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Recasting the Smooth Pursuit Eye Movement System

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Krauzlis, Richard J. Recasting the smooth pursuit eye movement system. *J Neurophysiol* 91: 591–603, 2004; 10.1152/jn.00801.2003. Primates use a combination of smooth pursuit and saccadic eye movements to stabilize the retinal image of selected objects within the high-acuity region near the fovea. Pursuit has traditionally been viewed as a relatively automatic behavior, driven by visual motion signals and mediated by pathways that connect visual areas in the cerebral cortex to motor regions in the cerebellum. However, recent findings indicate that this view needs to be reconsidered. Rather than being controlled primarily by areas in extrastriate cortex specialized for processing visual motion, pursuit involves an extended network of cortical areas, and, of these, the pursuit-related region in the frontal eye fields appears to exert the most direct influence. The traditional pathways through the cerebellum are important, but there are also newly identified routes involving structures previously associated with the control of saccades, including the basal ganglia, the superior colliculus, and nuclei in the brain stem reticular formation. These recent findings suggest that the pursuit system has a functional architecture very similar to that of the saccadic system. This viewpoint provides a new perspective on the processing steps that occur as descending control signals interact with circuits in the brain stem and cerebellum responsible for gating and executing voluntary eye movements. Although the traditional view describes pursuit and saccades as two distinct neural systems, it may be more accurate to consider the two movements as different outcomes from a shared cascade of sensory–motor functions.

INTRODUCTION

Why do we move our eyes at all? As with most animals with frontally directed eyes, our retinas contain a specialized central area with an especially high density of photoreceptors. To see things clearly, we continuously regulate the orientation of our eyes so that the images of interesting objects are projected on or near this part of the retina. We and other primates accomplish this using two types of eye movements. Saccades are discrete movements that quickly change the orientation of the eyes, thereby translating the image of the object of interest from an eccentric retinal location to the fovea. Smooth pursuit is a continuous movement that slowly rotates the eyes to compensate for motion of the visual object, minimizing blur that would otherwise compromise visual acuity.

Smooth pursuit is primarily driven by visual motion (Rashbass 1961), whereas saccades can be guided by a wide variety of signals, both real and imagined. This property of pursuit has been used to great advantage to examine the visual processing of motion, but it has also promoted the view that pursuit lacks many interesting properties associated with saccades. The standard pathways for the two types of movements also illustrate this apparent difference between pursuit and saccades. For pursuit, these pathways compose a seemingly simple circuit connecting areas in the temporal and frontal lobes of the cerebral cortex with pursuit-related motor regions of the cerebellum (Fig. 1*A*), as detailed in several previous reviews (Ilg 1997; Keller and Heinen 1991; Lisberger et al. 1987). The middle temporal (MT) and medial superior temporal (MST) areas in the superior temporal sulcus process visual motion and oculomotor signals that are typically required for pursuit, and these are conveyed to the flocculus and ventral paraflocculus (VPF) in the cerebellum via visuomotor nuclei in the pontine nuclei (PON), primarily through the dorsolateral pontinue nucleus (DLPN). These cerebellar regions access the output motor nuclei for the eye muscles by projections to the vestibular nucleus (VN). In parallel with this pathway, a second cortico–ponto–cerebellar pathway originates in the frontal eye field (FEF) and continues through the nucleus reticularis tegmenti pontis (NRTP), which, like the DLPN, provides outputs exclusively to the cerebellum, in this case lobules VI and VII of the vermis (VERM).

For saccades, the standard descending pathways likewise contain cortico–ponto–cerebellar connections like those for pursuit, but the primary circuit emphasizes several additional routes not imparted to the pursuit system (Fig. 1*B*) (Munoz 2002; Sparks 2002). These include direct projections from cortical eye fields to eye-movement–related structures in the brain stem such as the superior colliculus (SC) and premotor including nuclei in the reticular formation (PMN), and also pathways through the basal ganglia, including the caudate nucleus (CN) and the substantia nigra pars reticulata (SNr). Thus, whereas the standard pathways for pursuit simply link visual sensory areas to the cerebellum, consistent with pursuit's presumed role as a visuomotor reflex, the pathways for saccades include some degree of direct cortical control over the motor output, as befits a voluntary behavior. The saccadic pathways through the SC and the cerebellum provide additional levels of control, associated with triggering and shaping the motor commands, respectively.

The main goal of this review is to recast the traditional view of the pursuit system. The first part will consider the pieces of the pursuit system, reevaluating the role of established players and also introducing new pieces that have been identified recently. Next we will consider an alternative view of the pursuit system that is predicated on a functional architecture similar to that for saccades. The last part of the article will consider a few of the more salient differences between pursuit and saccades from the viewpoint of this new framework.

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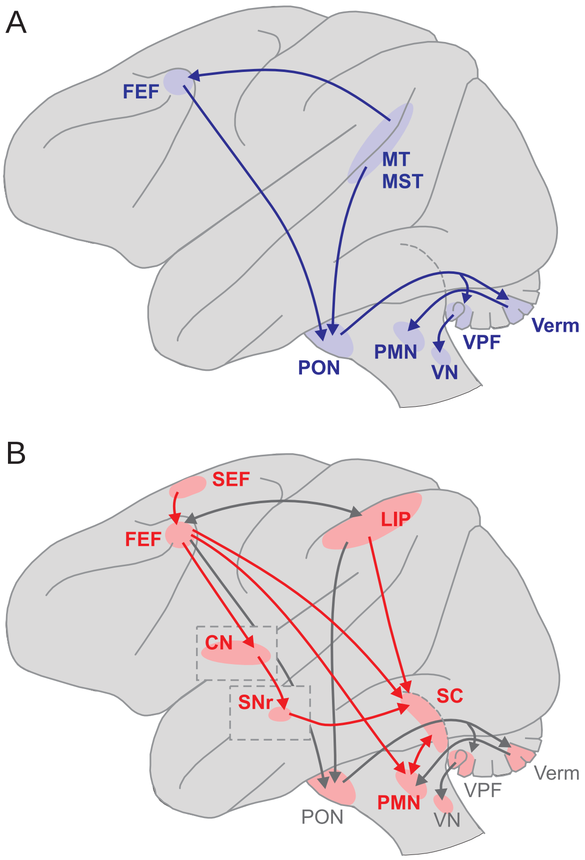

FIG. 1. Outline of the traditional descending pathways for pursuit (*A*) and saccades (*B*). Diagram depicts a lateral view of the monkey brain. Shaded regions indicate specific areas within the cerebral cortex, cerebellum, and brain stem, and arrows indicate the anatomical connections between these areas. Regions demarcated with dashed lines indicate structures normally covered by the cerebral cortex. For clarity, not all relevant areas are depicted and arrows do not always correspond to direct anatomical connections.

