Maintaining internal representations: the role of the human superior parietal lobe

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In sensorimotor integration, sensory input and motor output signals are combined to provide an internal estimate of the state of both the world and one's own body. Although a single perceptual and motor snapshot can provide information about the current state, computational models show that the state can be optimally estimated by a recursive process in which an internal estimate is maintained and updated by the current sensory and motor signals. These models predict that an internal state estimate is maintained or stored in the brain. Here we report a patient with a lesion of the superior parietal lobe who shows both sensory and motor deficits consistent with an inability to maintain such an internal representation between updates. Our findings suggest that the superior parietal lobe is critical for sensorimotor integration, by maintaining an internal representation of the body's state.

Damage to the human posterior parietal cortex (PPC) can cause perceptual and motor deficits, including visual mislocalization, neglect or unawareness of contralesional visual or tactile stimuli, impaired perception of contralesional body parts and misreaching to visual objects[1–4]. The study of these syndromes, together with more recent functional imaging investigations of individuals without brain injury[5–8] has led to proposals that the human PPC is important for spatial perception, association of sensory signals, directing attention, visuomotor control and motor planning[4,9–12]. Unfortunately, no unifying theoretical framework has emerged from human studies for understanding the function of the human PPC. Instead, there has been considerable reliance on neurophysiological investigations in monkeys[9,13–17], and the models developed from them, to formulate working hypotheses of human PPC function. Such extrapolations are, at best, indirect.

We propose a new theoretical framework for the visuomotor functions of the human parietal cortex. Recent developments in the field of sensorimotor integration have provided a new approach to understanding how sensory and motor signals are combined to provide an estimate of the state of both the world and one's own body[18,19]. The approach we use comes from the powerful observer framework in engineering[20]. This framework is relevant when there is a system under control, in our case the body, and the goal is to estimate the state of the system. By state we refer to a compact set of variables that captures both the configuration of the body, such as joint angles or hand position, and its interactions with the world, such as contact with an object. For optimal (most accurate) state estimates, the observer framework requires a system that monitors both the inputs and outputs of the system, in our case the sensory feedback and motor commands. The observer then uses these values to produce online state estimates, which are updated as further sensory and motor signals arrive. The major objective of the observer is to reduce the uncertainty in the state estimate that arises from noise inherent in both the sensory and motor signals. Integrating these signals over time, rather than relying on a single perceptual or motor snapshot, produces an improved estimate. The new estimate of state is made by recursively updating the current state estimate based on the incoming motor and sensory signals.

A key feature of the observer model is that it requires that the state representation be stored and updated as new information arrives (Fig. 1). Updating the state estimate and storing the state estimate between updates can be considered as two functionally and possibly anatomically distinct processes. Although such an internal state estimate has been proposed within the central nervous system[19,21], there have been no reports of lesions that result in a specific inability to maintain such an estimate. The key claim in this paper is that the state estimate is stored in the parietal cortex.

Results

We have studied a patient with a parietal lesion who has unusual sensory and motor deficits that become apparent only as the duration of a sensory stimulus or movement increases. The patient, PJ, is a right-handed, fifty-year-old woman who was first seen two years before our study by the Neurology service following episodes of involuntary jerking of the right arm, which were considered to be focal seizures. Magnetic resonance imaging at that time demonstrated an extra-axial cyst encroaching upon the cortex and subcortical white matter of the left superior parietal lobe. The images are consistent with those of an arachnoid cyst, but similar appearances may occur following trauma. The patient recalls that five years before these fits she sustained an accidental injury to the left side of her head with loss of con-

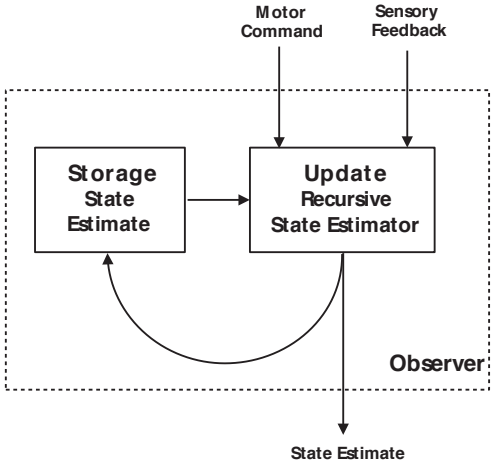

Fig. 1. A schematic of the sensorimotor integration process. The 'observer' monitors and integrates both the inputs and outputs of the system, the sensory feedback and motor commands, by recursively updating the stored state estimate.

