LETTER

Attention deficits without cortical neuronal deficits

Alexandre Zénon[1,2] & Richard J. Krauzlis[2,3]

The ability to process relevant stimuli selectively is a fundamental function of the primate visual system. The best-understood correlate of this function is the enhanced response of neurons in the visual cortex to attended stimuli[1,2]. However, recent results show that the superior colliculus (SC), a midbrain structure, also has a crucial role in visual attention[3–5]. It has been assumed that the SC acts through the same well-known mechanisms in the visual cortex[3,5]. Here we tested this hypothesis by transiently inactivating the SC during a motion-change-detection task and measuring responses in two visual cortical areas. We found that despite large deficits in visual attention, the enhanced responses of neurons in the visual cortex to attended stimuli were unchanged. These results show that the SC contributes to visual attention through mechanisms that are independent of the classic effects in the visual cortex, demonstrating that other processes must have key roles in visual attention.

Visual attention is a fundamental brain function that makes it possible to base perceptions and actions on the relevant parts of the environment. In the laboratory, visual attention is typically studied by asking subjects to respond to the properties of a cued stimulus while simultaneously ignoring the content of irrelevant, distracting stimuli. Twenty-five years ago, it was shown that in the primate visual cortex, the activity of neurons responsive to cued visual stimuli was higher than the activity evoked by un-cued distracters[6]. This finding, later termed 'gain modulation', has been subsequently observed in many different areas of the cerebral cortex[2,7,8], in many variants of the cueing task[8].

Visual attention is now understood to involve a network of areas, including the frontal and parietal cortex, as well as the visual cortex[9], and gain modulation of sensory responses is commonly considered to be the keystone of the neuronal mechanisms of attention[1,2].

Correlates of visual attention are not restricted to the cortex and have also been found in subcortical structures such as the SC[10,11] and thalamus[12–14]. Some of these effects could be inherited from the cortex. However, manipulation of neuronal activity in the SC alters or disrupts performance in tasks that test visual attention[3–5], indicating that the SC has a causal role. In a recent study using pharmacologic inactivation of the SC, monkeys had to report the direction of motion in a stimulus at a cued location, while ignoring equivalent motion in an irrelevant 'foil' stimulus located elsewhere[4]. After SC inactivation, the animals showed profound deficits in visual attention: they largely failed to report the direction of motion of the cued stimulus when it was placed in the part of the visual field affected by SC inactivation, and instead reported the direction of motion of the foil stimulus. Activity in the SC is therefore not simply updated about visual attention but seems to be necessary for its normal operation.

Previous studies have generally assumed that the SC plays a part in attention by influencing the well-known mechanisms in the visual cortex[3,5]. If so, then disrupting visual attention by inactivating the SC should change attention-related effects in the visual cortex. We tested this hypothesis by recording the activity of single neurons in the middle temporal area (MT) and medial superior temporal area (MST)—two cortical visual areas well known for their roles in processing motion signals[15] and their modulation by visual attention[16]—while monkeys performed a motion-change-detection task. We measured how neuronal activity was modulated by spatial cues before and during temporary pharmacological inactivation of SC. Contrary to the hypothesis, we found that attention-related effects in MT and MST remained intact even though SC inactivation caused major deficits in the visual attention task.

Two monkeys (J and M) performed a motion-detection task in which they were rewarded for pressing a button when they correctly detected a change in the direction of motion of the stimulus at the cued location and ignored changes in the direction of motion of a foil stimulus located diagonally opposite the cued stimulus (Fig. 1a). In trials in which the change occurred in the cued stimulus, the animals pressed the button correctly in about 50–60% of the trials (Fig. 1c, pre-injection 'hit rates' were $53 \pm 26\%$ for J and $57 \pm 21\%$ for M). Conversely, they correctly refrained from responding in most of the trials in which the change occurred in the distracter stimulus (pre-injection 'false alarm' rates were $9 \pm 15\%$ for J and $9 \pm 7\%$ for M).

To test the effects of SC inactivation on attention and sensory cortex activity during this task, we injected muscimol, a GABA$_A$ (γ-aminobutyric acid type A) agonist, in the intermediate and deep layers of the SC (Fig. 1b). The extent of the neuronal inhibition caused by the injection was assessed at the beginning and end of each session, by measuring eye peak velocity during visually guided saccades[17]. Each session included two data-collection phases, one before and one during SC inactivation.

Consistent with previous results[4], we found that SC inactivation caused large and spatially specific deficits in the ability of the animal to detect changes in the cued stimulus, with post-injection hit rates dropping to about 10–15% in the part of the visual field affected by SC inactivation (Fig. 1 c–e and Supplementary Information). We then tested whether SC inactivation induced comparable changes in the cue-related modulation of activity in MT and MST.

