

Disturbances of grip force behaviour in focal hand dystonia: evidence for a generalised impairment of sensory-motor integration?

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J Neurol Neurosurg Psychiatry 2005;76:953–959. doi: 10.1136/jnnp.2004.043943

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Received 21 April 2004
In revised form
9 October 2004
Accepted 11 October 2004

Background: Focal task specific dystonia occurs preferentially during performance of a specific task. There may be an inefficiently high grip force when doing manipulative tasks other than the trigger task, possibly reflecting a generalised impairment of sensory-motor integration.

Objective: To examine how well subjects with writer's cramp (n = 4) or musician's cramp (n = 5) adapted their grip force when lifting a new object or catching a weight.

Methods: Nine patients with focal hand dystonia and 10 controls were studied. Experiments addressed different motor behaviours: (A) lifting and holding an object; (B) adjusting grip force in anticipation of or in reaction to a change in load force by catching a small weight dropped expectedly or unexpectedly into a hand held receptacle.

Results: In (A), patients produced a grip force overshoot during the initial lifts; force overflow was most pronounced in those with writer's cramp. Patients and controls adjusted their grip force to object weight within one or two lifts, though patients settled to a steady force level above normal. In (B), patients with focal hand dystonia and normal controls showed similar predictive grip force adjustments to expected changes in object load, suggesting that this aspect of sensory-motor integration was normal. Patients had a shorter latency of grip force response than controls after an unexpected load increase, reflecting either a greater level of preparatory motor activity or a disinhibited spinal reflex response.

Conclusions: The overall increased grip force in patients with focal hand dystonia is likely to be a prelearned phenomenon rather than a primary disorder of sensory-motor integration.

Dystonia is a movement disorder characterised by sustained involuntary muscle contractions, frequently causing twisting and abnormal postures.^{1,2} By definition, focal, task specific dystonia affects one part of the body and occurs only during performance of a specific task, while other skills remain unaffected.^{1,3,4} The disorder typically affects the hand, as in writer's cramp and musician's cramp.^{1,3,5-8} Symptoms often start when holding the pen or instrument and increase when the subject begins to write or play.^{1,3,5} Although the association with specific activities is clear, the pathophysiology of task specific dystonia is still not well understood. Disturbed sensory-motor processing has been suggested by several groups.^{1,3,9,10} Indeed, the fact that many forms of focal dystonia can be relieved by "sensory tricks" is a clear indicator that sensory information plays an important role in focal dystonia. Based on such observations, the theory that sensory-motor integration is impaired in focal hand dystonia has attracted increasing interest.^{2,9}

Sensory-motor interaction is necessary in many tasks. For example, when subjects lift an object, their grip force is usually well matched to the weight being lifted.^{11,12} Previous work suggested that when individuals with writer's cramp lift an object, they grip it more forcefully than necessary.^{13,14} This inaccurate grip force scaling has been interpreted as a manifestation of impaired sensory-motor integration. Here we compared grip force behaviour in subjects with two forms of focal hand dystonia, writer's cramp and musician's cramp, and compared their performance with that in normal controls. We extended previous work by investigating how well subjects adapt their grip force when repeatedly lifting a new object, as well as how efficiently they can modulate their grip force in advance of expected increases in object load and in response to unexpected increases. Both of these require

integration of sensory information about the consequences of one movement with motor commands for subsequent movements, and hence can be considered additional ways of investigating sensory-motor interaction.

For the lifting task we reasoned that if task specific dystonia is associated with a generalised problem of sensory-motor integration then patients should not only produce an inefficient grip force overshoot in relation to the load, but they should also be unable to adjust their grip force output with increasing number of lifts performed. We analysed anticipation of grip force changes by asking subjects to grip a receptacle into which a weight was dropped either by the experimenter or by the subject with their opposite hand.¹⁵ An impaired sensory-motor processing might be predicted to result in deficient anticipation in people with focal hand dystonia.

METHODS

Subjects

We examined nine patients with focal hand dystonia (two women, seven men; age range 34 to 59 years, mean (SD) age, 43 (7) years). Clinical data on the patients are summarised in table 1. Ten normal subjects (two women, eight men; age range 34 to 58 years, mean 42 (7) years) served as a control group. All patients were able to relax their muscles completely and no dystonic movements or contractions were observed at rest. The diagnoses of writer's cramp and musician's cramp were based on characteristic clinical features: difficulties in writing and playing the instrument caused by abnormal muscle contractions or abnormal posturing, with preserved muscle strength. Task specificity was rated according to the classification of Sheehy and Marsden.¹⁶