PERUSING THE PIECES

MT and MST

The middle temporal (MT) and medial superior temporal (MST) areas are the major source of visual-motion information used to guide pursuit (Dürsteler and Wurtz 1988; Newsome et al. 1985). Recent studies have shown that these motion signals have interesting temporal properties related to features of pursuit. When two spot stimuli cross the receptive field together shortly after their appearance (150 ms), the firing rate of MT and MST neurons and the eye velocity of pursuit are best described by a vector average. However, when they cross the receptive field after a longer delay (450 ms), both firing rate and pursuit shift toward a winner-take-all outcome (Recanzone and Wurtz 1999). With a stimulus consisting of dots moving within a small patch, it is possible to separately control the local motion (by changing the speed of the dots) and the overall rate of displacement (by translating the entire patch); use of this stimulus has shown that the initial pursuit eye acceleration and MT firing rate are determined primarily by the local motion and not by displacement (Priebe et al. 2001). When the local motions can be perceptually grouped as a single moving object, some MT neurons initially respond to the local motion of the stimulus components, but over the course of a few hundred milliseconds they begin to respond to the global motion of the object as a whole. These changes in the directional tuning of the neural activity follow a time course similar to the changes in the direction of pursuit eye velocity (Pack and Born 2001).

The signals in MT and MST are also modulated by attention. When the monkey is presented with a relevant target and an irrelevant distractor stimulus, the initial activity of MT and MST neurons exhibits very little selectivity for the target (Ferrera and Lisberger 1997b; Recanzone and Wurtz 2000). The pursuit eye velocity evoked after comparable short delays mainly follows the average of the two motion signals. The later activity (450 ms after motion onset) of MT and MST neurons exhibits greater selectivity, and the eye movements elicited at these longer latencies selectively follow one or the other stimulus, reflecting a winner-take-all mechanism (Recanzone and Wurtz 2000). However, these changes are relatively small and occur in only a minority of neurons, so it is not clear that these changes are sufficient to account for the selectivity of pursuit.

Although they are often studied together, MT and MST are not homogeneous areas. Area MT neurons respond only when retinal motion is present, whereas some MST neurons maintain their response to object motion even when there is no retinal counterpart (Ilg and Thier 2003; Newsome et al. 1988). Lesions of MT produce retinotopic deficits in the initiation of pursuit eye movements (Newsome et al. 1985); lesions of MST also produce directional deficits that are especially pronounced during maintained pursuit (Dürsteler and Wurtz 1988). These results highlight a general distinction between the two areas—MT is largely involved in pursuit initiation, whereas MST is important for pursuit maintenance. Within MST, the dorsal portion (MSTd) contains neurons with large receptive fields that respond best to large-field stimuli and exhibit an extraretinal input that may be important for compensating for visual motion caused by self-movement (Ilg and Thier 2003; Newsome et al. 1988; Page and Duffy 1999; Shenoy et al. 2002; Upadhyay et al. 2000). Lateral MST (MSTl) contains some units like those in MSTd and also many units with smaller receptive fields that respond only in the presence of a visual stimulus (Newsome et al. 1988). The directional deficit in pursuit associated with damage to MST is primarily related to damage of MSTl rather than MSTd (Dürsteler and Wurtz 1988). Similarly, the most effective microstimulation sites for modifying pursuit are in MSTl; stimulation in MSTd is usually ineffective (Komatsu and Wurtz 1989). These distinctions highlight a conundrum about MST: the subregion most clearly associated with the presence of extraretinal signals (MSTd) is not the same subregion causally associated with pursuit (MSTl).

FEFsem

The smooth eye movement subregion of the frontal eye field (FEFsem) has emerged as the major cortical area involved in the control of pursuit, with effects larger and more immediate than those found for MT and MST. Neurons in the FEFsem exhibit directionally selective responses during pursuit and represent a wide range of preferred directions (MacAvoy et al. 1991; Tanaka and Lisberger 2002b). About half of the neurons discriminate the direction of motion before pursuit onset; the median lead time is approximately 8 ms (Tanaka and Lisberger 2002b). When pursuit targets are placed at more peripheral locations, FEFsem neurons exhibit lesser firing rates at pursuit initiation that are strongly correlated with the behaviorally observed decreases in pursuit acceleration (Tanaka and Lisberger 2002b).

Damage to the FEFsem has dramatic effects on pursuit. Unilateral lesions result in difficulty in generating ipsilateral pursuit, as with MST lesions, but recovery appears to be much slower (Lynch 1987). Inactivation of the FEFsem reduces both initial pursuit acceleration and steady-state velocity to about 25% of its normal value, but produces only minor effects on pursuit latency (Shi et al. 1998). The resulting velocity traces look like scaled-down versions of normal pursuit, arguing that what has been reduced by inactivation is the overall eye velocity command for pursuit. Lesions also abolish the predictive pursuit eye movements that are normally elicited by stimuli with periodic trajectories (Keating 1991, 1993; MacAvoy et al. 1991).

Stimulation of the FEFsem is especially effective in evoking smooth eye movements. Eye movements can be evoked even during fixation with relatively modest currents and at relatively short latencies, within 25–40 ms (Gottlieb et al. 1993; Tanaka and Lisberger 2002a). Microstimulation appears to have two effects on pursuit (Tanaka and Lisberger 2001, 2002a). The first effect is the introduction of a directional signal that drives pursuit eye velocity. The amplitude of this component increases by approximately 60% when applied during pursuit compared with during fixation. A second smaller effect is an increase in the gain of ongoing pursuit eye speed regardless of direction.

Anatomical studies of the FEFsem and the adjacent saccade-related region, FEFsac, have illuminated how pursuit and saccades are represented across areas of the cerebral cortex (Tian and Lynch 1996a,b). The primary inputs to the FEF come from four other areas involved in the control of eye movements: area MST, the lateral intraparietal area (LIP), the supplementary eye field (SEF), and the principal sulcus region (PSR). Importantly, the pursuit- and saccade-related regions within each of these areas are adjacent but are also mostly nonoverlapping. Thus the cortical control of pursuit does not involve wholly different areas, but separate subregions within many of the same areas. Parallel but distinct cortical networks for pursuit and saccades have also been found with functional imaging studies in humans (Petit and Haxby 1999; Rosano et al. 2002).

