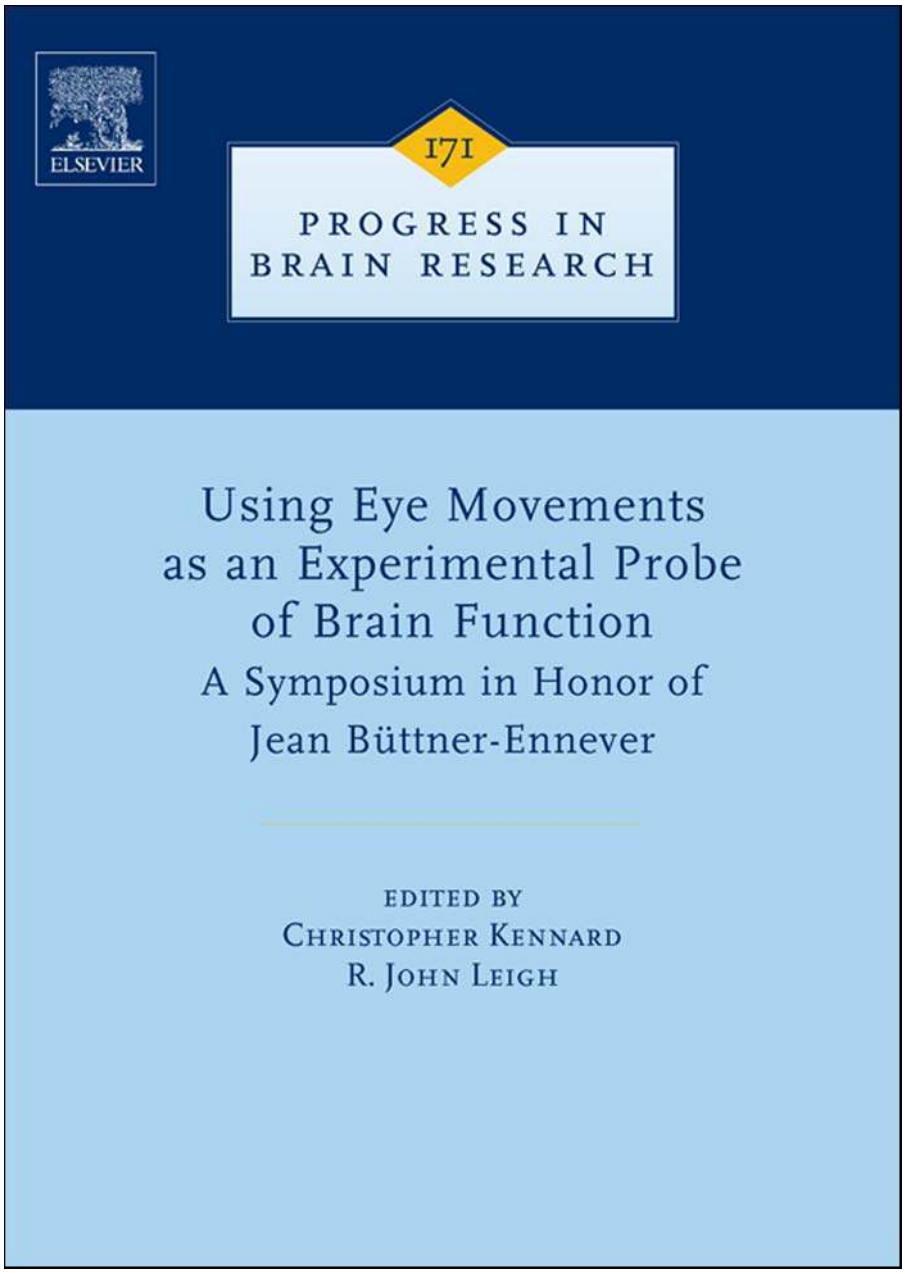

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CHAPTER 3.5

Oculomotor anatomy and the motor-error problem: the role of the paramedian tract nuclei

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Abstract: Anatomical evidence indicates that copies of premotor commands to ocular motoneurons are sent to the cell groups of the paramedian tract, whose projections constitute a major mossy-fibre input to the floccular region of the cerebellum. Damage to this relay impairs gaze-holding, so producing retinal slip signals that are also relayed to the flocculus, in this case as climbing-fibre input. These observations suggest that the relation between efference copy and sensory error is important, and might be used by the cerebellum to learn accurate movements. By modelling the flocculus as an adaptive filter using a covariance learning rule, we show that in simulation the cerebellar cortex can in fact learn to decorrelate efference copy from motor command, and thereby compensate for changes to the oculomotor plant. This mechanism appears to be very robust with respect to plant characteristics and noise, and can cope with error-signal delay provided there is a brainstem site of plasticity. Its general significance is that it removes the need for motor-error signals, which are typically unavailable and in any case not conveyed by climbing fibres. This appears to be an example where anatomical findings have helped address a long-standing problem in adaptive control.