Table 1 Clinical details of patients with task specific dystonia

Patient	Sex	Age (y)	Symptom duration (y)	Diagnosis (instrument)	Affected hand (hand dominance)	Associated symptoms	Task specificity	Treatment
1	F	34	3	Musician's cramp (piano)	R (R)	None	Dystonic	None
2	M	46	8	Musician's cramp (mandolin)	R (L)	None	Dystonic	None
3	M	59	1	Musician's cramp (piano)	R (R)	None	Simple	None
4	M	38	15	Musician's cramp (guitar)	L (R)	None	Dystonic	BTX
5	M	43	1	Musician's cramp (piano)	R (R)	None	Simple	None
6	M	47	5	Writer's cramp	R (R)	None	Simple	None
7	M	40	7	Writer's cramp	L (L)	None	Dystonic	None
8	M	35	10	Writer's cramp	R (R)	None	Dystonic	BTX
9	F	43	5	Writer's cramp	R (R)	None	Simple	None

Rating of task specificity was adopted according to the classification of Sheehy and Marsden.¹⁶ Patients treated with botulinum toxin were examined >6 months after the last injection.

BTX, botulinum toxin; F, female; L, left; M, male; R, right, y, years.

All participants gave their informed consent. The study was conducted in accordance with the Declaration of Helsinki and the methods were approved by the local ethics committee. All participants were naive to the specific purpose of the experiments.

Instrumented object

Subjects grasped, lifted, and moved a cylindrical and cordless instrumented object. All participants grasped the object for the first time during the experiments. The object and the configuration of the hand and fingers used to grasp it are illustrated for the experimental conditions in fig 1. The mass of the object was 400 g in both experimental conditions. The object had a diameter of 9.0 cm and a depth of 4.0 cm. Grip surfaces were sandpaper at a medium grain (No 240) in all trials undertaken. The object incorporated a force sensor for grip force registration (digital resolution: 0.0125 N/Bit) and linear acceleration sensors for registration of acceleration signals in three dimensions (fig 1A). The linear acceleration sensors measured acceleration within a range of $\pm 50 \text{ m/s}^2$. Recorded grip force and acceleration data were converted from analogue to digital with a sampling rate of 100 Hz and stored within the object by FLASH data storage. Data were transferred to a personal computer for analysis following each experimental setting with a single subject.

Procedures

The experiments addressed different motor behaviours: (A) lifting and holding an object; (B) compensating for a small weight dropped either expectedly or unexpectedly into a hand held receptacle. Before the experiments subjects washed their hands with soap and water and carefully dried them. Patients carried out the tasks with their affected hand. Each patient was age matched with a control subject, and the affected hand of the patient was matched with the left or right hand of the control subject according to hand dominance. For example, if the right hand was affected in a right handed patient, it was matched with the right hand of a right handed control subject. Subjects were seated in a stable chair in front of a table in a sufficiently lighted room.

(A) Lifting and holding the object

The subjects sat in a chair with their upper arm parallel to their trunk, and with their unsupported forearm extending anteriorly. In this position, they lifted the object, which was placed on the table before them. The object was grasped between the tips of the thumb and index finger on either side (fig 1A). The lifting movement took place mainly by flexion of the elbow joint. During the lifting trials the object was lifted 1 cm above the table, held in this position for five seconds, and then replaced and released. The lifting amplitude was indicated by a stable marker. Each subject