SEF

The supplementary eye field (SEF), located in the supplementary motor area, appears to play a higher level, and less direct, role in the control of pursuit. Stimulation of the SEF increases pursuit eye velocity, especially when applied during pursuit initiation, but has little effect when applied during fixation (Missal and Heinen 2001; Tian and Lynch 1995). Stimulation of the SEF also delays the occurrence of catch-up saccades (Missal and Heinen 2001), similar to the effect observed in the FEFsem (Tanaka and Lisberger 2002a), presumably because the increase in pursuit velocity reduces the need for saccades. SEF neurons exhibit some preference for the direction of pursuit and also maintain their discharge in the absence of a visual target, indicating that their responses are not wholly visual in origin (Heinen 1995). Their activity tends to be highest when target motion changes, especially when the timing of these changes is predictable (Heinen and Liu 1997). These findings suggest that the SEF might participate in the planning of pursuit eye movements, similar to the role that the supplementary motor area appears to play for other types of movements (Tanji 1996).

LIP

The lateral intraparietal area (LIP) also appears to be involved in the control of both saccades and pursuit, and projections from the FEF indicate that there are separate subregions related to the two types of movements (Tian and Lynch 1996a). Stimulation of LIP can evoke smooth eye movements as well as saccades (Kurylo and Skavenski 1991). Somewhat fewer than half of the neurons in LIP exhibit direction-specific activity during pursuit and continue to fire when the visual stimulus is briefly turned off (Bremmer et al.; Sakata et al. 1983). The pursuit-related activity of many of these neurons is also modulated by eye position and extraretinal signals, suggesting that this area may play a role in representing space in a nonretinopic reference frame (Bremmer et al. 1997; Schlack et al. 2003). The results for pursuit are consistent with other studies indicating that the posterior parietal cortex is a region where a variety of signals converge to represent the spatial goals for movements in coordinate frames appropriate for the effector organs (e.g., eye, head, and hand); these representations are modulated by attention and updated by feedback about ongoing movements (Andersen et al. 1997; Colby and Goldberg 1999).

Basal ganglia

The principle of parallel but distinct pathways for pursuit and saccades extends to circuits involving the basal ganglia and thalamus. Cortical areas such as the FEF project to the substantia nigra pars reticulata (SNr) via the caudate nucleus (CN) of the striatum, and a series of classic studies has shown how tonic inhibition exerted on the superior colliculus (SC) through this pathway (Fig 1*B*) is involved in regulating the triggering of saccades (Hikosaka and Wurtz 1989). Recently, it has been demonstrated that the caudate receives input from the FEFsem, as well as from the FEFsac (Cui et al. 2003). The projections from the two regions are of similar strength, but, as in cortex, they fall into mostly nonoverlapping portions. The caudate has not yet been studied during pursuit, but neurons in the SNr are modulated during pursuit (Pokorny and Basso 2003). The basal ganglia also provide projections back to the FEF via different portions of the thalamus (Tian and Lynch 1997), consistent with the idea that the circuits linking the cortex and basal ganglia consist of a set of parallel and segregated pathways (Alexander et al. 1986). In particular, the newly identified pursuit pathway through the basal ganglia suggests that the oculomotor circuit identified by Alexander et al. (1986) may be further subdivided into pursuit and saccade components.

Pontine precerebellar nuclei

The properties of the dorsolateral pontine nucleus (DLPS) are consistent with a role in conveying signals from areas MT and MST to the cerebellum (Mustari et al. 1988; Suzuki and Keller 1984; Thier et al. 1988). The visual activity is broadly tuned for the direction of motion, has relatively large receptive fields, responds to both large and small stimuli, and has speed preferences similar to those of MT. The pursuit-related activity for many units includes extraretinal signals, and the preferred direction of this activity can be either the same or the opposite as the preferred direction for visual motion. Lesions of the DLPN produce both a retinotopic deficit in the initiation of pursuit and a directional deficit during maintained pursuit (May et al. 1988).

The dorsomedial pontine nucleus (DMPN) also has inputs from the parietal and occipital cortex, but receives an additional input from the FEF. DMPN neurons exhibit directionally selective visual responses and are driven best by large visual stimuli; some units also exhibit pursuit-related activity (Keller and Crandall 1983). At its caudal boundary, the DMPN merges with the nucleus reticularis tegmenti pontis (NRTP). The caudal portion of the NRTP is involved with saccades (Crandall and Keller 1985), but the rostral NRTP is involved in pursuit. Lesions here cause dramatic deficits in pursuit, reducing both the initial eye acceleration and the maintained eye velocity by about 50% (Suzuki et al. 1999). During pursuit, the activity of neurons in the rostral NRTP is primarily related to eye velocity and exhibits broad tuning for direction (Suzuki et al. 2003).

Cerebellar flocculus and ventral paraflocculus

The cerebellar flocculus and ventral paraflocculus (VPF) have long been known to be critical for the generation of pursuit eye movements. Ablation of the flocculus and paraflocculus causes large and lasting deficits in pursuit eye movements (Zee et al. 1981). A recent study involving more selective lesions has shown that the VPF, rather than the flocculus, is the structure critical for the control of pursuit (Rambold et al. 2002).

Several properties highlight the proximity of the VPF to the final output pathways. Microstimulation of the VPF can cause smooth eye movements within 10 ms, reflecting the short length of the downstream output pathways (Belknap and Noda 1987; Lisberger 1994). Purkinje cells (P-cells) in the VPF have firing rates that are proportional to eye speed, and this activity persists when the stimulus is electronically stabilized on the retina, indicating that this activity is related to the motor command (Stone and Lisberger 1990). The preferred directions of VPF P-cells fall into two categories, each roughly aligned with the motion vector for one of the vestibular labyrinths, showing that the pursuit motor command has been converted to a vestibular-based coordinate system (Krauzlis and Lisberger 1996). By receiving a copy of the outgoing pursuit command, the VPF may also form part of a positive-feedback loop that continuously provides a command signal related to the desired eye speed (Lisberger and Fuchs 1978; Miles et al. 1980).