Keywords: flocculus; vestibulo-ocular reflex; oculomotor; cerebellum; motor learning; retinal slip; climbing fibre; paramedian tract; decorrelation control

Introduction

Jean Büttner-Ennever has drawn attention to the anatomy of the cell groups of the paramedian tracts (PMT) in a series of reviews (Büttner-Ennever et al., 1989; Büttner-Ennever, 1992; Büttner-Ennever and Horn, 1996). Input to these cell groups appears to be a copy of the inputs sent to the ocular motoneurons, and their outputs are primarily to the floccular region of the cerebellum (flocculus and ventral paraflocculus, hereinafter simply flocculus). In fact, this cerebellar region receives mossy-fibre input from more neurons in the PMT cell groups than from vestibular neurons (Fig. 1A). These anatomical findings raise an intriguing question about the role of the flocculus in oculomotor control: what useful purpose could be served by sending it a massive efference copy of oculomotor commands?

An early clue was provided by clinical observations, suggesting that damage to PMT cell groups might impair gaze-holding (Zee, referred to in Büttner-Ennever et al., 1989, p. 538). Impaired

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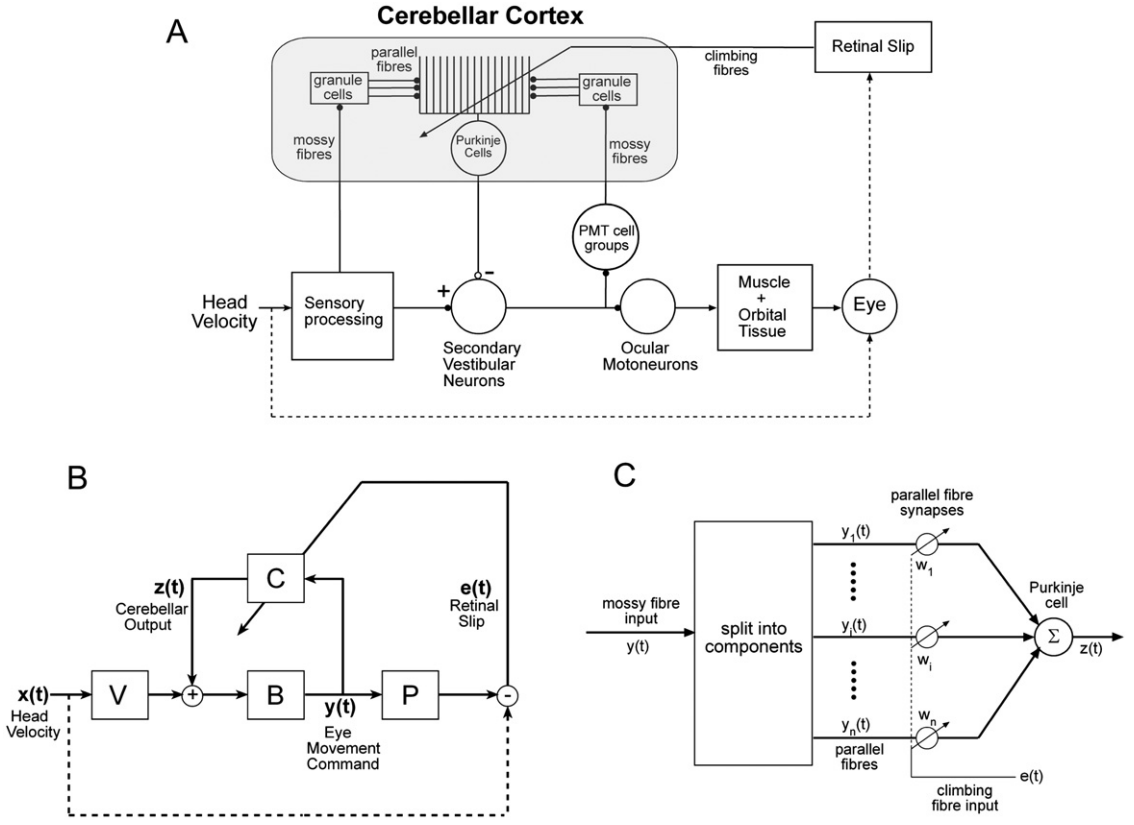