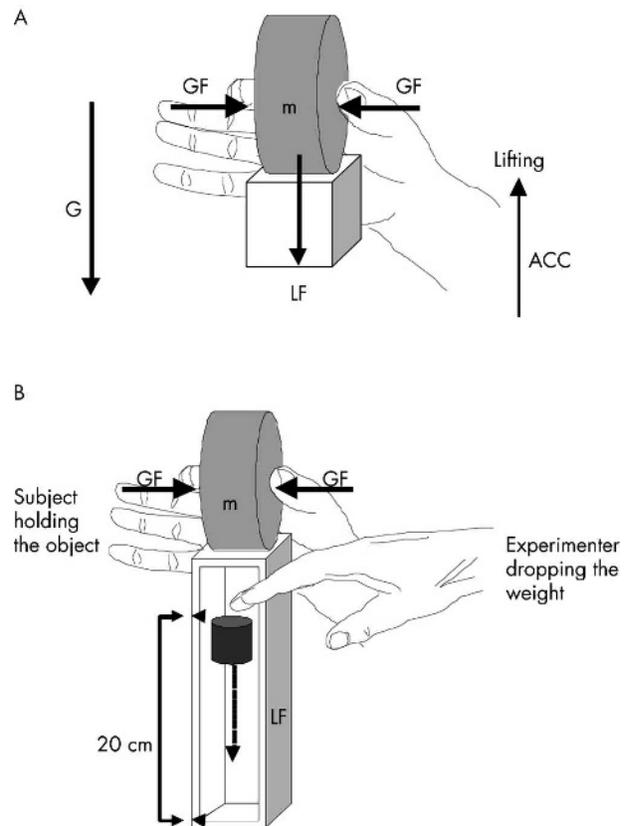


Figure 1 (A) Lifting the object. The instrumented object was mounted on a container. The object was grasped between the tips of the index finger and thumb, lifted 1 cm from the support, held in that position for five seconds, and then replaced and released. The object, the configuration of the hand and fingers used to grasp it, and the forces produced during a single lift are illustrated. (B) Catch up trial in the experimenter release condition. The subject, with eyes closed, held the object mounted on a receptacle between the tips of the index finger and thumb. The experimenter unexpectedly dropped a 100 g weight from a 20 cm height into the receptacle. A mark indicated a height of 20 cm between the dropping point and the bottom of the receptacle. The object incorporated a force sensor to record grip force and linear acceleration sensors to measure kinematic (ACC) and gravitational (G) accelerations in the three dimensions (X, Y, and Z axes). ACC, acceleration in the direction of movement; G, gravity; GF, grip force; LF, load force; m, mass (0.4 kg).

carried out 10 lifts with inter-trial intervals of five seconds. Before the experiments the subjects received verbal instructions from an experimenter, who also did demonstration trials. Following the 10 lifts, subjects were asked to hold the

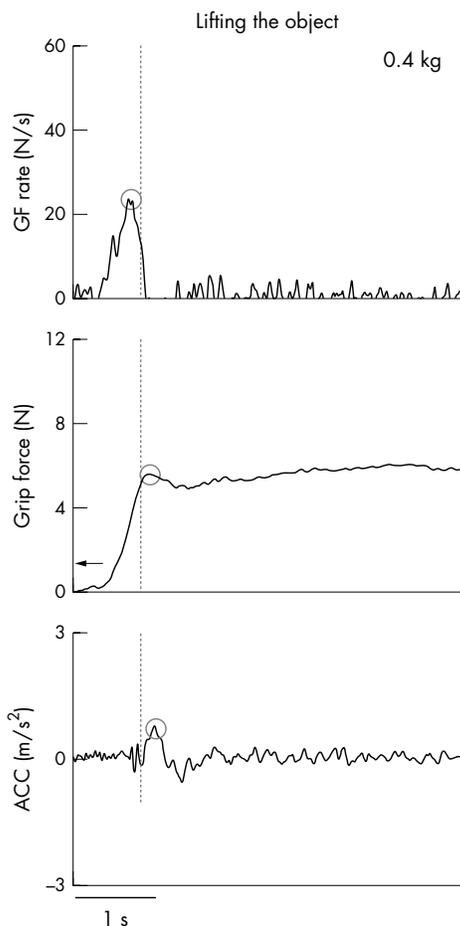


Figure 2 Traces of acceleration (ACC), grip force (GF) rate, and grip force over time for a single lift with the object undertaken by patient 4. Maximum values are indicated by circles within the traces of acceleration, grip force, and rate of grip force increase. The dotted vertical lines indicate the time of object lift off. The arrow in the grip force panel shows the minimum grip force required to prevent the object from slipping (slip force).

object just above the table surface and separate their thumb and index finger until the object dropped. This procedure was carried twice to obtain an estimate of the minimum grip force (slip force) necessary to prevent the object from slipping. The slip point was defined as the first detectable change in acceleration along the object's vertical axis, and the minimum grip force was determined at this time point.

(B) Weight catching trials

Subjects sat in a stable chair with the arm slightly abducted and the forearm held unsupported and rotated in front and in parallel to the trunk with the elbow flexed at about 90°. Subjects held the object mounted on a receptacle between the tips of the index and thumb (fig 1B). They were instructed to hold the object stationary and to prevent it from slipping. In the experimenter release condition, subjects were asked to keep their eyes closed for the entire experiment. The experimenter dropped a 100 g weight unexpectedly into the receptacle from a height of 20 cm indicated by a surface mark (fig 1B). In a second experiment, subjects themselves dropped the 100 g weight into the receptacle with their eyes open. The weight was dropped the same height of 20 cm. Ten such trials, with inter-trial intervals of five seconds, were carried out for the experimenter release and self release conditions, respectively.

Data processing and analysis

Positive acceleration of the object (along the object's vertical axis, see fig 1A) was directed upward during all experiments. When the object was held stationary no acceleration was measured. The net load force (LF) was calculated from the product of the object mass and the summation of gravity (9.81 m/s²) and inertial acceleration in three dimensions. This method included additional inertial loads which arose from incidental acceleration components in the X and Y directions (fig 1B).

Lifting the object

Grip force rate, acceleration data, and grip force data obtained from a single lift of patient 4 are illustrated in fig 2. Three time points within the course of each lifting movement were determined: (1) maximum rate of grip force change; (2) maximum acceleration; (3) maximum grip force. The maximum load force occurred at the time of maximum acceleration. The ratio between grip and load force is considered a sensitive measure of the efficiency of grip force scaling in relation to the lifting induced load. The ratio between grip force and load force was calculated at the time of maximum acceleration. We analysed the maximum grip force rate as well as peak grip force because the former occurs earlier in the lift, well before lift off (as measured by the first detectable change in acceleration), and thus provides a better index of the participant's prediction of object weight. In contrast, maximum forces occur later during the lift and may therefore be influenced by reactive control mechanisms.