Neurons were recorded in either the MT or MST area while the monkeys performed the task, during the same behavioural sessions documented above. The location and direction of motion of the stimuli were based on the tuning properties of the neurons, and the size of the motion patch was adjusted to the size of the receptive fields (see Methods). In brief, either the cued or the foil stimulus was placed in the receptive field of the neuron under study, and the direction of motion on each trial was set as the preferred or anti-preferred direction of the neuron, and was always opposite in the two stimulus patches. We recorded a total of 69 MST (monkey J, 31; monkey M, 38) and 44 MT (J, 34; M, 10) neurons before inactivation and 77 MST (J, 26; M, 51) and 55 MT (J, 47; M, 8) neurons during inactivation. Some of these neurons were isolated continuously throughout the experiment ($n = 36$ cells for MST and $n = 18$ cells for MT). We provide additional analyses for this particular set of neurons in Supplementary Information.

Before SC inactivation, as expected from previous studies demonstrating attention-related modulation of visual responses in MST and MT[16], we found that neurons recorded in MST (Fig. 2c) and MT (Fig. 2g) showed higher discharge rates when the motion stimulus in their receptive field was cued ('cue in') than when it was not cued ('cue out'). As in previous studies, we quantified this modulation by

[1]Institute of Neuroscience, Université Catholique de Louvain, 1200 Brussels, Belgium. [2]Systems Neurobiology Laboratory, Salk Institute for Biological Studies, 10010 North Torrey Pines Road, La Jolla, California 92037, USA. [3]Laboratory of Sensorimotor Research, National Eye Institute, Bethesda, Maryland 20892, USA.
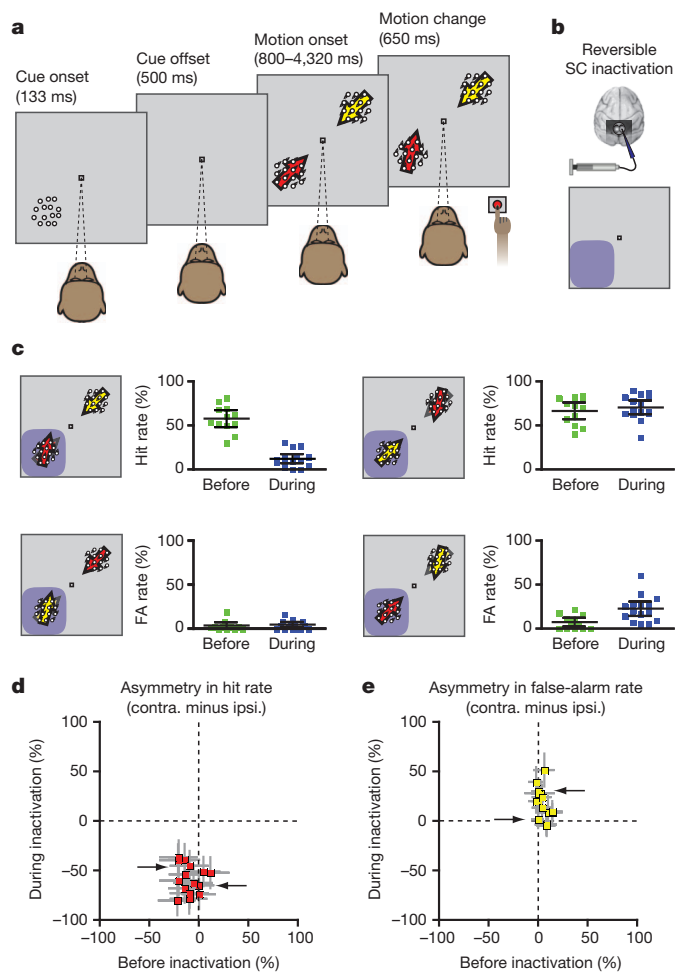
Figure 1 | Task design and behavioural performance. a, After a brief static cue, two motion stimuli moving in opposite directions were displayed in diagonally opposite locations. After a variable delay, the motion direction of one of the stimuli changed slightly. The monkey had to press a button when the change occurred at the cued location. **b**, We recorded single neurons in the MT or MST area after we injected muscimol into the intermediate and deep layers of the SC. The extent of the effect of the inactivation was assessed by mapping saccade velocities across the visual field. The affected part of the visual field is shown here schematically in blue. **c**, Response rates for changes at cued location (top) and un-cued location (bottom), before (green) and during (blue) SC inactivation. The red arrow denotes the cued patch and the yellow arrow denotes the un-cued patch. The affected part of the visual field is illustrated by blue shading. Error bars indicate 95% confidence intervals of the mean. FA, false alarm. **d**, **e**, Difference in cued change-detection rate (red) and false-alarm rate for un-cued motion changes (yellow) between the sides contralateral (contra.) and ipsilateral (ipsi.) to the injection before (x axis) and during (y axis) SC inactivation. Each dot corresponds to a different experiment and the grey lines show the 95% confidence interval (the computation of which is based on a method described in ref. 29). The arrows point to the data corresponding to the two sample experiments shown in Fig. 2.