An aspect of VPF function that has recently been reassessed is whether it processes visual motion signals, in addition to motor commands for pursuit. When pursuit velocity increases or decreases, VPF P-cells exhibit overshoots in firing rate; these overshoots have been interpreted as evidence of visual motion signals directly modulating the firing rate of VPF P-cells, presumably the result of visual inputs originating from areas MT and MST and relayed to the VPF by the DLPN (Krauzlis and Lisberger 1991; Stone and Lisberger 1990). This interpretation identifies the VPF as the nexus of the pursuit pathways, because it assigns it the dual functions of both forming the final motor command and updating that command based on visual information. An alternative interpretation is that the overshoots in firing rate are the result of computations performed within the motor pathways for pursuit, rather than visual signals received from the cerebral cortex. According to this view, the transient overshoots in firing rate reflect a calculated attempt by the brain stem and cerebellum to compensate for the sluggish mechanics of the eye muscles and orbital tissues, ensuring that the physical movement of the eyes matches the trajectory specified by descending control signals (Krauzlis 2000; Shidara et al. 1993). This interpretation identifies the VPF as a critical component of the pursuit pathways, but assigns it a role more similar to that given the cerebellum in the saccadic system–an expert regulator of the final motor command that operates under adaptive control.

Several findings argue in favor of the latter view. First, several of the descending control signals for pursuit appear to be related to eye or target velocity, rather than retinal slip. The extraretinal signals observed in area MST indicate that signals related to visual motion and eye speed have already been combined at the level of cortical neurons (Newsome et al. 1988), making it redundant to combine these signals again downstream. Similarly, in the FEFsem, prolonged microstimulation evokes a step change in eye velocity (Tanaka and Lisberger 2002a), suggesting that activation of this area changes the command for pursuit velocity, rather than pursuit acceleration. Second, the VPF is not the primary target of visual motion neurons in the pontine nuclei. The VPF receives only a weak projection from the DLPN; the major target of the DLPN is the dorsal paraflocculus (Glickstein et al. 1994), a structure that has received little attention (Noda and Mikami 1986) and that has connections very different from the VPF. The VPF does have input from the NRTP, but the pursuit-related activity of neurons in the NRTP appears to be related mostly to eye velocity (Suzuki et al. 2003). Third, the output of the VPF does not indicate the presence of visual motion signals. Using a multiple regression technique to test the relationship between the firing rate of VPF P-cells and variables related to the pursuit movement and retinal slip, P-cell activity is best explained by eye position and eye velocity, and not visual signals (Kettner et al. 2002). Similar results have been found for neurons in the medial vestibular nucleus (MVN) that are the targets of projections from the VPF. During pursuit eye movements, the activity of MVN neurons can be described by a combination of resting discharge, eye position, and eye velocity. They exhibit no modulation related to retinal slip, indicating that any antecedent sensory influences have been assimilated into the pursuit motor command (Roy and Cullen 2003).

Cerebellar vermis

Although initially recognized for its role in modulating the motor command for saccades (Noda and Fujikado 1987), the cerebellar "oculomotor" vermis (lobules VI and VII) is also involved in the control of pursuit. P-cells in the vermis respond to a combination of visual, eye velocity, and head velocity signals (Kase et al. 1979; Shinmei et al. 2002; Suzuki and Keller 1988a,b). In contrast to the VPF, many P-cells in the vermis respond to a moving spot stimulus presented during fixation, as well as moving stimuli used to guide pursuit. Many of the neurons in the fastigial nucleus, to which vermal P-cells project, exhibit bursts of activity during the initiation of pursuit

(Fuchs et al. 1994). Accordingly, removal of the vermis or inactivation of the fastigial nucleus alters pursuit, primarily by disrupting the initial acceleration at pursuit onset (Robinson et al. 1997; Takagi et al. 2000). Earlier studies had found that the same manipulations cause marked changes in the trajectories of saccades (Robinson et al. 1993; Takagi et al. 1998). Microstimulation of the vermis can evoke either pursuit or saccadic eye movements depending on the behavioral context, and these effects can be explained by assuming that the vermis and fastigial nucleus regulate a motor drive signal that contributes to both pursuit and saccades (Krauzlis and Miles 1998). These findings support the idea that this region of the cerebellum plays a similar role in the control of pursuit and saccades–it shapes the trajectories of pursuit and saccades, perhaps by modifying the commands that accelerate and decelerate the eye.

Another portion of the cerebellar vermis, the uvula (lobule IX), appears to play an unconventional role in the control of pursuit. It receives strong projections from the visual pontine nuclei, but P-cells in the uvula do not respond particularly well to the moving spot stimuli that are typically used for pursuit, although they do show modulation with sustained full-field motion (Heinen and Keller 1992, 1996). Lesions of the uvula have a unique effect on pursuit–they cause increases in pursuit acceleration for eye movements directed away from the side of the lesion (Heinen and Keller 1992, 1996). The uvula is unlikely to provide a primary drive for pursuit movements, but it may act to compensate for the visual consequences of pursuit.

Pretectal nuclei

The pretectal nucleus of the optic tract (NOT) has a well-established role in the control of horiztonal optokinetic eye movements and might also provide directional signals for pursuit. Although some NOT neurons prefer large-field motion stimuli, others respond to motion within smaller parafoveal receptive fields (Hoffmann and Distler 1989; Mustari and Fuchs 1990). This latter type of neuron also exhibits short-latency (approximately 60 ms) visual responses during smooth pursuit of small visual targets. Disruption of the NOT reduces the velocity of pursuit eye movements, as well as the velocity of short-latency ocular following movements (Ilg et al. 1993; Inoue et al. 2000), a different type of smooth eye movement that rapidly stabilizes gaze when an observer translates through the environment (Miles 1997). The NOT projects to precerebellar nuclei, such as the DLPN, but also directly to eye motor nuclei in the caudal brain stem, such as the MVN and the nucleus prepositus hypoglossi (Mustari et al. 1994). Thus the NOT can influence pursuit indirectly via the cerebellum, but it could also provide a short-latency subcortical pathway that provides visual motion signals directly to the motor output pathways.

SC

Although the superior colliculus (SC) has not traditionally been considered part of the pathways for pursuit, there is now substantial evidence that it plays some role. The intermediate layers of the SC form a retinotopic map for the control of eye and head movements (Sparks 1999; Wurtz and Albano 1980), and neurons in most portions of this map modulate their activity during the preparation and execution of saccades (Glimcher and Sparks 1992; Munoz and Wurtz 1995). In the rostral SC (rSC), corresponding to the representation of the central visual field, many neurons modulate their firing rates during pursuit eye movements as well as during small saccades, and this activity depends on the location of the stimulus vis-à-vis the location of the neuron's response field (Krauzlis et al. 1997, 2000). This activity is not simply a visual response, because it persists in the absence of a visual target (Krauzlis 2001). It also does not appear to convey visual motion information, because it is not directionally selective (Krauzlis 2003a). However, it does exhibit selectivity for stimuli that will be the target of pursuit and saccades, and this selectivity can predict the timing of pursuit and saccade choices (Krauzlis and Dill 2002). It is also modulated by the early removal of a fixated stimulus in the "gap paradigm," providing a neural correlate for the shared effects on pursuit and saccade latencies observed in this paradigm (Krauzlis 2003b). Together, these results suggest that one function of the rSC is to specify the eye movement goal, regardless of the final motor strategy used to acquire the goal, an idea supported by other recent results concerning the SC (Bergeron et al. 2003; Krauzlis and Carello 2003).