Statistical analysis was aimed to address the issue of whether patients produced a grip force overshoot compared with controls. We used unpaired *t* tests for independent variables to compare group means of each variable for the lifts by patients and normal controls. A second issue was the question of whether the level of grip force output in relation to load was similarly influenced by repeated lifting by patients and controls. Separate multivariate analyses of variance (ANOVA) with "group" (writer's cramp, musician's cramp, and normal subjects) as a between subject factor and "lift trials" (1 to 10) as a within-subject factor were carried out on the average ratios between grip and load forces. A probability (*p*) value of 0.05 was considered statistically significant.

Weight catching trials

The maximum of load perturbation was only indirectly assessed by determining the maximum of downward acceleration of the receptacle caused by impact. When controlling this measure with an additional load sensor applied between the grip object and the receptacle the maximum load indeed occurred synchronously with the maximum peak in downward acceleration. However, we were unable to calculate accurate load magnitudes from the acceleration signal and thus we focused our analysis on the temporal measures. To exclude learning effects, only the last five weight catching trials were analysed in each condition. The first five trials were excluded from data analysis, given the fact that in the self release condition normal subjects need two to three trials to learn the time of weight impact and to adjust their grip force in anticipation. Two time points within the grip and acceleration traces were assessed: (1) maximum acceleration (coinciding with maximum load) and (2) maximum grip force. The two time points are indicated by dotted vertical lines and grey circles within the grip force and acceleration traces of a single catch up response of patient 4 in the experimenter release condition (fig 3A) and self release condition (fig 3B). The time between maximum grip force and maximum acceleration was assessed to provide a

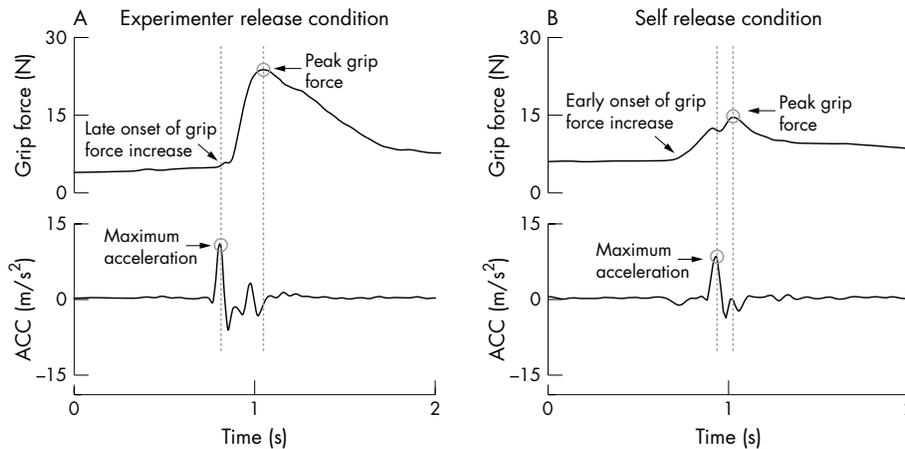


Figure 3 Grip force and acceleration signals obtained from single catch up trials of patient 4 in the experimenter release condition (A) and in the self release condition (B). Grip force started to increase after the impact, and maximum grip force lagged behind maximum acceleration in the experimenter release condition. In the self release condition grip force started to increase before impact, and maximum grip force coincided closely in time with maximum acceleration.

measure of the precision of the temporal coupling between grip and load forces.

Statistical analysis should answer the question of whether the time lag between maximum grip force and maximum acceleration differed between the two experimental conditions within each group, and between patients and controls. Unpaired *t* tests for independent variables were carried out to compare first, the weight catching trials for the experimenter release and self release conditions, and second, the perfor-

mance of patients and controls. A *p* value of 0.05 was considered significant.

RESULTS

The average slip forces obtained from the slip experiments were similar (*p* = 0.3) for patients (mean (SD), 1.7 (0.1) N) and controls (1.7 (0.2) N), implicating similar friction between the skin of the grasping digits and the object surface.

Lifting the object

In fig 2, profiles of the rate of grip force development (grip force rate), grip force, and acceleration (ACC) of the lifting movement are illustrated for a single lift carried out by patient 4. Circles indicate the maximum of each variable obtained for data analysis. In all patients and controls, the maximum force rate regularly occurred before lift off (see dotted vertical line in fig 2). The average peak accelerations of the lifting movement were similar (*p* = 0.2) in patients with musician’s cramp (0.6 (0.1) m/s²) and controls (0.5 (0.1) m/s²), but significantly higher in patients with writer’s cramp (1.0 (0.2) m/s²) compared with the normal subjects (*p* < 0.05) and with the patients with musician’s cramp (*p* < 0.05). The maximum load force depended directly on the maximum lifting acceleration.