measuring the discharge rate during the delay period of the task (300–800 ms after motion stimuli onset), and computed a modulation index, defined as the difference in discharge rates between cue in and cue out conditions, divided by their sum. For the two sample neurons shown in Fig. 2, the modulation indexes were 0.16 and 0.07 for the MST and MT neurons, which corresponded to increases in the discharge rate of 39% and 15%, respectively.

During SC inactivation, this modulation was intact. Neurons in MST (Fig. 2d) and MT (Fig. 2h) continued to show higher discharge rates for the motion stimulus in their receptive field when it was cued than when it was not cued. The post-injection modulation indexes
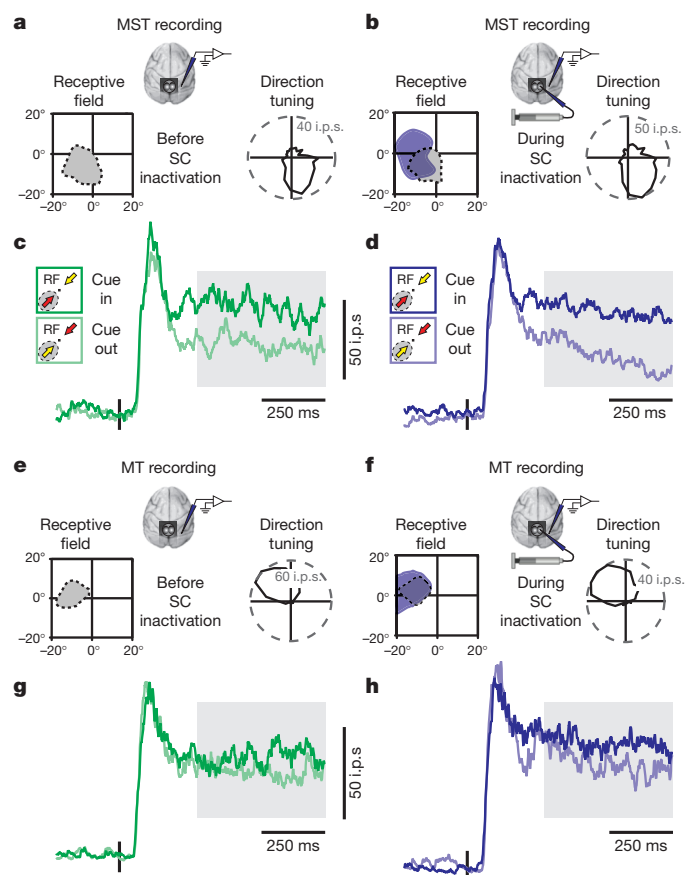

Figure 2 | Sample neuronal activity before and during SC inactivation. a, **b**, **e**, **f**, Receptive field and tuning properties of a sample MST (**a**, **b**) and MT (**e**, **f**) neuron recorded both before (**a**, **e**) and during (**b**, **f**) SC inactivation. The blue shading illustrates the extent of the effect of the muscimol injection in these experiments, on the basis of saccade velocities. **c**, **d**, **g**, **h**, Response of the same sample MST (**c**, **d**) and MT (**g**, **h**) neurons before (**c**, **g**, in green) and during (**d**, **h**, in blue) SC inactivation, for trials in which the cued patch was in (darker line) or out (lighter line) of the receptive field (RF). The vertical lines mark the onset of the motion stimuli. The grey box illustrates the time period used to compute the cue-related modulation analyses. ips, impulses per second.

were 0.21 and 0.08 for the MST neuron and MT neuron, respectively, which were not significantly different from their pre-injection values, but remained significantly greater than chance (both $P < 0.0001$, Wilcoxon rank-sum test, cue in versus cue out). This cue-related modulation in discharge rate was intact, despite the deficits in detection performance observed simultaneously during the SC inactivation (Fig. 1d).