Fig. 1. Circuitry for oculomotor-plant compensation in VOR. (A) Simplified diagram of the circuitry that mediates the horizontal VOR. Head-velocity signals are processed by the semicircular canals and primary vestibular neurons, relayed to secondary vestibular neurons in the brainstem, and then passed to ocular motoneurons. Motor command signals from the motoneurons control the oculomotor plant, i.e., eye muscles plus orbital tissue, in order to produce eye movements that counteract the effects of the head velocity on the retinal image. Inaccurate eye movements produce retinal slip, which is detected by the visual system. A side-loop to the main 3-neuron arc passes through the floccular region of the cerebellum, which receives as mossy-fibre input vestibular information and a copy of the motor command sent to the eye muscles via the PMT cell groups. These mossy-fibre inputs are converted into parallel-fibre signals by granule cells and associated circuitry in the granular layer, and the parallel-fibre signals influence simple spike firing in Purkinje cells. Variation in simple spike firing is transmitted to a subset of secondary vestibular neurons (floccular target neurons) in the brainstem, The flocculus also receives a retinal slip signal as climbing-fibre input, which produces complex spikes. (B) Linearised model of horizontal VOR, derived from the neural circuitry illustrated in panel A. Head velocity $x(t)$ is processed by the filter V, then added to the output $z(t)$ of the adaptive filter C (which corresponds to the floccular region of cerebellum). The summed signal is then passed to the brainstem controller B. The output of B is a motor command $y(t)$, which acts on the plant P. A copy of $y(t)$ is sent back to the adaptive filter C. The command $y(t)$ acts on P to move the eyes, a movement which is added to the head velocity $x(t)$: net image movement is detected as retinal slip $e(t)$ and sent to C. (C) Structure of the adaptive filter shown as C in panel (B). The copy of the eye-movement command $y(t)$ arrives as mossy-fibre input, and is decomposed into components $y_1(t)$ … $y_n(t)$ by the granule cell layer. Each output component $y_i(t)$ is weighted by w_i, corresponding to the efficacy of the corresponding synapse between a parallel fibre and the Purkinje cell. The weighted components are summed by the Purkinje cell and constitute the filter output. The value of each weight w_i is adjusted according to the current value of the correlation between its component $y_i(t)$ and the global retinal slip signal $e(t)$, which arrives as climbing-fibre input. (Adapted from Porrill and Dean, 2007a.)

gaze-holding would result in retinal slip, which so happens to be the major climbing-fibre signal to the flocculus (Simpson et al., 1996). Perhaps then the relation between the two floccular inputs, retinal slip and an efference copy of eye-movement commands, can somehow be used to ensure stable gaze-holding.

The nature of this relationship suggests a possible mechanism. If there is no correlation between movement commands and subsequent retinal slip, then it can be assumed there is no causation: if retinal slip is in fact occurring, then it must be produced by some factor other than inaccurate eye-movement commands (e.g., rotation of an optokinetic drum). If however there is a correlation, then the inference is that inaccurate motor commands *are* causing the retinal slip. Thus, in principle, correlations between mossy-fibre and climbing-fibre inputs to the flocculus could be used to drive motor learning to ensure accurate motor commands and hence stable gaze-holding. Although this idea is consistent in general terms with the influential framework proposed for cerebellar function by early modellers (Marr, 1969; Albus, 1971), the critical question remains of whether it could work in practice. Our first step in addressing this question was to test how the proposed mechanism fared in simulation.

Modelling strategy

The oculomotor plant (that is the extraocular muscles and orbital tissue, Fig. 1A) is primarily viscoelastic (Robinson, 1964). If the plant were a simple viscosity, the force applied to it would be proportional to eye-velocity, so that velocity commands to the eyes would require only a scaling factor for accurate movement. However, the elasticity in the system "diverts" some of the force intended for eye-velocity. For example, at the end of a completed head movement the desired eye-velocity, as specified by the vestibulo-ocular reflex (VOR), is zero. But in the absence of plant compensation the elasticity will pull the eye back to the primary position, which produces eye-velocity when none is required. Impaired gaze-holding is thus just one aspect of inadequate compensation, occurring in the particular circumstances when desired eye-velocity is zero. We therefore modelled gaze-holding as part of the more general process of oculomotor-plant compensation.