In fig 4, average group data on peak rate of grip force increase, peak grip force, and the ratio between grip and load forces are illustrated for the 10 lifts. Patients with musician’s cramp and writer’s cramp produced higher maximum grip force rates than the controls (*p* < 0.01 and *p* < 0.001, respectively). There was no significant difference in maximum force rates between the two patient groups (*p* = 0.2). Also, maximum grip forces were higher for the patients with musician’s cramp (*p* < 0.001) and writer’s cramp (*p* < 0.05) than for the controls. However, there was no significant difference between the two patient groups (*p* = 0.4). To exclude the possibility that the different grip force output observed for patients and controls resulted from differences in the kinetics of the lifting movement, the ratio between grip and load forces at the time of maximum acceleration was calculated. Importantly, patients with musician’s cramp (*p* < 0.001) and writer’s cramp (*p* < 0.05) generated higher force ratios than the controls. The ratios were similar when comparing the two patient groups (*p* = 0.8). These data suggest that all patients produced excessive grip forces when lifting, regardless of the underlying pathology.

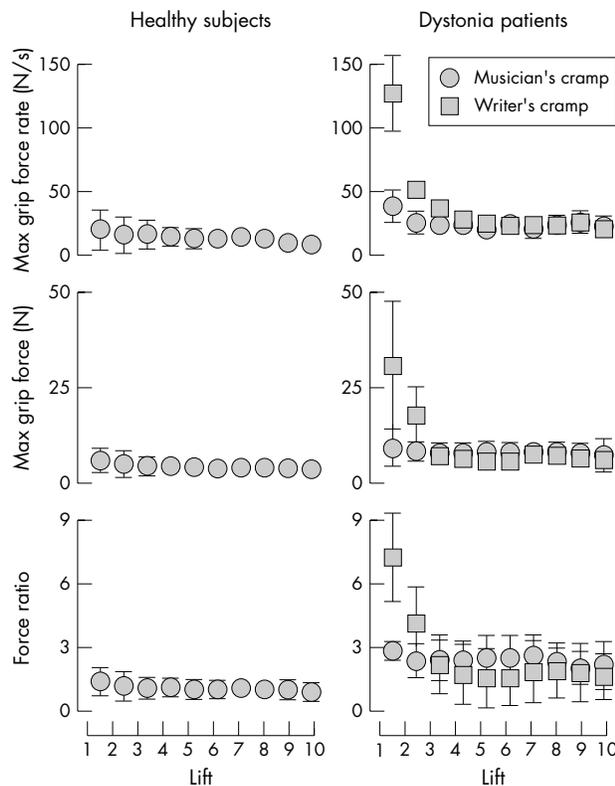


Figure 4 Average data on maximum rate of grip force increase, maximum grip force, and the ratio between maximum grip and load forces for all 10 lifts carried out by normal subjects and patients with musician’s cramp and writer’s cramp.

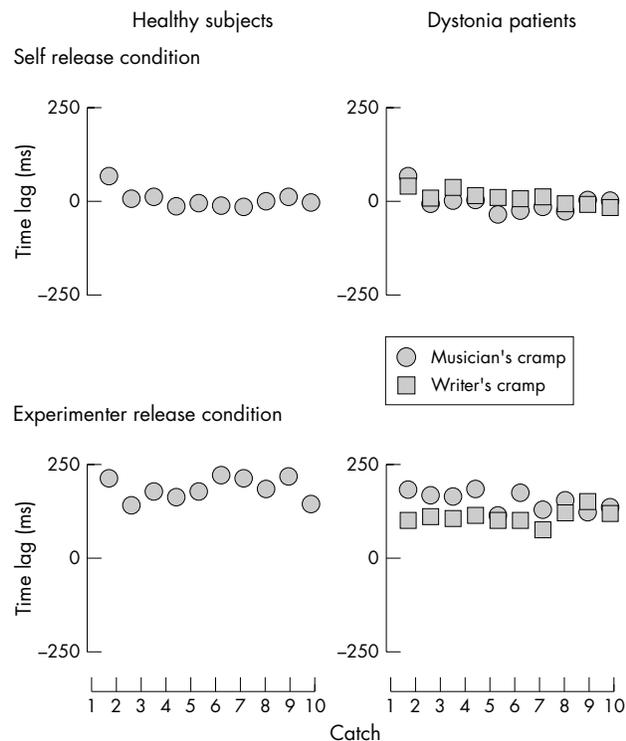


Figure 5 Average time lags between maximum grip force and maximum acceleration for all 10 weight catching trials carried out by normal subjects and by patients with musician's cramp and writer's cramp.