To quantify the effect of SC inactivation across our population, we measured a modulation index for each neuron before and during inactivation. Pre-injection, the average modulation index in our sample of neurons was 0.075 ± 0.029 (mean $\pm$ 95% confidence interval; median, 0.051) in MST and 0.061 ± 0.023 in MT (median, 0.048; significantly greater than zero, Wilcoxon signed-rank test, all $P < 0.001$) (Fig. 3a) corresponding to average increases in the discharge rate of 24% and 15%, respectively. Post-injection, the average modulation index was 0.071 ± 0.025 (median, 0.048) in MST and 0.057 ± 0.022 in MT (median, 0.041) (Fig. 3a); these values remained significantly greater than zero (Wilcoxon signed-rank test, all $P < 0.001$), and were not different from the values before inactivation (Wilcoxon rank-sum test, $P > 0.5$; Bayesian posterior probability of the null (no-change) hypothesis ($p(H_0)$), MST, 0.99; MT, 0.985). Thus, SC inactivation produced no appreciable change in the cue-related modulation of the average discharge rate across our sample of MST and MT neurons. Similar
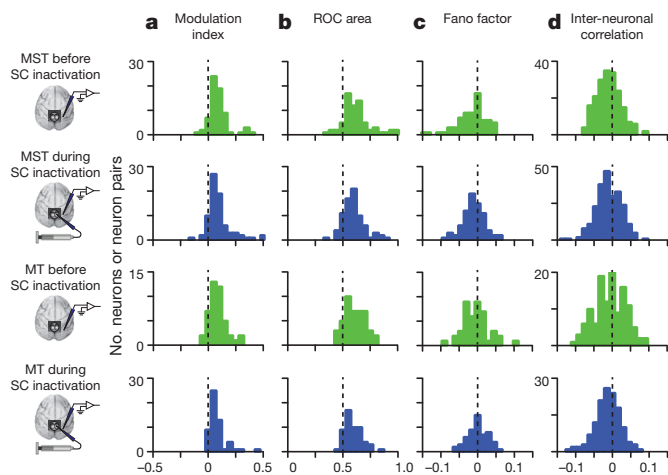
Figure 3 | Population results before and during SC inactivation.
a–d, Distribution of modulation indices (a), area under ROC curves (b), Fano factor indices (c) and difference in interneuronal correlations (d) during the delay period, before (green) and during (blue) SC inactivation for all MST and MT neurons.

results were found when the non-preferred stimulus was presented inside the receptive field (Supplementary Information).

We considered whether SC inactivation might have altered other aspects of cue-related changes in MST and MT neuronal activity. Although modulation of average discharge rate is the standard method for documenting attention-related changes in neuronal activity, it does not measure how noise or variability of discharge rate might change with attention.

To address this point, we computed three additional values for each neuron. First, we computed the area under the receiver operating characteristic (ROC) curve[18], which indicates how well an ideal observer could classify the condition based on the activity of the neuron; in our case, whether the cued or un-cued stimulus was in the receptive field of the neuron. Second, we computed the Fano factor (the ratio of the variance over the mean of the response), which has been found to be lower for cued stimuli than for un-cued stimuli[19], indicating that attention decreases the variability of neuronal activity. Third, we computed the noise correlation between pairs of simultaneously recorded neurons, which has recently been found to decrease with attention[20,21], improving the signal-to-noise ratio of visual signals across the population of neurons.

These additional measurements were also unchanged by SC inactivation. The ROC areas were significantly higher than chance (Fig. 3b), both before and during SC inactivation in both MST and MT (Wilcoxon signed-rank test, all $P < 0.001$), and were not changed by SC inactivation (Wilcoxon rank-sum test, MST, $P = 0.30$; MT, $P = 0.28$; Bayesian $p(H_0)$, MST, 0.978; MT, 0.981); this result indicates that the ability of an ideal observer to discriminate the cued location was unchanged by SC inactivation.

The Fano factor index was significantly less than zero (Fig. 3c), both before (Wilcoxon signed-rank test, MST, $P < 0.0001$; MT, $P = 0.007$) and during (MST, $P < 0.0001$; MT, $P = 0.04$) inactivation, and not different from each other (MST, $P = 0.66$; MT, $P = 0.23$; Bayesian $p(H_0)$, MST, 0.988; MT, 0.979); this result shows that the variability in the discharge rate was reduced by spatial cueing both before and during SC inactivation.