It has been further suggested that the same signals in the rSC that are involved in the covert preparation of saccades might also mediate the selection of goals for pursuit (Krauzlis 2003b). In particular, the changes in tonic activity of neurons in the rSC might directly gate the initiation of pursuit, whereas the triggering of saccades requires the recruitment of the saccade-related burst. It is known that activation and inactivation of activity in the rSC modifies the metrics of pursuit, indicating that there is some causal link between rSC activity and pursuit (Basso et al. 2000). The saccade-related function of the SC is somehow linked to pursuit, because saccades evoked by microstimulation of the SC alter the target choice made by the pursuit system (Gardner and Lisberger 2002). A recent report hints that this link occurs at a common preparatory stage. Activating the SC with currents too weak to directly evoke eye movements can nonetheless bias the choices made by both pursuit and saccades (Carello and Krauzlis 2003).

Brain stem premotor nuclei

As with the SC, to which it is intimately related, the deep core of the brain stem has been traditionally associated with the control of saccadic eye movements and not pursuit. However, our understanding of this byzantine brain region is also beginning to change. The primary brain stem nuclei for controlling horizontal and vertical gaze (the PPRF, riMLF, and the cMRF, defined in Table 1) all receive direct inputs from the pursuit subregion of the FEF as well as from the saccade-related subregion (Yan et al. 2000, 2001). This argues strongly that, in addition to the traditional cortico–ponto–cerebellar pathways, there are also direct cortico–bulbar pathways for pursuit.

Now that they are being examined more thoroughly during pursuit, it is becoming clear that subsets of the neurons in these nuclei have pursuit-related, as well as saccade-related activity. In the PPRF, some "burst" neurons are active only during saccades, but a second category of burst neurons is active during both saccades and pursuit (Missal and Keller 2001). Similarly, in the riMLF of the cat, some "burst" neurons fire in relationship to eye velocity not only during saccades, but during pursuit as well (Missal et al. 2000). "Omnipause" neurons (OPNs) exhibit pauses in their tonic activity for saccades in all directions and are believed to act as the final gatekeepers for saccades. Remarkably, about half of the OPNs also show significant decreases in activity during the onset of pursuit—they do not completely stop firing as for saccades, but reduce their activity by about one-third (Missal and Keller 2002). Microstimulation in the region of the OPNs halts saccades, and also strongly decelerates pursuit, although it apparently does not completely stop pursuit (Missal and Keller 2002). Although our understanding of premotor organization is far from complete, these studies indicate that the construction of the motor commands for pursuit and saccades, and also the gating of those commands by inhibitory mechanisms, involves shared circuitry in the brain stem.

DO PURSUIT AND SACCADES HAVE THE SAME FUNCTIONAL ARCHITECTURE?

The picture that emerges from these recent studies (Fig. 2*A*) is quite different from the traditional view of the pursuit system (Fig. 1*A*). In short, the pursuit system has started to look much more like the saccadic system. To summarize, there are several important features of pursuit that suggest a functional homology with saccades.

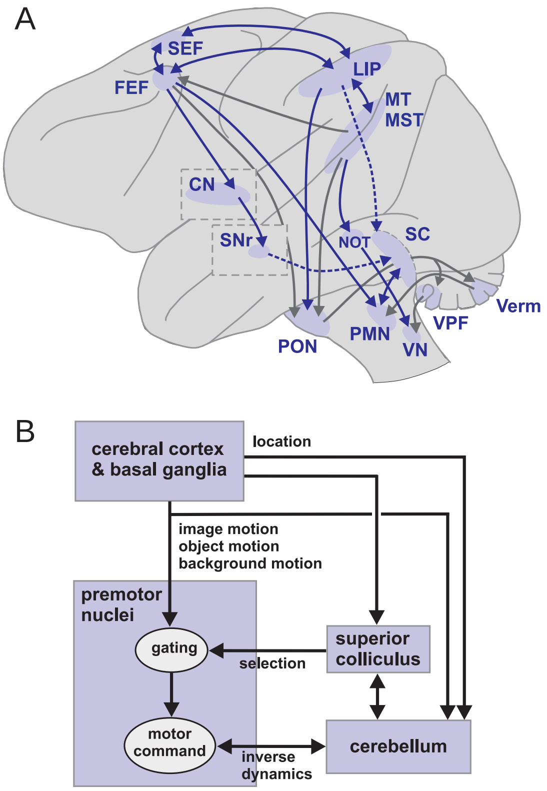

FIG. 2. Revised outline of the descending control pathways for pursuit eye movements. *A*: schematic anatomical diagram of the descending pathways depicted on a lateral view of the monkey brain. Dashed arrows indicate presumed connections; other conventions as in the legend to Fig. 1. *B*: functional diagram of the pathways controlling pursuit. Descending pathways from the cerebral cortex and basal ganglia provide sensory and command signals to the premotor nuclei, superior colliculus, and cerebellum. The premotor nuclei act to gate the descending signals and to construct the motor command. The superior colliculus contributes to the gating of pursuit and the cerebellum adjusts the final motor commands.

First, of the cortical areas involved in pursuit, the FEF appears to exert the strongest control. The traditional view of pursuit emphasizes the role of areas MT and MST, but lesion and stimulation data indicate that activity in the FEFsem has the biggest effects, just as the FEFsac appear to be of particular importance for saccades. This is probably a consequence of the fact that FEFsem has direct access to the premotor nuclei in the brain stem (PPRF, riMLP, and cMRF) as well as projections through the cerebellum, as does the FEFsac.

Second, there are subcortical pathways that can mediate shorter latency responses, and these appear to be under the tutelage of cortical areas. For pursuit, the pretectal nuclei (e.g., NOT) can mediate short-latency smooth eye movements and are influenced by cortical areas MT and MST. For saccades, the SC mediates short-latency saccades, abetted by LIP. The subcortical pathways for pursuit also include feedback loops through the basal ganglia and thalamus, just as is true for saccades.