Figure 4 shows the adjustment of the grip force output across consecutive lifts in normal subjects and patients. These differed between the groups, with the percentage decrease of the force ratio from the first to last lift being 35% for normal controls, 25% for musician's cramp patients, and 78% for writer's cramp patients. Two way ANOVAs with the factors "group" and "lift trial" along with follow up analyses were carried out to address the question of whether these differences resulted from differences in the general force levels or from adaptation.

When comparing normal subjects and patients with musician's cramp there was no interaction between the factors, but each had a significant main effect on its own ("group", $F_{1,16} = 107.6$; $p < 0.001$; "lift trials", $F_{1,16} = 15.8$; $p < 0.01$). Additional one way ANOVAs showed a significant main effect of "lift trial" in each group separately (normal controls, $F_{1,18} = 7.3$; $p = 0.01$; musician's cramp, $F_{1,18} = 10.8$; $p < 0.01$), implying that grip force adaptation takes place in both groups. Thus the adaptation of the force ratio across the lift trials was similar in both groups, implying that the main effect of "group" resulted from the fact that the general force ratios were greater for patients with musician's cramp than for normal subjects.

A similar analysis was carried out for the data on normal subjects and subjects with writer's cramp. There was a two way interaction of "group" and "lift trial" ($F_{1,16} = 6.0$; $p < 0.03$), with each factor having a significant main effect on its own ("group", $F_{1,16} = 13.2$; $p < 0.01$; "lift trials", $F_{1,16} = 8.4$; $p < 0.01$). One way ANOVAs showed a significant main effect of "lift trial" for writer's cramp patients ($F_{1,18} = 21.3$; $p < 0.001$), as it did for normal subjects. These findings suggest that not only the general level of grip force but also the amount of decrease in the force ratio with increasing number of lifts performed was different in

between the two groups and clearly more pronounced for patients with writer's cramp.

Comparing the two patient groups, there was a two way interaction of the factors "group" and "lift trial" ($F_{1,16} = 5.5$; $p = 0.03$), with each factor again having a significant main effect on its own ("group", $F_{1,16} = 5$; $p = 0.04$; "lift trials", $F_{1,16} = 8.8$; $p < 0.01$). One way ANOVAs showed a significant main effect of "lift trial" for each group separately (writer's cramp, $F_{1,18} = 21.3$; $p < 0.001$; musician's cramp, $F_{1,18} = 10.8$; $p < 0.01$; controls, $F_{1,18} = 7.3$; $p = 0.01$). These findings indicate that the two patient groups differ in their general level of grip force and their amount of adaptation. The analysis also implies that an overall increase in grip force and the amount of force adaptation are not necessarily interdependent: Despite only slightly increased general grip force levels in comparison with writer's cramp, patients with musician's cramp showed a smaller degree of adaptation than those with writer's cramp (and even normal subjects).

Weight catching trials

In fig 3, acceleration and grip force traces are illustrated for weight catching trials in the experimenter release (fig 3A) and self release conditions (fig 3B). The maximum arm acceleration reliably coincided with maximum load force at the time of impact (see Methods). Following maximum acceleration, additional oscillations in acceleration occurred, probably because of corrective movements of the hand triggered by impact. In the experimenter release condition, grip force started to rise after impact and the maximum peak in grip force lagged some 100 ms behind the maximum peak in acceleration (fig 3A). In the self release condition, grip force started to rise well before impact and the maximum peaks in grip force and acceleration coincided in time (fig 3B).

The time lags between grip force and acceleration peaks were calculated for the last five of 10 trials of each individual and experimental condition (fig 5). The average time lag (mean (SD)) calculated for the experimenter release condition were 154 (26) ms for patients with musician's cramp, 111 (19) ms for patients with writer's cramp, and 188 (31) ms for normal subjects. The average time lags for the self release condition were 10 (19) ms for patients with musician's cramp, -3 (28) ms for patients with writer's cramp, and 5 (24) ms for normal controls. The time lags were longer for the experimenter release condition than for the self release condition within each group ($p < 0.001$ for all comparisons). We found no significant difference in time lags obtained from trials in the self release condition when comparing each group ($p \geq 0.1$ for all comparisons). The time lags obtained from trials in the experimenter release condition were longer for controls than for patients with musician's cramp ($p < 0.03$) or writer's cramp ($p < 0.001$). The time lags calculated for the experimenter release condition were longer for patients with writer's cramp than for those with musician's cramp ($p < 0.01$). Thus, as described for normal subjects,¹⁵ all patients were capable of predicting the load perturbation at impact and of regulating grip force in anticipation in the self release condition. In the experimenter release condition the perturbation was not predictable, and a reactive control mechanism was triggered by impact, with the consequence that grip force lagged behind load.