sciousness for thirty minutes. At the time she presented with focal seizures affecting the right arm, she declined cystoperitoneal shunting but agreed to regular follow-up scanning. She started on carbamazepine for prevention of further focal seizures. On a maintenance dose of 600 mg per day, she had no recurrence.

Two years later, she has returned complaining of new symptoms. She now reports that she perceives her right arm and leg to drift and then fade unless she is able to see them. For example, while lying in bed, she might realize that she has 'lost' her right arm. Only when she looks at her arm does she know where it is. Similarly, she can be sitting on a bus and find that another passenger has tripped over her right foot, which is situated, without her knowledge, in the middle of the aisle. Repeat magnetic resonance imaging now demonstrates that the cyst has grown radially by 2.5 mm (Fig. 2). Unlike other parietal cases, in which patients are permanently unaware of their limbs, her symptoms are novel because she only becomes unaware of her right arm and leg over tens of seconds. Furthermore, she can regain awareness of the location of her right arm and leg by looking at them, although she cannot voluntary regain awareness otherwise.

On examination, her visual fields were full to confrontation (normal). There was no visual extinction or neglect as assessed by line bisection, cancellation on the Mesulam shape cancellation task[22] and object drawing. Saccadic and smooth-pursuit eye movements seemed normal. Tone and power in the upper and lower limbs were normal bilaterally. Deep tendon reflexes were slightly brisker in the right limbs than the left. Plantar responses were flexor (normal). Although there was subjective diminution of light touch in the right arm, her ability to discriminate sharp from blunt tactile stimuli was good. However, if a tactile stimulus was held still on her right hand, when her eyes were closed, she reported that the sensation faded over seconds until she could no longer detect the stimulus. Proprioception and vibration were also subjectively considered to be different in the right upper limb than the left, but there seemed to be no objective difference on clinical testing. She had right tactile extinction. Astereognosis (impaired ability to recognize objects by their size, shape, and texture) and agraphaesthesia (inability to identify numbers written on the palm) were detected in the right hand. When asked to point to the examiner's finger with free vision, she had no difficulty using either left or right hand. However, when she was asked to fixate centrally on the examiner's nose and point slowly to his finger, she consistently missed when using her right hand, regardless of whether reaches were made into the left or right visual field. There was no limb dyspraxia (motor deficits which are not a consequence of weakness or sensory loss), and her gait was normal.

We investigated the time course of PJ's tactile perception by placing weights on the back of her hand. Although PJ was able to detect, without vision, when an object was placed on the back of her right hand, the percept then faded until she could no longer detect the object (Methods). The time to fade increased linearly ($r^2 = 0.63, p < 0.001$) with the mass of the object (Fig. 3). For the lightest weight of 10 grams, the percept took 2.9 seconds to disappear, whereas for a weight of 150 grams, the time to fade increased to 10.5 seconds. Although PJ could no longer detect the object at the end of each trial, she could always detect its removal. If the weight was moved to another part of her hand, she could both detect its removal and its replacement. However, once the weight was stationary, the percept of the weight would eventually disappear. In contrast, when the weight was applied to her left hand, the percept did not fade, and she never reported being unable to detect the object. When vibratory stimuli were applied, the percept did not fade on either hand. Whereas parietal patients with central deafferentation are unable to perceive either constant or changing stimuli[23,24], PJ becomes effectively deafferented only after a period of several seconds in the presence of a constant tactile stimulus.