The change in interneuronal correlation was significantly less than zero (Fig. 3d), both before (MST, $P < 0.0001$; MT, $P = 0.0001$) and during (MST, $P = 0.0001$; MT, $P = 0.0005$) inactivation, and not different from each other (MST, $P = 0.76$; MT, $P = 0.71$; Bayesian $p(H_0)$, MST, 0.996; MT, 0.995); this result indicates that spatial cues reduced the correlation in activity between neurons, and this reduction was unchanged by SC inactivation. Similar findings were made with a

wide range of bin sizes used to compute the correlations (Supplementary Information).

Finally, we examined cue-related modulations in neuronal activity during other intervals in the task as well as changes in neuronal activity unrelated to the cue, and these were also unchanged during SC inactivation (Supplementary Information). We also confirmed that neuronal activity in the parts of MST we recorded were indeed necessary for the performance of the attention task (Supplementary Information).

In summary, we found that during SC inactivation, the enhanced responses of neurons in the visual cortex to attended stimuli were preserved despite large behavioural impairments in a covert attention task. This result was found in two visual areas well known for their roles in processing motion signals[15] and their modulation by visual attention[16]. Moreover, the attention deficit induced by SC inactivation not only preserved the cue-related changes in visual responses, but it also left intact the other known correlates of attention in the visual cortex: the ability of neurons to discriminate cued from un-cued spatial locations, the reliability of neuronal discharge (that is, Fano factor) and cue-related changes in noise correlations between neurons. These effects cannot be explained by a sensory impairment, because previous studies have shown that attention deficits during SC inactivation are not caused by changes in local motion perception[4]. The effects also cannot be explained by a motor deficit, because the single-button response in our task was unimpaired for stimuli outside the affected region of the visual field (Fig. 1c).

These findings demonstrate that the known modulations of activity in the visual cortex are not the only mechanisms involved in the control of attention and that other processes must have a key role. One possibility is that visual attention involves other aspects of neuronal activity in these same visual areas. For example, although we found no changes in correlations between nearby neurons, there could be changes between more distant sites or across different areas. A second possibility is that the crucial steps take place in other brain areas entirely, for example, in the parietal or prefrontal cortex[22], the SC or the basal ganglia[23]. In particular, the frontal eye fields (FEF) exert effects on attention qualitatively similar to the SC[24,25]. However, because of prominent feedback from FEF to the visual cortex, SC-induced changes in FEF might have been expected to also change responses in the visual cortex. Finally, it is possible that distinct circuits mediate different aspects of attention. For example, changes in the visual cortex might be important for feature-based attention[26] and for regulating the perceptual appearance of stimuli[27], whereas the mechanism targeted by SC inactivation is important for the all-or-none aspects of spatial attention (for example, change blindness[28]).