DISCUSSION

We investigated grip force scaling during repeated lifts with a novel object and predictive/reactive grip force adjustments during a weight catching task in patients with musician's cramp and writer's cramp. There were three major findings:

- Patients with musician's cramp and writer's cramp produced an inefficient increase in grip force when lifting a weight.
- Despite this, patients with musician's cramp and writer's cramp adjusted their force output with increasing accuracy over the first 10 trials of lifting a new object. The extent of grip force adjustment in relation to the load was similar for patients with musician's cramp and normal controls, but more pronounced for patients with writer's cramp.
- Patients with focal hand dystonia generate both predictive and reactive grip force adjustments when catching an expectedly or unexpectedly released weight.

Our data support earlier investigations on grip force behaviour in writer's cramp^{13, 14} and extend these to patients with musician's cramp, suggesting that the conclusions may be characteristic of all forms of focal hand dystonia. We discuss our findings with regard to the current theory that focal hand dystonia represents a deficit of sensory-motor integration.

In their previous studies Odergren *et al* asked their patients to perform five practice lifts that were not included in data analysis, and focused their analysis on the average of five additional lifts.¹³ Consequently, their patients were already familiar with the object when the measurements were taken. Our measurements made after subjects had practised the task gave similar results: patients grasped the object with greater force than normal. However, in our present study we also examined the initial adaptation to the new object over the first 10 lifting trials. As described previously by Johansson and colleagues,¹¹ normal subjects often overestimate the grip force required to lift a new object, but then adapt the force rapidly within the first few lifts. Such an adaptation relies mainly on cutaneous mechanoreceptors that indicate the relevant mechanical properties of the object, such as weight and surface friction. This allows subjects to update their predictions of the necessary grip force to match the actual requirements of the task.^{12, 17–19} The important feature of the present results is that this initial adaptation still occurred and was of the same extent in patients with musician's cramp as in normal subjects, but more pronounced in patients with writer's cramp.

We can only speculate why the grip force overflow during the initial lifts was more pronounced in patients with writer's cramp. Clinically, the motor deficit in these patients usually appears to be a problem of co-contraction of antagonistic hand and finger muscles.^{1, 2} In comparison, the motor problem of patients with musician's cramp is more suggestive of deficient coordination than of excessive force production. The detailed mechanisms underlying these differences remain unknown, but they may contribute to the differences in the initial force overflow observed in both patient groups when first manipulating a novel weight. An alternative explanation could be that the task of grasping and lifting is more similar to the task of writing than to the task of playing an instrument. As writer's cramp is a task specific dystonia, it could be that the grip force task used in our study is closer to the "primary" deficit in writer's cramp than to that in musician's cramp. Interestingly, both patient groups retained the capacity to reduce the excessive grip force output with increasing number of lifts performed and settled down at similar force levels, which were nevertheless greater than those of the normal controls. This observation implies that patients with focal hand dystonia do not suffer a general problem of force calibration, but rather are able to match their fingertip forces more accurately to the mechanical object properties the more experienced they become with the task.

The emphasis on overestimation of grip force in previous studies led the investigators to speculate that it reflected an underlying impairment in the capacity to integrate sensory information into motor programmes.^{13, 14} However, Schenk and Mai²⁰ raised some doubts about the general validity of such a theory, suggesting that the increased grip force levels found in patients with writer's cramp may be prelearned and a consequence of an effortful writing style. They found that the increased grip force levels in patients with writer's cramp could be reduced by visual feedback based training; conversely, a period of effortful writing in normal subjects increased their grip force levels. Indeed, it has recently been shown that in normal subjects the amount of grip force produced when lifting a particular object is significantly influenced by the level of a preceding pinch, produced with either the same or the opposite hand.²¹ It seems likely, therefore, that the force overshoot observed in our patients does not reflect a general inability to use sensory information, but is simply related to the expectation by patients with dystonia that they will experience a certain force when holding an object.

As observed for normal controls, all our patients showed a predictive increase in grip force before impact when they dropped the weight from the unaffected hand into the receptacle held with the affected hand. In contrast, when the weight was unexpectedly dropped from the experimenter's hand none of the participants was able to predict the time of impact, with the consequence that grip force lagged behind load. Time lags of 100–200 ms between grip and load force peaks suggest feedback based grip force responses.¹⁵ In line with our observations, predictive and reactive grip force adjustments to expected and unexpected perturbations were mainly unaffected in patients with writer's cramp doing a drawer opening task.¹⁴ Interestingly, in our experiment the time lag between load impact and the reactive grip force peak was longer for normal subjects than for patients with focal hand dystonia. Odergren and colleagues also observed shorter latencies of the grip force responses to a sudden unexpected perturbation in individual patients with writer's cramp.¹³ Why this should be is unclear. The short grip force latencies may be related to the greater level of preparatory motor activity, as reflected by the overall larger grip force levels in dystonic patients. Alternatively, they may reflect disinhibited spinal reflex responses. Indeed, the long latency stretch reflexes in subjects with non-focal dystonia are characterised by spread to adjacent muscles,²² compatible with deficient reflex inhibition at the spinal level.

