 0* and persisting throughout the data collection period. There is some indication in Fig. 4 that the OP-OA divergence occurred in two stages, an initial divergence between 34 and 54 ms and a secondary divergence starting ~20 ms later. The mean delay between first and second digit contact was 20 ms, so the two stages may represent digit-specific responses (Burstedt et al. 1997).

The relevant sensory signals likely follow several routes through the nervous system. The EMG responses therefore probably reflect the summation of activity in all these pathways. In nine subjects (15/46 muscles), the leading edge of the changes occurred 34–44 ms after contact. Responses as rapid as these presumably are mediated segmentally. Longer latency components of the response in the range 50–70 ms likely involve ascending pathways, cerebellum and sensorimotor cortex (Jenner and Stephens 1982). Motor cortical excitability changes during reaching and grasping have been studied by several groups (Datta et al. 1989; Johansson et al. 1994; Lemon et al. 1996; Schieppati et al. 1996). Excitability was shown to increase during reaching in regions controlling extrinsic muscles and during grasping in regions controlling intrinsic hand muscles (Lemon et al. 1996). The high excitability in cortical regions controlling intrinsic muscles at the time of contact with the object ''may reflect a powerful interaction, at the cortical level, between cutaneous inputs signaling contact with the object and motor cortex excitability'' (Lemon et al. 1996). Similarly the interaction between cutaneous inputs from the hand and motoneuronal excitability is also somewhat task dependent (Evans et al. 1989). The extent to which continuing sensory input acting through segmental circuits contributes to EMG activity at medium and longer latencies is not clear.

The mean minimal voluntary reaction time we recorded in

FDI in response to pulsatile contralateral somatosensory stimulation of the digits during a single trial was 113 ms. Allowing 12 ms for interhemispheric transfer of motor commands (Schieppati et al. 1985), we infer that most EMG responses before 100 ms after contact are involuntary, although from our observed absolute minimum latency of 92 ms to the contralateral stimulus, voluntary contributions after 80 ms cannot be ruled out. Propriospinal mechanisms and sensory input to them have been shown to contribute to the control of reaching and grasping movements in cats (Alstermark and Lundberg 1992). This also may apply to human grasping (Pierrot-Deseilligny 1996). Abnormal grip forces seen in patients with disorders of the cerebellum (Muller and Dichgans 1994) or basal ganglia (Muller and Abbs 1990) suggest that these structures also may be involved.

Functional implications

Our results show that, on average, contact-dependent sensory signals initiated EMG responses that were functionally relevant to the task at hand. Typical responses included contact-driven enhancement of the activity in the prime movers (FDI, finger flexors). This would contribute to the build-up of pinch-grip forces required to lift the object from the table. Also all subjects showed a contact-evoked enhancement of wrist extensor activity that would help stabilize the wrist for the lift. The coactivation of muscles controlling the fingers and wrist during precision grip is thought to contribute to grasp stability (Werremeyer and Cole 1997). The most common response in the thenar muscles was less activity when the object was present. These muscles probably act as antagonists in our task, as evidenced by the inverse modulation of thenar and FDI EMG in Fig. 2. This relationship during precision grip was described previously for abductor pollicis brevis, one of the thenar muscles (Johansson and Westling 1988b). A contact-driven inhibition may serve to terminate activity in muscles that oppose the task. The variability of thenar responses we observed may reflect nonspecificity in the surface EMG recording from the three muscles of the thenar eminence, each of which performs a slightly different biomechanical function. Likewise the variability in FDI responses also may reflect their dual function as adductors and flexors of the index finger (Bremner et al. 1991). Figure 1 shows that at the moment of contact with the object, the index finger was partly flexed. After contact FDI no doubt contributes flexor pinch-grip force to prevent slippage, but depending on hand posture and motor strategy, it also may provide adductor force during the lift, to resist abduction caused by the weight and inertia of the object.

Our results highlight the importance of afferent signals in regulating phase transitions in movements: only when contact signals were present were successive phases of the motor program for the grasp executed (see Fig. 2). This is consistent with studies showing a delay in the onset of appropriate lifting forces while grasping during digital anesthesia (Johansson and Westling 1984). Also afferent signals evoked when a hand-held object contacts the table are important in terminating motor commands for grasp (Johansson and Westling 1988b). Similar afferent-controlled phase transitions are seen in the cat step cycle (Pearson and Collins 1993).

Our results may have relevance for the sensory control of grasp in active orthotic devices for people with spinal cord injury or stroke (Hoffer et al. 1996; Prochazka et al. 1997a). Such a device could use sensors on the digits to detect contact with objects to trigger stimulation of specific muscle groups to mimic the role of the contact signal in human grasp or other tasks. To avoid inappropriate force application, such a device may have to modulate the feedback gain according to the task. Indeed, there are many examples of task-dependent gain modulation of sensory pathways throughout the nervous system (Prochazka 1989) and grip forces are known to be adjusted according to the properties of the held object (Johansson and Westling 1984; Westling and Johansson 1984). Infant grasping tends to be indiscriminately strong, and it could well be that one of the important functions of motor learning is to develop appropriate task-dependent sensory gain control (Forssberg et al. 1995).

Our results underline the important role of cutaneous feedback, including segmental mechanisms, in controlling hand and finger movements. It long has been known that cutaneous receptors are crucially important in the control of hand movements (Mott and Sherrington 1895), and in recent years, there has been a resurgence of interest and research into the precise role of these receptors and their central actions (Collins and Prochazka 1996b; Edin and Johansson 1995; Gentilucci et al. 1997; Johansson 1996).

Summary

Our study showed that sensory input signaling first contact with a grasped object significantly modifies the subsequent activation of the hand muscles. The onset latencies of sensory-dependent EMG activity were mostly less than voluntary reaction time, suggesting mediation by reflexive mechanisms. Abolishing cutaneous sensory input from the fingertips changed and in some cases eliminated the contact-related components of EMG. This indicates that skin input plays a dominant role in the short-latency control of grasp onset, as previously shown for adaptations of grasp to load or load changes. Muscle afferent contributions to longer-latency components of response cannot be ruled out. The variation in contact-related EMG patterns we observed between muscles and also between subjects suggested that sensorimotor integration during grasp is highly task-dependent and also may vary from one individual to another.

We thank M. Gauthier and A. Denington for technical help.

This work supported by the Alberta Heritage Foundation for Medical Research (AHFMR) and the Canadian Medical Research Council (MRC). D. F. Collins was supported by AHFMR, MRC, and Izaac Walton Killam studentships.

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Received 23 June 1998; accepted in final form 21 January 1999.

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