METHODS SUMMARY

1. Desimone, R. & Duncan, J. Neural mechanisms of selective visual attention. *Annu. Rev. Neurosci.* **18,** 193–222 (1995).
2. Reynolds, J. H. & Chelazzi, L. Attentional modulation of visual processing. *Annu. Rev. Neurosci.* **27,** 611–647 (2004).
3. Müller, J. R., Philiastides, M. G. & Newsome, W. T. Microstimulation of the superior colliculus focuses attention without moving the eyes. *Proc. Natl Acad. Sci. USA* **102,** 524–529 (2005).
4. Lovejoy, L. P. & Krauzlis, R. J. Inactivation of primate superior colliculus impairs covert selection of signals for perceptual judgments. *Nature Neurosci.* **13,** 261–266 (2010).
5. Cavanaugh, J. Subcortical modulation of attention counters change blindness. *J. Neurosci.* **24,** 11236–11243 (2004).
6. Moran, J. & Desimone, R. Selective attention gates visual processing in the extrastriate cortex. *Science* **229,** 782–784 (1985).
7. Treue, S. Neural correlates of attention in primate visual cortex. *Trends Neurosci.* **24,** 295–300 (2001).
8. Roelfsema, P. R., Lamme, V. A. & Spekreijse, H. Object-based attention in the primary visual cortex of the macaque monkey. *Nature* **395,** 376–381 (1998).
9. Corbetta, M. & Shulman, G. L. Control of goal-directed and stimulus-driven attention in the brain. *Nature Rev. Neurosci.* **3,** 201–215 (2002).
10. Kustov, A. A. & Robinson, D. L. Shared neural control of attentional shifts and eye movements. *Nature* **384,** 74–77 (1996).
11. Ignashchenkova, A., Dicke, P. W., Haarmeier, T. & Thier, P. Neuron-specific contribution of the superior colliculus to overt and covert shifts of attention. *Nature Neurosci.* **7,** 56–64 (2003).
12. O'Connor, D. H., Fukui, M. M., Pinsk, M. A. & Kastner, S. Attention modulates responses in the human lateral geniculate nucleus. *Nature Neurosci.* **5,** 1203–1209 (2002).
13. Bender, D. B. & Youakim, M. Effect of attentive fixation in macaque thalamus and cortex. *J. Neurophysiol.* **85,** 219–234 (2001).
14. Robinson, D. L. & Petersen, S. E. The pulvinar and visual salience. *Trends Neurosci.* **15,** 127–132 (1992).
15. Rudolph, K. Transient and permanent deficits in motion perception after lesions of cortical areas MT and MST in the macaque monkey. *Cereb. Cortex* **9,** 90–100 (1999).
16. Treue, S. & Maunsell, J. H. R. Attentional modulation of visual motion processing in cortical areas MT and MST. *Nature* **382,** 539–541 (1996).
17. Hafed, Z. M., Goffart, L. & Krauzlis, R. J. Superior colliculus inactivation causes stable offsets in eye position during tracking. *J. Neurosci.* **28,** 8124–8137 (2008).
18. Hanley, J. A. & McNeil, B. J. The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology* **143,** 29–36 (1982).
19. Mitchell, J. F., Sundberg, K. A. & Reynolds, J. H. Differential attention-dependent response modulation across cell classes in macaque visual area V4. *Neuron* **55,** 131–141 (2007).
20. Cohen, M. R. & Maunsell, J. H. R. Attention improves performance primarily by reducing interneuronal correlations. *Nature Neurosci.* **12,** 1594–1600 (2009).
21. Mitchell, J. F., Sundberg, K. A. & Reynolds, J. H. Spatial attention decorrelates intrinsic activity fluctuations in macaque area V4. *Neuron* **63,** 879–888 (2009).
22. Moore, T. The neurobiology of visual attention: finding sources. *Curr. Opin. Neurobiol.* **16,** 159–165 (2006).
23. Redgrave, P., Prescott, T. J. & Gurney, K. The basal ganglia: a vertebrate solution to the selection problem? *Neuroscience* **89,** 1009–1023 (1999).
24. Moore, T. & Armstrong, K. M. Selective gating of visual signals by microstimulation of frontal cortex. *Nature* **421,** 370–373 (2003).
25. Wardak, C., Ibos, G., Duhamel, J.-R. & Olivier, E. Contribution of the monkey frontal eye field to covert visual attention. *J. Neurosci.* **26,** 4228–4235 (2006).
26. Treue, S. & Martínez Trujillo, J. C. Feature-based attention influences motion processing gain in macaque visual cortex. *Nature* **399,** 575–579 (1999).
27. Carrasco, M., Ling, S. & Read, S. Attention alters appearance. *Nature Neurosci.* **7,** 308–313 (2004).
28. Rensink, R. A., O'Regan, J. K. & Clark, J. J. To see or not to see: the need for attention to perceive changes in scenes. *Psychol. Sci.* **8,** 368–373 (1997).
29. Ross, T. D. Accurate confidence intervals for binomial proportion and Poisson rate estimation. *Comput. Biol. Med.* **33,** 509–531 (2003).

METHODS

Monkey preparation. We performed MT and MST neuronal recordings and reversible inactivation of the intermediate and deep layers of the SC in two adult rhesus monkeys (subjects J and M) that were 12–16 years of age and weighed 14–16 kg. The monkeys were prepared using standard surgical techniques described in detail in ref. 17. All experimental protocols were approved by the Institutional Animal Care and Use Committee and complied with US Public Health Service policy on the humane care and use of laboratory animals. The laboratory set-up for behavioural control and monitoring was identical to that described in ref. 17.

Attentional task. Trials began with the appearance of a central dot on which the monkey had to fixate during the whole trial duration. Achievement of fixation triggered the display of a peripheral stimulus, the cue, consisting of a 5–7°-wide patch of static dots. The actual size of the patch was chosen as not to exceed the size of the receptive fields of the neurons being recorded. On each trial, the cue could be displayed at one of two possible locations, chosen randomly. One of these locations was chosen to be in the centre of the receptive fields of the recorded neurons and the other one was the symmetric location across the fixation point. The cue was displayed for 133 ms and was followed by a 500-ms delay, during which only the fixation point was displayed. Two patches of moving dots were then displayed at the two previously described locations. The dots were moving in opposite directions in the two patches, one of which being the preferred direction of the neurons being recorded. The characteristics of the stimulus have been described elsewhere[4]. In the present case, the dots had an eight-frame lifetime (corresponding to 107 ms). The direction of motion of each dot was drawn from a normal distribution centred on the direction of motion of the patch and with a 16° standard deviation.