To assess the motor consequences of her tactile fading, we examined PJ's ability to maintain a precision grip with and without visual feedback of her performance (Methods). She held a force transducer with her thumb and index finger in a precision grip and was asked to maintain a constant grip force. Grip force was displayed on an oscilloscope screen together with the target level of grip force. Having matched the grip force under visual feedback of the oscilloscope screen, PJ was asked to maintain the grip force constant with either full vision or no vision. When given visual feedback of her performance, PJ was able to maintain her grip level using either hand (Fig. 4a and b). Like neurologically normal subjects[24], without visual feedback, PJ was still able to maintain her force level using her unaffected left hand (Fig. 4c). However, when using her right hand, the grip force decayed to near zero over 10–15 seconds (Fig. 4d; the removal of

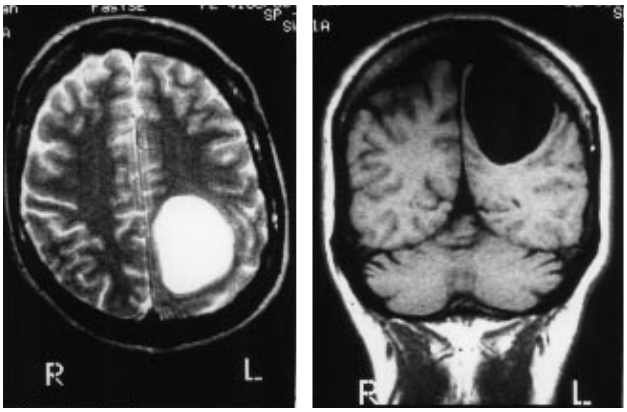

Fig. 2. The lesion found in the patient P. T2-weighted axial (left) and T1-weighted coronal (right) magnetic resonance imaging sections demonstrating a left superior parietal cyst.

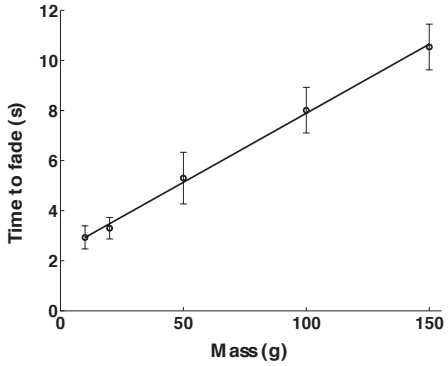

Fig. 3. Mean time to fade (with standard error bars) against the mass of the object placed on PJ's right hand. The solid line shows a linear regression fit to the data.

visual feedback occurred at the transition from the dotted to solid lines). The time course of the grip-force decay was similar to that of the perceptual fading to constant tactile stimuli.

To investigate the perceived drifting of her arm, we measured PJ's perception of the location of her right arm in the absence of vision (Methods). The affected right hand remained at a fixed location in contact with a table and did not move. PJ was asked to track, with her left hand, the perceived and not actual (and unchanging) position of the right hand. The perceived location of her right hand began to drift after a latency of about 18 seconds (Fig. 5b), and the path taken by the 'perceived' hand was similar for four starting locations (Fig. 5a), moving initially upwards and then to the right of the body, travelling about 30 cm from its true location. The arm continued to drift and eventually it seemed to PJ to 'disappear' altogether. A study of neurologically normal subjects demonstrated that the perceived location of their stationary limb drifted by less than 1 cm over the course of 120 seconds[25] . Like normals, when PJ's left hand was stationary, she never reported that it drifted or faded.

Corresponding to this proprioceptive drift, PJ had a deficit when making slow pointing movements to peripheral targets while fixating a central stimulus (Methods). A comparison of the accuracy of movements made at a normal pace (mean movement duration, left 2.2 ± 0.1 s, right 2.4 ± 0.3 s) showed no difference in accuracy between the left and right hands (Fig. 6). However, for slow movements (mean durations, left 14.5 ± 0.8 s, right 18.6 ± 1.0 s) PJ had a significantly higher error ($p < 0.001$) when using her right hand compared to the left (mean increase in error, 16.8 cm). Similar to a previous study of patients with optic ataxia[26] , who have lesions of the superior parietal lobe and adjacent intraparietal sulcus, PJ demonstrated misreaching to peripheral visual targets while maintaining central fixation. However, the novel finding here is that PJ was only inaccurate when she was required to make very slow movements. This suggests that the fading observed to constant stimuli is a process that continues when the sensory signal is changing, such as during movement, and was therefore manifest only when the time course of movement was prolonged to that of the temporal decay.