Third, both pursuit and saccades are gated motor responses that involve a break from ocular fixation. The operation of the gating mechanism for pursuit has long been discussed, but the site of the mechanism is unknown. Based on the partial overlap in the brain stem pathways for pursuit and saccades, we can now speculate that the gating of pursuit involves some of the same players in the premotor nuclei (PPRF, riMLF, cMRF, and OPNs) that regulate the gating of saccades (Fig. 2*B*), but with the circuitry cast into functional states that have not yet been identified.

Fourth, the SC and the FEF may act to regulate the onset of pursuit by influencing the activity of neurons within the premotor nuclei, analogous to the way that these structures regulate the onset of saccades. For saccades, output from the SC not only contributes to gating the movement, but also provides an important drive signal. For pursuit, SC activity may gate the movement (Fig. 2*B*, *selection*), but because the SC conveys spatial rather than directional signals, it probably does little to drive pursuit.

Finally, the cerebellum (VPF, vermis) is an important auxiliary structure that expertly tweaks the pursuit commands–for example, by augmenting the drive signal to compensate for the sluggish mechanics of the eye muscles and orbit (Fig. 2*B*, *inverse dynamics*). Contrary to the traditional view, it does not constitute the only descending pathway, and it may not even be the primary descending pathway–just as the descending commands for saccades are not required to travel through the cerebellum. The dramatic effects of VPF lesions on pursuit might reflect the role of the UPF in the homeostatic regulation of the premotor nuclei, through which a direct pathway for pursuit does appear to pass.

WHAT MAKES PURSUIT DIFFERENT FROM SACCADES?

Despite the possible homology, there are obvious differences between the pursuit and saccadic systems in what they do and in how they operate, and the idea of a common functional architecture can only be a conjecture at this point. However, it now seems probable that there are functional properties that can only be clearly identified and understood by considering the two systems jointly and, in this regard, it may be especially informative to look at properties where the two systems exhibit differences.

Why does pursuit have shorter latencies than saccades?

Pursuit is often described as having much shorter latencies than saccades, 100–125 ms for pursuit compared with 200–250 ms for saccades. If pursuit and saccades are similarly organized, why are their latencies so different? One part of the answer is that the range of latencies for pursuit and saccade latencies are actually largely overlapping. On the one hand, pursuit latencies are not always so short. If the target stimulus is accompanied by a second "distractor" stimulus moving in the opposite direction, then the latencies of pursuit typically increase to 150–200 ms (Ferrera and Lisberger 1995, 1997a; Krauzlis et al. 1999). The competing stimuli need not be moving; the addition of a stationary distractor also increases the latency of pursuit (Knox and Bekkour 2002). On the other hand, saccade latencies are not always so long. The extinction of a fixated stimulus before the appearance of a new stimulus results in the well known "gap effect" for saccades, which consists of a general reduction in the latencies of saccades and the production of some saccades with very short latencies (100 ms or less) referred to as "express" saccades (Fischer and Boch 1983; Fischer and Ramsperger 1984 Fischer et al. 1984; Saslow 1967).

Factors that change the latency of saccades also have similar effects on the latency of pursuit. For example, there is a "gap effect" for pursuit, as well as for saccades (Knox 1996; Krauzlis and Miles 1996a,c; Merrison and Carpenter 1995). The decreases in pursuit and saccade latencies with this paradigm show the same dependence on the duration of the temporal gap, leading to the suggestion that there are common inputs for triggering the two types of eye movements (Krauzlis and Miles 1996c). Subjects that naturally tend to make express saccades in the gap paradigm also exhibit significantly shorter pursuit latencies than other normal subjects (Kimmig et al. 2002).

At least some of the factors that jointly affect pursuit and saccade latencies exhibit a spatial dependence, consistent with the idea that the latencies of pursuit and saccades involve retinotopically organized structures such as the SC. A study comparing the effects of different types of prior information found that spatial cues had a larger effect on the latencies of pursuit and saccades than either color or motion cues (Adler et al. 2002). Even though stimulus motion is crucial for driving pursuit eye movements, these results show that, for triggering pursuit, it is more important to know where the target will be than to know in which direction it will be moving. Stationary distractors placed in the hemifield opposite the pursuit target increase the latency of pursuit, and the degree of increase depends on the eccentricity of the distractor (Knox and Bekkour 2002); the spatial dependence of these latency changes is reminiscent of the effects found with remote distractors for saccades (Walker et al. 1997).

Despite the overlapping ranges and shared influences, the latencies of pursuit and saccades do tend to be different. In the limited number of studies that have measured both, the latencies of pursuit are typically 25–75 ms shorter than the latencies of saccades (Adler et al. 2002; Krauzlis and Miles 1996c; Krauzlis et al. 1999). What accounts for this difference? One basic factor is the difference in the stimuli used to evoke pursuit and saccades. The moving stimuli used in pursuit experiments are inherently more salient than the stationary stimuli used to study saccades, and this may produce a speed advantage for the sensory processing of inputs for pursuit.

A second possibility is that pursuit and saccades operate with different tradeoffs between speed and accuracy. In addition to having shorter latencies, pursuit also tends to commit more errors than saccades, which are often corrected within a few hundred milliseconds with "turn-around" pursuit movements (Krauzlis et al. 1999; Krauzlis and Dill 2002). This behavior is consistent with the finding that the putative "decision criterion" for pursuit is less stringent than that for saccades and may reflect the smaller penalties associated with making pursuit errors (Krauzlis and Dill 2002). Rather than being fixed quantities, the value of these criteria likely change depending on circumstances. For example, corrective saccades during pursuit appear to be based on a prediction of when the eye trajectory will cross the target's path–if this time is too far in the future, or has already passed, then a corrective saccade occurs (de Brouwer et al. 2002). These motor decisions therefore depend, not only on the probability that the target has been correctly identified, but also on the probability that the target can be acquired with pursuit or saccadic motor outputs. The criteria associated with these decisions might be yet another motor parameter regulated by the cerebellum.

The motor command for saccades might also take a little longer to construct than that for pursuit. The premotor commands for pursuit and saccades are specified with a rate code that is related primarily to desired eye velocity. For pursuit, many of the descending control signals are already related to target or eye velocity, meaning that the final motor transformations are relatively simple. For saccades, the control signals are laid out in spatial maps (e.g., the SC), requiring a more elaborate transformation from a place code to a rate code (Sparks and Mays 1990). Similarly, based on a study of how long it takes to cancel pursuit and saccades, it has been suggested that the motor pathways for saccades include an additional "ballistic" interval lasting tens of milliseconds that is not part of the pathways for pursuit (Kornylo et al. 2003).