The general principles of oculomotor-plant compensation, in the context of velocity commands supplied by the VOR, were first analysed by Skavenski and Robinson (1973). They suggested that it required two pathways to convert the vestibular input to the eye-command output. The first direct pathway was a simple gain, corresponding to the plant's viscosity term. The second indirect pathway was a neural integrator, producing an eye-position command from the desired-velocity input. The two pathways together can exactly compensate for a first-order viscoelastic plant. Although more complex plants (Sklavos et al., 2005) require more complex compensation (Optican and Miles, 1985), the framework proposed by Skavenski and Robinson remains applicable, with the proviso that the neural mechanisms responsible for plant compensation may now be referred to as an "inverse plant model."

To simulate how an accurate inverse plant model could be learnt from the relation between efference copy and retinal slip, we simplified the circuitry illustrated in Fig. 1A in two ways. First, the three main elements (brainstem, cerebellum, and plant) were linearised, to facilitate mathematical analysis of our proposed mechanism. Secondly, we considered only the efference copy mossy-fibre input to the flocculus (Fig. 1B), termed the "recurrent architecture." That was because the problem of plant compensation applies to all eye movements, not just those produced by the VOR, so specific information from the vestibular system is not required. In effect we were modelling the effects on VOR calibration of changing the plant (e.g., muscle weakness), rather than the more familiar effects of changing vestibular processing (magnifying goggles), where indeed the vestibular mossy-fibre inputs to flocculus do become relevant.

In the model, the plant was represented as a first-order system with time constant 0.1 s (further details in Porrill and Dean, 2007a). The properties of the brainstem were deduced from the effects of floccular lesions (Zee et al., 1981), which include a

gaze-holding deficit where eccentric gaze returns to the primary position with a time constant of ~1 s. This effect can be simulated by assuming that the brainstem on its own contains a leaky integrator in the indirect pathway; it is the role of the flocculus to assist. Such a role is consistent with Robinson's view of the cerebellum as the "repair shop" of the oculomotor system. In addition, to test the proposed learning mechanism further, the gain of the direct pathway was reduced from its desired value of 1.0 to 0.5.

The flocculus itself was represented as an adaptive filter (Fig. 1C), a development by Fujita (1982) of the Marr–Albus framework alluded to above, and one that is increasingly used in cerebellar modelling. The (mossy-fibre) inputs to the filter are split into components (parallel-fibre signals), which are weighted individually (synapses between parallel fibres and Purkinje cells) then summed to produce the filter's output (Purkinje cell simple spikes). The weights are altered by the error signal (climbing-fibre input) using a learning rule that can be stated qualitatively as follows. If parallel-fibre firing is positively correlated with climbing-fibre firing (which signals retinal slip), reduce the weight (Long Term Depression); if it is negatively correlated, increase the weight (Long Term Potentiation); if uncorrelated, no change. In quantitative form it corresponds to Sejnowski's (1977) covariance learning rule that implements the standard Least Mean Squares algorithm used in adaptive signal processing. Since stability is achieved when the weights cease to change, and this happens when the two input signals are uncorrelated, we have referred to the proposed adaptive mechanism as "decorrelation control."

It is important to emphasise that the brainstem and cerebellar models, and the learning rule, are conventional. The key new feature is the use of the recurrent architecture (Fig. 1B) based on the anatomy of the PMT cell groups (see also Glasauer, 2003) for purposes of adaptive plant compensation.

Modelling results

Before training, all the weights in the simulated cerebellum C were set to zero. The performance of the system thus reflected that of the brainstem alone, with its low gain and leaky integrator. Thus, the "pre" trace in Fig. 2A shows that after a brief head-displacement the compensatory eye movement is too slow and therefore too small, and the globe then returns to the primary position with time constant of 1 s (impaired gaze-holding). This performance is shown in relation to input frequency in Fig. 2B. The "pre" trace here shows the gain of the brainstem alone is roughly constant at 0.5 for frequencies above ~0.3 Hz, but then drops sharply for lower frequencies (reflecting the increasing contribution of plant elasticity). The contribution of the brainstem leaky integrator can also be seen in Fig. 2B: the trace labelled "$B = 0.5$" shows the performance of a brainstem with no integrator, very substantially worse that the actual brainstem for frequencies less than ~5 Hz). Finally, Fig. 2C shows the response of the brainstem to a mixed frequency (coloured-noise) head-velocity input in terms of retinal slip.

Training with the decorrelation-control mechanism sharply reduced the retinal slip error (Fig. 2C, D), and gaze-holding became essentially perfect (Fig. 2A). The gain of the VOR became ~1.0 over a wide range of frequencies (Fig. 2C). These results indicate that removing the correlation between retinal slip and an efference copy of the eye-movement command can achieve plant compensation in a simplified system.