The direction of motion of the patches remained constant for 800 ms plus a geometrically distributed delay of mean 480 ms (range, 0–3520 ms). This distribution allowed the hazard function to remain flat during the delay. After this delay, the direction of motion of one of the patches changed. The monkey had to press a button whenever the change in direction occurred at the previously cued location. The change varied from 16° to 20° and was adjusted on the basis of the performance of the monkey at the beginning of each session to keep a global performance of about 75%.

After the beginning of the change in direction, stimuli remained on the screen for 650 ms or until the response of the animal. Monkeys received a liquid reward only for correct responses in completed trials (button press after change occurred at cued location or absence of response when no change occurred or change occurred at un-cued location). If the monkey broke fixation midtrial, the trial was aborted and repeated later in the session. This paradigm has been referred to as a 'filtering' task because it requires the monkey to actively ignore stimulus changes at the un-cued location. The advantage of this task design is that correct performance requires the filtering out of signals from irrelevant distracter stimuli. This paradigm is similar to that used originally to demonstrate attentional modulation in areas MT and MST[19] and more recently to show a causal role of the SC in the control of spatial attention[4]; it is also similar to that described in ref. 30.

All stimuli were displayed on a cathode ray tube display with a refresh rate of 75 Hz. The background luminance of the monitor was 14 cd m^{-2}. Luminance of the fixation dot and of each dot in the patches was 50 cd m^{-2}. Subjects pushed buttons mounted on a button box at waist level within easy reach of the left hand. Each subject used only its left hand to push buttons.

Procedure. At the beginning of each inactivation session, we lowered a recording tetrode in a track selected on the basis of previous recording sessions. After identification of a good recording spot for MT and MST neurons, we mapped the receptive fields (see examples in Fig. 2a, b, e, f; 50–80 trials) and motion-direction tuning properties (Fig. 2a, b, e, f; 30–60 trials) of the isolated neurons and recorded them during performance of the attentional task (232–366 trials). After completion of the pre-injection data collection, an injectrode, the tip of which was previously sitting above the quadrigeminal cistern, was lowered into the intermediate and deep layers of the SC and muscimol was injected as per the procedure described in ref. 4. After the injection, the extent of the effect of inactivation was evaluated by measuring eye peak velocity during visually guided saccades (60–120 trials).

By carefully choosing the injection site on the basis of exploratory recordings, by adjusting the volume of muscimol injected (between 0.4 and 0.6 μl) and the orientation of the bevel of the injection cannula, we were able to localize the affected region of visual space such as to encompass in all experiments the contralateral visual-stimulus location used during the attentional task.

The affected part of the visual field was defined as the portion of space where velocities were inferior to the lower bound of the confidence interval ($\alpha = 0.05$) of the baseline velocities. Next, the MT and MST neurons were recorded again during the receptive field and tuning-mapping procedures and during the attentional task. The pre-injection part, including isolation and mapping of the MT and

MST neurons, lasted for 1.5–2.5 h, the lowering of the injectrode and the injection lasted together about 4 min and the post-injection part lasted between 1 and 2 h. The duration of a whole session lasted between 3.5 and 4.5 h. There was a total of 12 successful sessions with SC inactivation combined with recordings before and during SC inactivation.

For the MST injection experiment (Supplementary Fig. 3), we first lowered the injectrode to a depth previously recognized as being 500 μm above the lower limit of MST. We then injected a first muscimol dose of 0.5 μl, moved the injectrode up 500 μm and injected again, and so forth up to the upper limit of the area. This led to a total of four injections.

Behavioural analysis. Performance in the task was evaluated by the number of correct and incorrect trials (binomial variable) in each condition (ipsilateral versus contralateral and before versus during inactivation). The statistical tests used to assess the significance of the behavioural change induced by the inactivations were logistic regressions, with each condition and their interaction used as categorical predictor variables. Inactivations were considered as having a significant effect when the P value for the interaction between the conditions ipsilateral versus contralateral and before versus during was inferior to 0.05. When conducting these analyses on all sessions together, subject identity was added as a random categorical predictor to take into account repeated measurements.

Neuronal recordings. Recordings were conducted with a tetrode (Thomas Recording GmBH). Neuronal signals were amplified, band-pass filtered and digitized (Plexon recording system). Neurons were isolated during the experiment to allow for online mapping of their receptive fields and motion-direction tuning properties. In parallel, all waveforms passing a manually set threshold were stored for offline sorting. Offline sorting was conducted first automatically (Klustakwik sorting algorithm[31]) and was then refined manually. On average, we recorded 7.5 neurons per experimental session.