Discussion

Although PJ was able to detect both tactile and proprioceptive sensory inputs, she showed a profound tactile fading to constant stimuli and a concomitant inability to maintain a constant force output. Without vision of her right arm, she perceived it as drifting in space, and correspondingly when required to make slow pointing movements, she produced large errors with the right arm. Our approach to understanding these deficits is within the observer framework, in which the state estimate must be stored between recursive updates by sensory and motor signals. We propose that in PJ the storage mechanism is damaged so that the stored state estimate is perpetually decaying over time. The recursive state estimation process, which would use this corrupted, inaccurate estimate to form the next estimate, would result in an accumulation of error over time.

Although the sensory and motor signals that are used to update the representation may be normal in PJ, they would be integrated into a representation that is perpetually decaying. When there is an unvarying sensory or motor signal, the state estimate would be expected to decay, whereas a strong sensory or motor signal (for example a changing one) acting on the decaying representation could effectively bring it back to nearer the true value. We suggest that PJ lacks the ability to maintain the representation of her arm across time because of damage to the storage of the state estimate. All PJ's symptoms can be considered as an accumulating error in the internal estimate of her arm's state. This provides a concise and parsimonious description of her deficits. These experimental findings suggest that PJ, though able to detect sensory events and make fast, accurate limb movements, is unable to maintain an accurate internal representation of her state across time. Furthermore, her inability to maintain this representation leads to deficits in both perception and action that emerge over time.

PJ demonstrates sensory and motor deficits that have not previously been reported in lesion studies. Most reports have inves-

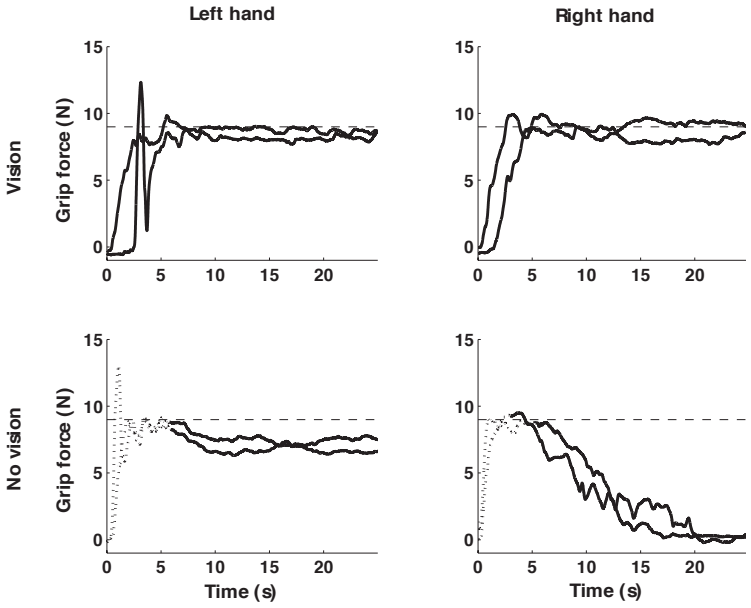

Fig. 4. Grip force against time when trying to maintain a constant force level with and without visual feedback of performance. Top row, full vision. Bottom row, no visual feedback. The dashed lines indicate the target force level. In the no-vision trials, the dotted line indicates the period before the removal of visual feedback.