Subsequent manipulation of model parameters indicated that this finding was robust. (i) Higher-order plants that require a "slide" as well as a step command (Optican and Miles, 1985) can also be compensated (Dean et al., 2002). (ii) Different methods of splitting mossy-fibre inputs into parallel-fibre components primarily affect speed of learning rather than final convergence (Dean et al., 2002). (iii) The scheme can be readily extended to three dimensions (Fig. 3) (Porrill et al., 2004). (iv) It continues to be effective if the climbing-fibre input signals only the direction of retinal slip, or if its retinal slip signal is delayed (Porrill and Dean, 2007a). This latter result is of particular interest in the context of the VOR, since it showed that a *brainstem* site of plasticity is required for high-frequency VOR performance in the presence of substantial (~100 ms) retinal slip

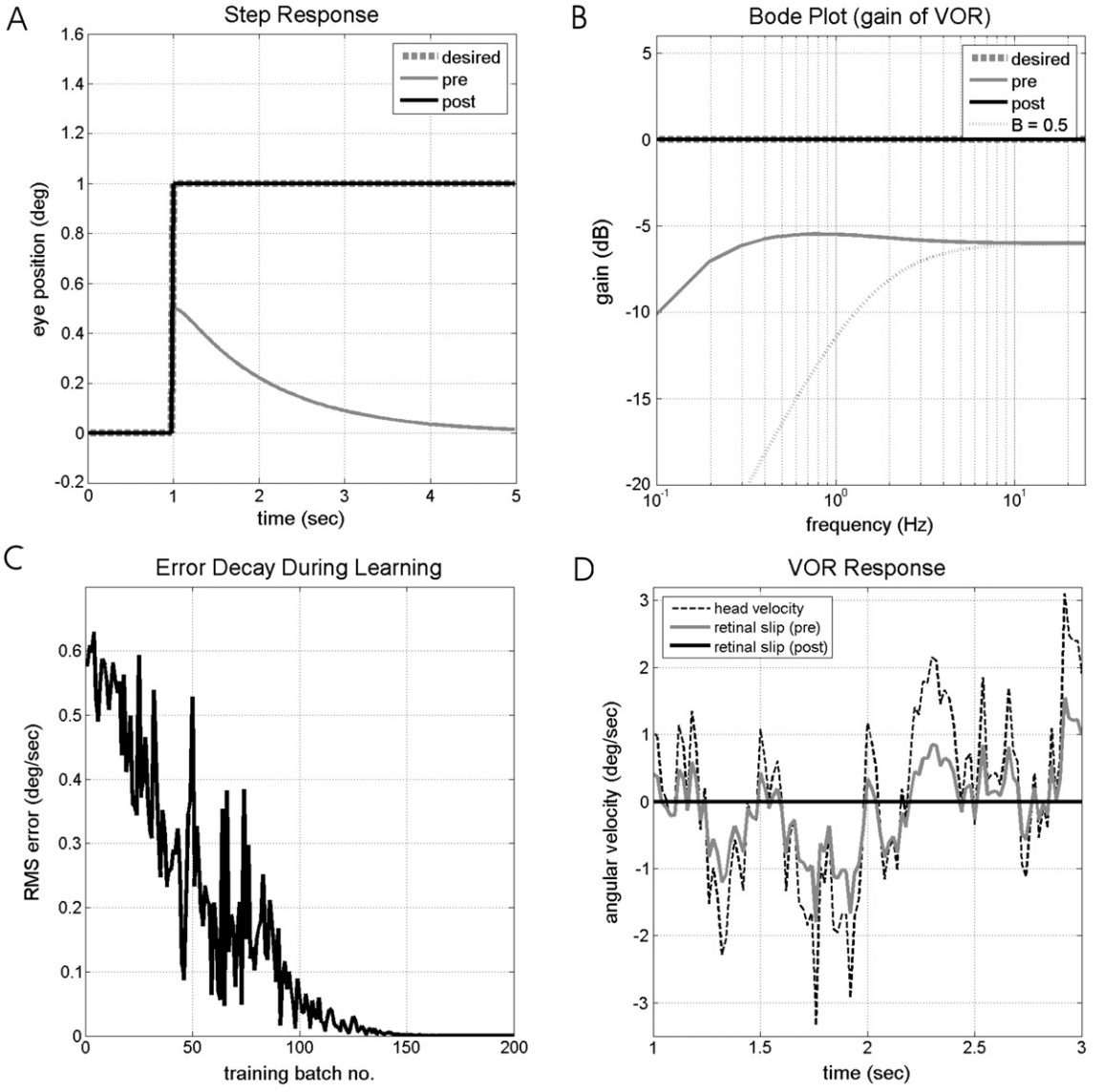

Fig. 2. Model performance before and after training with an undelayed retinal slip signal. (A) Eye-position response to sudden head-displacement. The desired and post-training performances are effectively identical. (B) System gain for sinusoidal input signals as a function of frequency (Bode gain plot). Gain is measured as ratio of eye-velocity amplitude to head-velocity amplitude. Performance before training is shown both for the complete brainstem controller ("pre"), and for the brainstem controller as simple gain ("$B = 0.5$") which corresponds to the direct pathway on its own. After training, the desired and post-training performances overlap. (C) Decline in retinal slip amplitude with training. Root-mean-square (RMS) retinal slip amplitudes, measured over a 5 s training batch, plotted against number of training batches. (D) Example of retinal slip to mixed-frequency head-velocity input before and after training. (Adapted from Porrill and Dean, 2007a.)