In summary, the latencies of pursuit and saccades are not stereotyped, but are labile and influenced by many of the same factors. Differences between the latencies of pursuit and saccades might arise from independently controlled decision criteria for the two movements and from the specialized processing steps required to construct the motor commands.

What controls the gating of pursuit?

Fixation has sometimes been viewed as pursuit of a stationary stimulus, but several observations indicate that this is not the case. For example, changes in visual feedback have little effect when subjects fixate a stationary target, but the same changes produce large oscillations in eye velocity when subjects track a moving target (Robinson 1965). This suggests that there is a special significance associated with zero velocity and that fixation and pursuit are controlled differently. Later experiments showed that high-frequency vibrations imposed on a fixated target do not evoke changes in eye velocity, but the same vibrations imposed during pursuit cause large oscillations in eye velocity (Goldreich et al. 1992). Similarly, discrete perturbations in the trajectory of a target can produce smooth changes in eye speed when they occur during pursuit, but not when they occur during fixation (Krauzlis and Miles 1996d; Morris and Lisberger 1987; Schwartz and Lisberger 1994). These and other observations have led to the suggestion that there is a gain controller in the pathways for pursuit eye movements (Grasse and Lisberger 1992; Keating and Pierre 1996; Krauzlis and Lisberger 1994)

Unlike saccades, the gating mechanism for pursuit is not all or nothing. The sensitivity of the pursuit system to visual inputs is a graded attribute that depends on a variety of factors, including eye velocity and expectations about the future target trajectory (Keating and Pierre 1996; Krauzlis and Miles 1996d). These effects can be modeled by including a variable gain element in the forward pathways for pursuit that acts on the command for desired eye velocity, downstream of visual processing (Krauzlis and Miles 1996d). Recent behavioral experiments suggest that the regulation of this gain element can undergo long-term plasticity (Madelain and Krauzlis 2003).

The neural basis for these effects has been elusive, largely because the traditional view of the pursuit system does not provide any candidates consistent with the results from stimulation and lesion studies. However, the recent data on the role of premotor brain stem structures in the control of pursuit provide new possibilities. In particular, the inhibitory relationship between OPN activity and pursuit suggests that they may be involved in gating pursuit, as well as saccades (Missal and Keller 2002). The exact circuit is not yet known, but Fig. 3 shows one recent proposal (Keller and Missal 2003). In this scheme, OPNs regulate the gain of pursuit through their inhibitory effect on pursuit neurons (PN) in the vestibular and prepositus nuclei, analogous to the way that they are believed to gate the occurrence of saccades through inhibitory effects on excitatory burst neurons (EBN). The PNs in this diagram denote the eye–head neurons that receive direct projections from the paraflocculus and are thought to provide the major input to abducens motoneurons during pursuit eye movements (Lisberger et al. 1994a,b; McFarland and Fuchs 1992; Roy and Cullen 2003). Another novel class of pursuit-related neurons, the burst neurons in the PPRF and riMLF (S/PN), might acquire their smooth-eye-velocity modulation through excitatory inputs from the PNs. By inhibiting the OPNs, and completing a loop with PNs, the S/PNs might act to latch the pursuit system in an ON state.

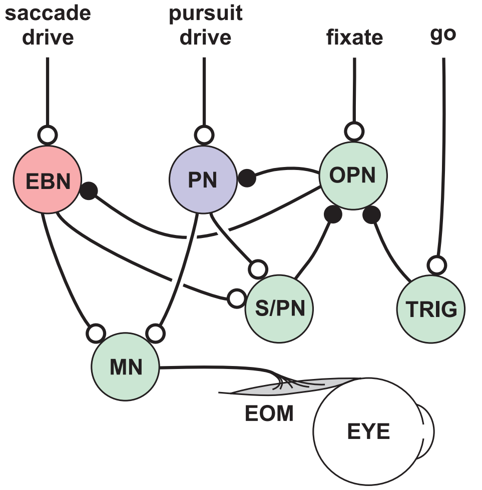

FIG. 3. Possible diagram of how premotor nuclei contribute to both pursuit and saccades, adapted with permission from Keller and Missal (2003). Excitatory synapses are shown with small white circles; inhibitory synapses are shown with small black circles. Red indicates neuron involved in saccades only (EBN), blue indicates neuron involved in pursuit only (PN), and green indicates neurons involved in both.

This type of scheme has several interesting properties. First, the circuit can account for the variable gain of pursuit, because the graded inhibition of the OPNs by the S/PNs would produce a smoothly graded disinhibition of the PNs. The inhibition of OPNs exhibits selectivity for pursuit direction (Missal and Keller 2002), consistent with the observation that the changes in sensitivity during pursuit exhibit directional tuning—the largest changes occur for directions near the current axis of target motion (Schwartz and Lisberger 1994). Second, inhibition of the OPNs may be sufficient to gate pursuit, but a complete cessation of activity is required to trigger saccades. This may explain why a "gap effect" on saccade latencies occurs not only for saccades made during static fixation but also during pursuit, even though pursuit itself involves a release from fixation (Krauzlis and Miles 1996b). From the viewpoint of triggering saccades, the sustained, albeit lesser, OPN activity during pursuit may exert much the same inhibitory effect as that encountered during frank fixation. Third, the "fixate" and "go" signals that regulate the OPNs are presumed to come largely from the SC. This explains how activity in the SC could contribute to the gating of pursuit without necessarily providing a drive signal for pursuit and why the gating of pursuit exhibits many of the same spatial properties as saccades. Fourth, although the pursuit drive in this scheme is assumed to come from the cerebellum (Keller and Missal 2003), another possibility is that it comes from the FEFsem, perhaps via other pursuit-related neurons in the brain stem. The presence of such a direct input to the gating mechanism would explain why stimulation of the FEFsem is especially effective in driving pursuit, even during fixation, whereas stimulation at most other sites along the pursuit pathways are effective only after pursuit is underway.

Although the idea of a central gating mechanism is appealing, gain control in the pursuit system is probably a multifarious business. In particular, mechanisms associated with visual attention can have dramatic effects on signals in the cerebral cortex, before those signals reach any downstream premotor mechanism (Yantis and Serences 2003). The finding that stimulation within the FEF with currents too weak to evoke saccades can nonetheless enhance visual responses in extrastriate area V4 demonstrates the close linkage between the preparation of eye movements and the mechanisms of visual attention (Moore and Armstrong 2003), an idea that is also supported indirectly by a variety of behavioral observations (Rizzolatti et al. 1987). The apparent ease with which sensory and motor signals interact argues that we should not restrict our thinking to a single site or form of gating, but instead consider how the different types of gain control might work together. For example, are the mechanisms of visual attention selective enough to account for the selectivity of pursuit? If so, then downstream mechanisms need not provide any additional filtering—they could act as a simple gate that, when open, grants all descending signals equal access to the output motor pathways. If visual attention were not sufficiently selective, then the downstream mechanisms would have to provide additional filtering to block those descending signals that were inappropriate.