For inactivation experiments, the four-channel waveforms and interspike interval distributions of each neuron isolated before muscimol injection was correlated with the waveforms and interspike interval distribution of each neuron isolated after injection[32]. We then used these correlation values to identify the neurons that were putatively the same before and during inactivation (see also Supplementary Information).

Motion-direction tuning and receptive-field mapping. After isolation of the neurons, the motion-direction tuning of the cells was first evaluated, following a procedure similar to that described in ref. 33. In brief, the monkey had to fixate on a central dot while a whole-screen patch of dots was moving coherently in a direction changing on every frame, leading to a circular motion. The direction of rotation (clockwise or anticlockwise) was selected randomly on every trial. The response of the isolated neurons as a function of the direction of motion of the patch was used to determine their preferred direction of motion.

After the direction-tuning procedure, the receptive fields of the neurons were assessed. The monkey had to fixate on a central dot while patches of dots moving coherently in the preferred direction of motion of the cells were displayed in quick succession at locations selected randomly from a grid encompassing the whole screen. Typically, 48 different locations were probed.

Bayesian analysis. In order to estimate the probability of an absence of difference between the pre- and post-injection data, we computed the Bayes factor for the comparison between a model assuming a change in mean value during inactivation and a model assuming no change (H_0). When necessary, data were transformed to achieve a normal distribution. We computed the Bayes factor by means of different methods: fractional Bayes factor[34], Bayesian information criterion[35] and Bayesian t-test based on the Savage–Dickey ratio test[36]. These different methods provided comparable results. We mention in the main text only the $p(H_0)$ computed with the fractional Bayes factor method.

Interneuronal correlations. Interneuronal correlations were computed following the same procedure as described in ref. 21. In brief, the delay period (between 300 and 800 ms following stimuli onset) was divided into non-overlapping bins (4, 6, 7, 11, 16, 22, 31, 45, 63, 83, 125, 250 or 500 ms long) in which spike counts were computed. The average spike count in each bin was subtracted out from the spike-count values to remove any stimulus-locked response variation. Similarly, the slow variation in discharge rate over consecutive trials was also removed by subtracting the Gauss-weighted smoothing of spike-count changes (σ = five trials). Pearson correlations were computed for all pairs of units having a minimum discharge rate of five spikes per second (MT before inactivation, 122 pairs; MT during, 134 pairs; MST before, 194 pairs; MST during, 235 pairs).

We then estimated the effect of attention on interneuronal correlations by computing the difference in correlations (cue in minus cue out) for MST and MT. These differences are shown in Supplementary Material for all bin sizes. To illustrate these results in the main article, we chose a bin size of 31 ms (shown in Fig. 3), on the basis of the timescale of interneuronal correlations estimated in ref. 37. Because the spike counts obtained with this bin size were not always

normally distributed, we also performed the same analysis using non-parametric Spearman correlations and obtained similar results.

30. Palmer, J. & Moore, C. M. Using a filtering task to measure the spatial extent of selective attention. *Vision Res.* **49,** 1045–1064 (2009).
31. Harris, K. D., Henze, D. A., Csicsvari, J., Hirase, H. & Buzsáki, G. Accuracy of tetrode spike separation as determined by simultaneous intracellular and extracellular measurements. *J. Neurophysiol.* **84,** 401–414 (2000).
32. Dickey, A. S., Suminski, A., Amit, Y. & Hatsopoulos, N. G. Single-unit stability using chronically implanted multielectrode arrays. *J. Neurophysiol.* **102,** 1331–1339 (2009).
33. Schoppmann, A. & Hoffmann, K. P. Continuous mapping of direction selectivity in the cat's visual cortex. *Neurosci. Lett.* **2,** 177–181 (1976).
34. Berger, J. & Pericchi, L. in *Model Selection* Vol 38 (ed. Lahiri, P) 135–207 (Institute of Mathematical Statistics Lecture Notes – Monograph Series, 2001).
35. Wagenmakers, E. J. A practical solution to the pervasive problems of *p* values. *Psychon. Bull. Rev.* **14,** 779–804 (2007).
36. Wetzelsls, R., Raaijmakers, J. G. W., Jakab, E. & Wagenmakers, E. J. How to quantify support for and against the null hypothesis: a flexible WinBUGS implementation of a default Bayesian *t* test. *Psychon. Bull. Rev.* **16,** 752–760 (2009).
37. Bair, W., Zohary, E. & Newsome, W. T. Correlated firing in macaque visual area MT: time scales and relationship to behavior. *J. Neurosci.* **21,** 1676–1697 (2001).