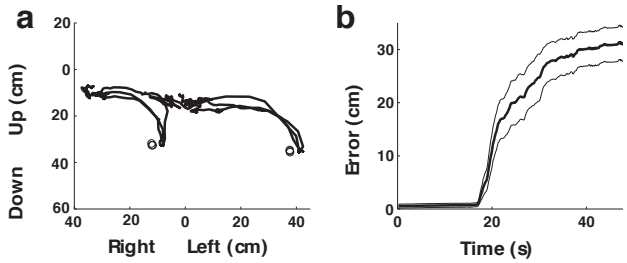

Fig. 5. Perceived drift of right arm. **(a)** Paths of perceived position of PJ's right hand in the vertical plane for two of the four different starting positions. The midpoint of the eyes is at (0,0), and the circles show the location of the right index finger. Each line represents a different trial. For clarity, only the two extremal starting locations are shown. **(b)** Mean error, the difference between actual and perceived position of the right hand, against time with outer standard error lines (n = 12).

tigated patients with conditions of sudden onset, such as strokes and rapidly growing tumours. Parietal patients who demonstrate astereognosis without any associated primary somatosensory disturbance have been described[27]. However, to the best of our knowledge, there are no reports of patients in whom perception of tactile stimuli fades with time, is restored by vision and is associated with a motor impairment that emerges over a similar time-course. The uniqueness of PJ's impairments may be attributable to the nature of her lesion, which has been observed over two years to be slowly progressive. This slow evolution may have allowed us to observe an intermediate stage in parietal dysfunction that has not been observed previously. Although patients with strokes of the parietal lobe may have sensory or motor deficits involving their contralateral limbs, the damage may be so absolute that the time course of sensorimotor integration is not amenable to experimental observation.

Although two previous studies have reported an accelerated visual fading of peripheral targets, known as Troxler fading, in patients with parietal lesions[28,29], this has been attributed to an attentional deficit. It is unlikely that PJ's disorder can be attributed simply to dysfunction in an attentional mechanism. PJ did not demonstrate a disorder in sustaining attention as at the end of the weight trials; when she could no longer feel the weight on her hand, she had no difficulty feeling it when it was unexpectedly removed. Even when asked voluntarily to attend to the hand supporting the weight, she could not detect the presence of the weight. Furthermore, the 'fading' of her awareness was so predictable and unvarying with any given stimulus that it seems implausible that it is due to a failure to maintain attention voluntarily. This is unlike previous reports of somatosensory disturbance following parietal lesions, in which patients' report of tactile stimuli could be improved by asking them voluntarily to attend to their affected hand[27,30]. We suggest instead that the profound somatosensory fading and concomitant misreaching found in PJ indicate that the superior parietal lobe has a key role in actively maintaining an internal representation of the body's state.

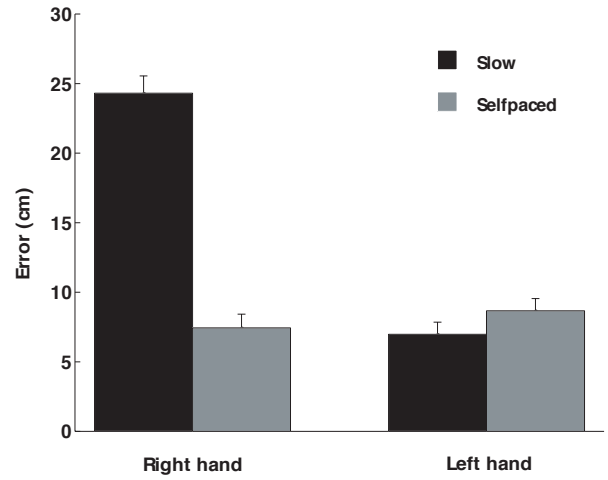

Fig 6. Mean error with standard error bars for pointing movements made with the right and left hand at self-paced or slow speeds to peripheral visual targets.

Although the radiological features suggest that the lesion was centered on the superior parietal lobe, there is distortion of the precuneus (medial superior parietal lobe) and inferior parietal lobe. It is possible to argue that PJ had a superior parietal lobe lesion for some time without symptoms, and that her sensory and motor problems emerged only after pressure on these adjacent structures. However, lesions of the inferior parietal lobe in man are usually associated with the visual and visuomotor disturbances of the neglect syndrome[31,32], and PJ showed no evidence of neglect. In contrast, PJ demonstrated misreaching to peripheral visual targets while maintaining central fixation. Such optic ataxia is known to follow lesions of the human superior parietal lobe[26]. Therefore, although the lesion may affect all regions of the parietal lobe, we feel that PJ's symptoms are most consistent with a superior parietal site.