How voluntary is pursuit?

Unlike saccades, pursuit is only sometimes referred to as a voluntary movement. This largely stems from the uncertainty surrounding the distinctions between pursuit and two other types of smooth eye movements, the optokinetic response (OKR) and ocular following, which are reflexive movements driven by full-field and large-field visual motion. Pursuit is often distinguished from these other smooth eye movements on the basis of the small foveal stimuli that pursuit can track, but this is not the most useful distinction, because pursuit can also track large extrafoveal stimuli (Ilg and Thier 1999; Stone et al. 2000). A better distinction is the ability to selectively track a subset of motion signals even in the presence of alternative stronger motion signals. For example, pursuit of a target against a textured background (Collewijn and Tamminga 1984; Keller and Khan 1986) results in full-field motion that would otherwise (e.g., during fixation) result in an oppositely directed OKR. Generating voluntary pursuit therefore requires that the OKR be overridden. This may involve including a "target-to-background" component in the drive signals for pursuit (Fig. 2*B*) to cancel out the competing full-field motion signal from the OKR (Schweigart et al. 1999, 2003). This cancellation might explain why movement of the background can change smooth eye movements when it is in the same direction as ongoing pursuit, but not when it is in the opposite direction (Lindner et al. 2001; Schwarz and Ilg 1999; Suehiro et al. 1999). Accordingly, the directional deficits observed after MST lesions might be caused by disrupting the target-to-background motion signals that escort the target-motion signals to the final premotor circuits for pursuit, in addition to any effect that these lesions have on the target-motion signals themselves.

Unlike the OKR, pursuit can follow the perceived, rather than the retinal, motion. This qualitative point was illustrated some time ago with the demonstration that pursuit can follow the horizontal motion of a wagon wheel defined only by the cycloidal motion of points along the wheel's circumference (Steinbach 1976). More recent studies have used stimuli designed to distinguish between tracking based on perceived motion versus simpler computations such as vector averaging and have found that pursuit can follow the perceived object motion (Beutter and Stone 2000; Stone et al. 2000). However, accomplishing the integration of local motion signals into an object motion signal appears to take time. Initially, pursuit follows the average motion of the individual edges, but converges onto the true object motion with a time constant of about 100 ms (Masson and Stone 2002). Nonetheless, the role of perception in driving pursuit has remained surprisingly controversial and is complicated by several factors, including the likelihood that there are sources of noise that are not shared between pursuit and perception (Beutter and Stone 2000), and the possibility that part of a shared drive signal acts to neutralize the OKR and therefore is not present in the final pursuit output (Schweigart et al. 2003).

Pursuit is also strongly influenced by cognitive expectations. The same visual motion stimulus can produce different pursuit responses depending on whether a particular direction or speed of motion is more likely to occur (Kowler 1989; Krauzlis and Adler 2001). Cognitive expectations can also drive pursuit eye movements even in the absence of a visual stimulus (Barnes et al. 2002). In the "remembered pursuit task," subjects are presented with a regularly repeated target motion and, after only a few presentations, they initiate pursuit well before the target appears. These anticipatory responses occur even if subjects only view, rather than pursue, the target motion (Barnes et al. 1997), and the velocity store associated with these pursuit responses can be sustained for as long as 14 s (Chakraborti et al. 2002). Nonetheless, our capacity to represent motion information in the absence of extant sensory signals appears to be much more limited than our ability to abstractly represent spatial information. This limitation of our imaginations, rather than differences in overall organization, may explain why pursuit appears to be less voluntary than saccades.

CONCLUSION

Recent studies have shown that the functional organization of the pursuit system is much more similar to that of the saccadic system than previously recognized. Among the similarities with the saccadic system, pursuit includes relatively direct cortical control of the movement through the FEF, employs a gating mechanism that is regulated by the SC, and appears to use parts of the same premotor circuits in the brain stem for regulating and formulating the final motor command. As an alternative to the traditional view of pursuit and saccades as distinct oculomotor subsystems, the control of pursuit and saccades might be viewed as different outcomes resulting from a single cascade of sensory–motor functions. From this viewpoint, the several obvious differences between pursuit and saccades illustrate the variety of outcomes that are possible with these layers of control, rather than indicate the presence of independent systems of control.

Abbreviations used in the text and figures:

cMRF	central mesencephalic reticular formation
CN	caudate nucleus of the basal ganglia
DLPN	dorsolateral pontine nucleus
DMPN	dorsomedial pontine nucleus
EBN	excitatory burst neuron
EOM	extraocular muscle
FEF	frontal eye field
FEFsac	saccade subregion of the frontal eye field
FEFsem	pursuit subregion of the frontal eye field
LIP	lateral intraparietal area
MN	ocular motor neuron
MST	medial superior temporal area
MSTd	dorsal portion of the medial superior temporal area
MSTl	lateral portion of the medial superior temporal area
MT	middle temporal area
MVN	medial vestibular nucleus
NOT	nucleus of the optic tract
NRTP	nucleus reticularis tegmenti pontis
OKR	optokinetic response
OPN	omnipause neuron
P-cell	Purkinje cell (output neuron of cerebellar cortex)
PMN	brain stem premotor nuclei (PPRF, riMLF, cMRF) (these include interneurons that do not project directly to motoneurons)

PN	pursuit neuron in the vestibular or prepositus hypoglossi nuclei
PON	precerebellar pontine nuclei (DLPN, DMPN, NRTP)
PPRF	paramedian pontine reticular formation
PSR	principal sulcus region
riMLF	rostral interstitial nucleus of the medial longitudinal fasciculus
rSC	rostral superior colliculus (represents central visual field)
SC	superior colliculus
SEF	supplementary eye field
SNr	substantia nigra pars reticulate
S/PN	saccade burst neuron in the brain stem with pursuit-related activity
TRIG	trigger neuron (inhibitory interneuron in brain stem premotor nuclei)
Verm	vermis (cerebellum)
VN	vestibular nucleus
VPF	ventral paraflocculus (cerebellum)

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