The theory that sensory-motor integration is impaired in focal hand dystonia has attracted increasing interest in recent years.^{2, 9} Impaired integration may result from abnormal gating of sensory information at the level of the basal ganglia, causing an input-output mismatch in specific motor sub-routines, or alternatively from inaccurate sensory processing of ongoing motor programmes. Our data suggest that finger force adaptation to the mechanical object properties is well preserved in focal hand dystonia. The implication is that this aspect of sensory-motor coordination is intact, and so this casts doubt on the general validity of the sensorimotor disorganisation theory in focal dystonia. However, our study is limited by the small sample size and therefore any conclusions from our findings must be drawn with caution. Nevertheless, the fact that sensory input can be used effectively in these patients may be a useful clue to the success of any behaviour based rehabilitation techniques.

ACKNOWLEDGEMENTS

We are grateful to all patients and normal control subjects for their participation. This research was funded by a grant of the European Neurological Society (ENS fellowship stipend) to DAN.

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Competing interests: none declared

REFERENCES

- 1 **Chen R**, Hallett M. Focal dystonia and repetitive motion disorders. *Clin Orthop* 1998;**351**:102–6.
- 2 **Fahn S**. Concept and classification of dystonia. *Clin Neuropharmacol* 1998;**9**:S37–48.
- 3 **Lim VK**, Altenmüller E, Bradshaw JL. Focal dystonia: current theories. *Hum Mov Sci* 2001;**20**:875–914.
- 4 **Sheehy M**, Rothwell JC, Marsden CD. Writer's cramp. In: Fahn S, eds. *Dystonia 2*. New York: Raven Press, 457–72).
- 5 **Brandfondbrener AG**. Musicians with dystonia: a report of 58 cases seen in a ten-year period at a performing art medicine clinic. *Med Probl Perform Art* 1995;**10**:121–7.
- 6 **Lederman RJ**. Occupational cramp in instrumental musicians. *Med Probl Perform Art* 1988;**3**:45–51.
- 7 **Pascual-Leone A**. The brain plays music and is changed by it. *Ann NY Acad Sci* 2001;**930**:315–29.
- 8 **Poore GV**. Clinical lecture on certain conditions of the hand and arm which interfere with the performance of professional acts, especially piano-playing. *BMJ*, 1887;ii, 441–4.
- 9 **Abbruzzese G**, Beradelli A. Sensorimotor integration in movement disorders (2003). *Mov Disord* 2003;**18**:231–40.
- 10 **Bara-Jimenez W**, Catalan MJ, Hallett M, et al. Abnormal somatosensory homunculus in focal hand dystonia. *Neurology* 2000;**55**:1869–73.
- 11 **Flanagan JR**, Johansson RS. Hand movements. In: Ramshandran VS, eds. In: *Encyclopedia of the human brain*, vol 2. San Diego: Academic Press, 2002:399–414.
- 12 **Johansson RS**, Westling G. Roles of glabrous skin receptors and sensorimotor memory in automatic control of precision grip when lifting rougher and more slippery objects. *Exp Brain Res* 1984;**56**:550–64.
- 13 **Odergren T**, Iwasaki N, Borg J, et al. Impaired sensory-motor integration during grasping in writer's cramp. *Brain* 1996;**119**:569–83.
- 14 **Serrien DJ**, Burgunder JM, Wiesendanger M. Disturbed sensorimotor processing during control of precision grip in patients with writer's cramp. *Mov Disord* 2000;**15**:965–72.
- 15 **Johansson RS**, Westling G. Programmed and triggered actions to rapid load changes during precision grip. *Exp Brain Res* 1988;**71**:72–86.
- 16 **Sheehy MP**, Marsden CD. Writer's cramp – a focal dystonia. *Brain* 1982;**105**:461–80.
- 17 **Nowak DA**, Hermsdörfer J. Selective deficits of grip force control during object manipulation in patients with reduced sensory feedback from the grasping digits. *Neurosci Res* 2003;**47**:65–72.
- 18 **Nowak DA**, Hermsdörfer J, Glasauer S, et al. The effects of digital anaesthesia on predictive grip force adjustments during vertical movements of a grasped object. *Eur J Neurosci* 2001;**14**:756–62.
- 19 **Nowak DA**, Glasauer S, Hermsdörfer J. Grip force efficiency in long term deprivation of somatosensory feedback. *Neuroreport* 2003;**14**:1803–7.
- 20 **Schenk T**, Mai N. Is writer's cramp caused by a deficit of sensorimotor integration? *Exp Brain Res* 2001;**136**:321–30.
- 21 **Quaney BM**, Rotella DL, Peterson C, et al. Sensorimotor memory for fingertip forces: evidence for a task-independent motor memory. *J Neurosci* 2003;**23**:1981–6.
- 22 **Rothwell JC**, Obeso JA, Day BL, et al. Pathophysiology of dystonias. In: Desmedt JE, eds. In: *Motor control mechanisms in health and disease*. New York: Raven Press, 1983:851–63.