Previous studies have suggested that the superior parietal lobe in humans is involved in disengaging or maintaining attention to visual and tactile stimuli[5,33-35] (although a recent study suggests that it may not be the critical structure in disengaging attention[36]), feature binding of visual stimuli[6], prism adaptation[8] and visually-guided reaching[7,10,17,37]. It has also been proposed that the posterior parietal lobe in monkey is involved in processing both sensory signals and motor commands[9,10,13,17]. Our findings suggest that the superior parietal lobe has a key role in sensorimotor integration, by actively maintaining an internal representation of one's own body. This may be the representation over which attention is shifted. Alternatively, it is possible that this representation may be separate from those used for directing attention or holding a body schema.

In conclusion, the findings of the present study demonstrate, for the first time, deficits in sensory and motor processing that emerge over the same time course in a human subject with a parietal lesion. We propose that the temporal deficits observed are due to a failure to maintain an internal representation, common to both the sensory and motor systems, which is necessary for both perception and action.

Methods

For all experiments, the patient was seated comfortably and wore a pair of Plato liquid crystal glasses (Translucent Technologies), which could be switched under computer control between translucent and opaque, thereby controlling visual feedback. As only her right side is affected, her left arm was used as a control in all the experiments.

PJ's ability to perceive a tactile stimulus over time was tested by asking her to report when an object placed on the back of her hand could no longer be felt. PJ sat with her palms resting face down on a table and had her vision occluded by the Plato glasses. For each trial, a cylindrical

weight was placed on the back of her hand. The base of the weight was covered by a plastic disc to prevent temperature cues. PJ was asked to report when she could no longer feel the weight on the back of her hand, and the time was recorded. Five different weights (10, 20, 50, 100 and 150 g) were placed on her hand, each for 9 repetitions in a pseudorandom order. A small vibrator was used to examine whether dynamic stimuli faded.

To examine the consequences of the sensory fading, we determined PJ's ability to maintain a constant force output. PJ held a force transducer (Assurance Technologies) with her thumb and index finger 1 cm apart. The grip force produced was sampled at 250 Hz by a CED 1401plus (Cambridge Electronic Design). Grip force was recorded and simultaneously displayed on an oscilloscope screen. Also shown on the oscilloscope was a target level of grip force (either 4.5 or 9 N), which PJ was required to match. Having matched the grip force under visual feedback of the oscilloscope screen, PJ was required to maintain the grip force constant for a further 20 seconds under two different conditions. In the full vision condition, PJ had vision of both her own hand and the oscilloscope displaying the actual and target grip force. In the second, no vision condition, the Plato glasses prevented all visual feedback. Each of these four conditions (two force levels and two visual feedback conditions) was repeated twice for both the right and left hands. Results for the 4.5 N force level (not shown) were similar to those for the 9 N condition.

To quantify the perceived location of her right limb in the absence of visual feedback, PJ was required to track the perceived position of her right hand with her left hand. PJ was seated at a table with her hands placed palms down next to each other, with index fingers parallel. The positions of the fingers were recorded using an active infra-red tracking system (Optotrak 3020, Northern Digital) sampling at 250 Hz. At the beginning of each trial, PJ was instructed to look at the positions of her hands. Vision was then occluded, and PJ tracked the perceived position of the right hand with her left hand. The right hand remained stationary throughout the trial. Three trials for each of four different starting locations of her right hand were recorded for up to 80 seconds.

To quantify PJ's optic ataxia, we assessed her pointing accuracy to visual targets in her periphery by asking her to point, as accurately as possible, to the experimenter's finger while fixating on his nose. Movements were made with both the right and left hand at a self-paced and slow speed. The locations of both the experimenter's and PJ's fingers were recorded using the infrared tracking system. Ten repetitions to different targets were made with both hands and at both speeds.

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