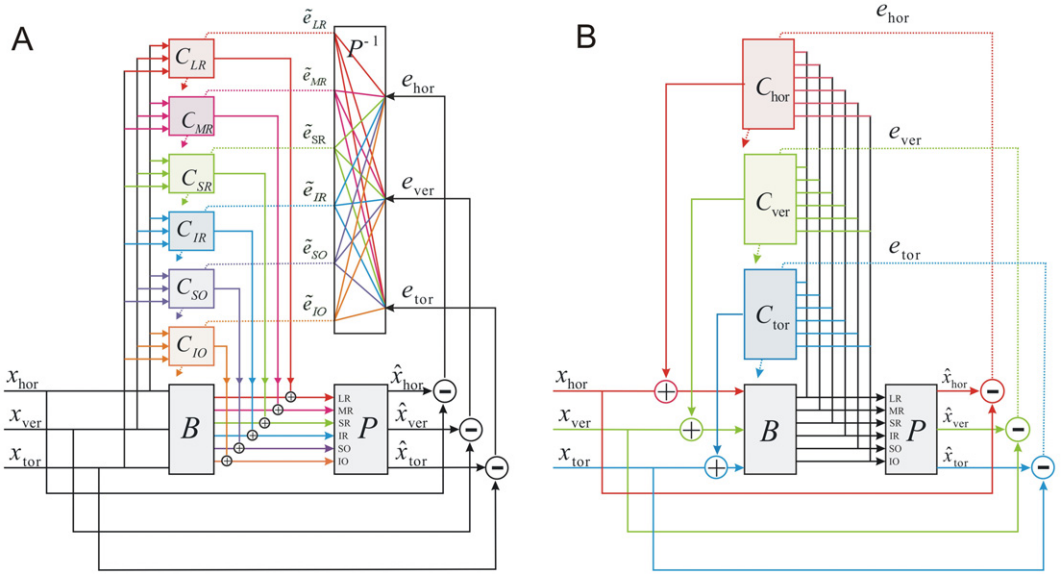

Fig. 3. Alternative architectures applied to simulated VOR in 3D. The vestibular system recovers three components: x_{hor}, x_{ver}, and x_{tor} (horizontal, vertical, and torsional) of head angular velocity. These are processed by the brainstem and cerebellum to produce motor commands to the six extraocular muscles so as to stabilise the eyes rotational position in space. The motor plant is a 3×6 matrix transfer function P and the brainstem contribution to the VOR is a 6×3 matrix transfer function B. (A) Feedback error architecture: the cerebellum takes the three vestibular signals as input and supplies corrections to the six motor commands. Colour is used to highlight the "motor space" modularity. For example, the green component shows those Purkinje cells that contribute to the superior rectus (SR) muscle command, and which require the corresponding motor command error $\tilde{e}_{SR}$ as training signal on their climbing fibres. This signal must be reconstituted from the horizontal, vertical, and torsional components of retinal slip. (B) Recurrent architecture: the cerebellar takes the six motor commands as input and supplies corrections to the three vestibular signals. Colour is used here to highlight the "task space" modularity. For example, the green component shows Purkinje cells contributing to vertical eye motion; these require the vertical component of optic flow as training signal. (Adapted from Porrill et al., 2004.) (See Color Plate 3.5.3 in color plate section.)

delays. Although the existence of brainstem plasticity in VOR adaptation is well-known, its role in the context of Marr–Albus cerebellar learning schemes has hitherto been unclear and contentious (Porrill and Dean, 2007a).

Part of the reason for the scheme's robustness for linearised plant compensation can be demonstrated mathematically (Porrill et al., 2004). The recurrent connectivity implements a partial state feedback controller and using Lyapounov methods it is possible to show that the algorithm can stably learn to adapt to changes in a wide class of motor plants. Thus, the method suggested by the anatomy of PMT cell groups may have applicability beyond eye movements themselves. We consider this first for control of artificial systems including robots, before returning to the role of the recurrent cerebellar architecture in biological sensorimotor processing.

Application to robotics

Adaptive filters and artificial neural nets are often used to implement supervised learning schemes in which weight change is driven by the difference between the device's actual output, and what that output should have been. In the case of motor control, the desired output is the correct command required for the movement in question to be accurate. The difference between this correct command and the actual inaccurate command is sometimes referred to as "motor error."

The correct commands and hence motor error may well be known for a manufactured and unchanging plant. However, if the plant does change in unforeseen ways because of damage or wear, the motor error will not be known. These circumstances apply both to autonomous robots and to biological systems, where the evidence of inaccurate motor commands is *sensory* and concerns the nature of the movement itself. How to use this sensory information to guide learning is a major problem — the distal error problem in adaptive control (Jordan and Wolpert, 2000).

The recurrent architecture suggested by the anatomy of PMT cell groups for eye-movement control may offer a solution to this general problem (Fig. 3). In this architecture the sensory signal denoting inaccurate movement drives learning directly, resulting in a simple modular circuit for multi-dimensional motor control (Fig. 3B). In contrast, the alternative scheme whereby sensory error is converted into an estimate of motor error (Fig. 3A) requires a complex device to extract the relevant combination of retinal slip error for each motoneuron pool. This is equivalent to requiring an estimate of the inverse plant, a potential problem since this is what the system is trying to learn in the first place.

These considerations suggest that the decorrelation-control algorithm may have applications to robotics, and initial tests in a robot head-camera system (Fig. 4A) suggest that it can produce effective gaze-stabilisation in the context of an artificial VOR (Lenz et al., 2007). We have also recently shown how the algorithm can be extended to kinematic control of a simulated two-joint robot arm (Porrill and Dean, 2007b), and this has been applied to the control of a real robot arm by Iyad Obeid (Fig. 4B) who is investigating its potential for control of brain-machine interfaces.

Application to biology

Although the decorrelation-control algorithm arose from biology, and has applications to artificial systems, it still remains only a candidate algorithm for the actual eye-movement control system that inspired it. In particular, specific tests of the algorithm's relevance to floccular function have yet to be carried out. A possible though technically demanding test would be to record from relevant Purkinje cells during simulated plant adaptation, and determine the changes if any in eye-position related firing.

It is however important to note that, notwithstanding this lack, the algorithm is in general consistent with the substantial amount of existing evidence regarding the role of the flocculus in gaze-stabilisation. (i) Inactivating or removing the flocculus abolishes VOR adaptation in general, and adaptation to simulated plant changes in particular (Optican et al., 1986). (ii) Inactivation of PMT cell groups relating to vertical movement in cat "has an effect on the integrator function similar to that found in previous experiments lesioning the flocculus" (Nakamagoe et al., 2000, p. 858). (iii) Electrophysiological evidence suggests that $\sim$75% of primate mossy fibre or other granular-layer input elements modulate their discharge in relation to eye movements (Miles et al., 1980), consistent with a powerful PMT cell group input. (iv) Brainstem cells receiving floccular outputs (floccular target neurons) carry an eye-position signal (Scudder and Fuchs, 1992) that appears to reinforce the function of the nucleus prepositus hypoglossi or brainstem horizontal integrator (Hazel et al., 2002). (v) Some floccular Purkinje cells fire in relation to eye position. The fact that the proportion of such cells is quite low ($\sim$20%) in primates (Miles et al., 1980) suggests that plant compensation is not the only function of the flocculus, consistent with the extensive evidence that has established a role for the flocculus in smooth-pursuit.

Finally, the recurrent architecture found for the flocculus appears to be a widespread feature of cerebellar organisation. It has been found for regions of the cerebellum associated with saccades and arm movements (references in Porrill and Dean, 2007b), and it has been suggested that "closed-loop circuits are a fundamental unit of architecture for cerebellar interconnections with the cerebral cortex" (Kelly and Strick, 2003, p. 8441). Interestingly, a "challenge for future studies is to determine the computations that are supported by this architecture" (p. 8443). The results outlined

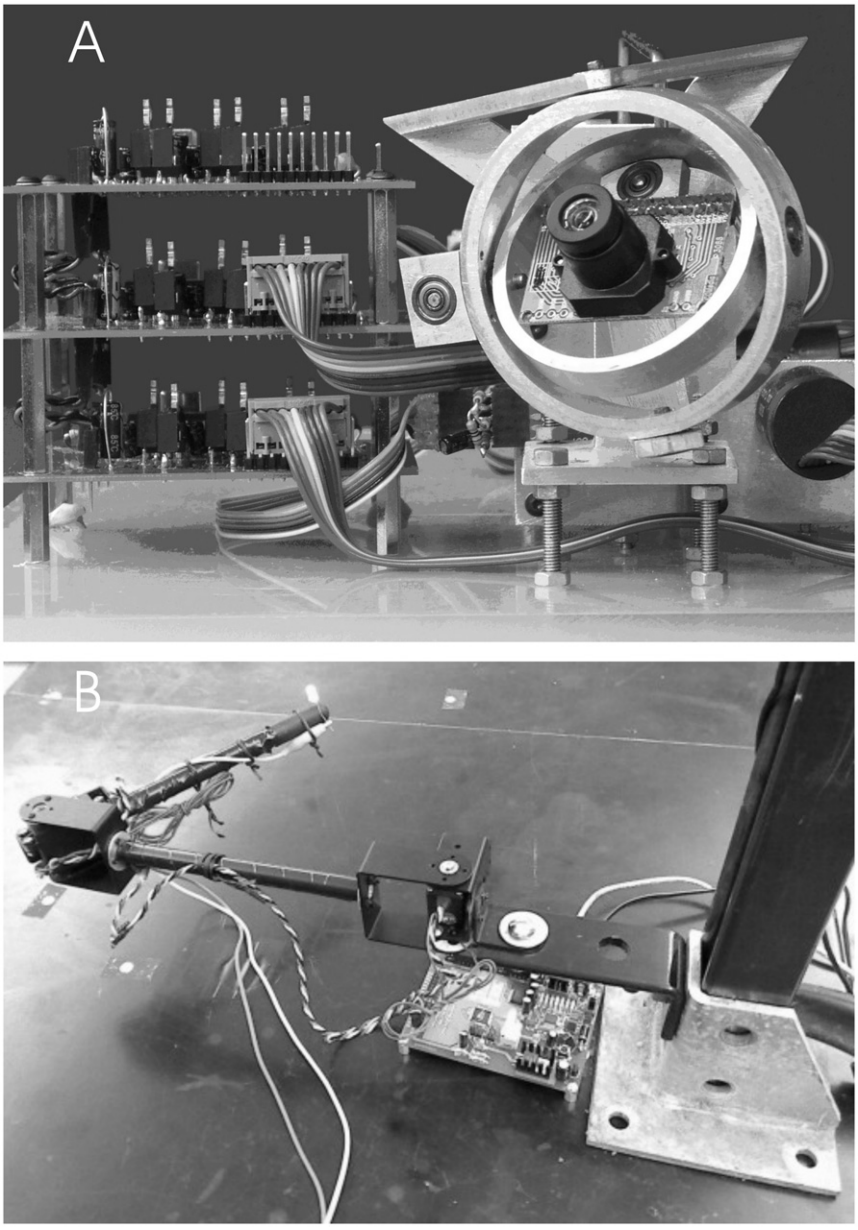

Fig. 4. Application of decorrelation control to robotics: (A) Eye-Robot, a camera-head rig developed by Bristol Robotics Laboratory (Lenz et al., 2007). Adapted with permission from Bristol Robotics Laboratory, appearing in PLoS Computational Biology, Vol. 3, Issue 10, October 2007. (B) A two-joint arm, developed by Dr. Iyad Obeid, Department of Electrical and Computer Engineering, Temple University, Philadelphia PA, USA.

here suggest a candidate computation, giving any cerebellar area the power to adjust its output appropriately in the face of downstream changes.

Conclusions

The anatomy of the PMT cell groups points to the importance of sending an efference copy of eye-movement commands to the floccular region. We suggest that one of the functions of this signal is to learn accurate compensation of the oculomotor plant, and we describe a procedure where this is achieved by adaptively decorrelating efference copy from retinal slip. The "decorrelation-control" procedure works well in simulation, and can be shown to solve the classic problem of motor error in a principled manner. It may therefore have applications in the field of autonomous robotics, and also to biological motor control of responses besides eye movements, given that recurrent connections are a common feature of cerebellar anatomy.

Abbreviations

PMT	paramedian tract
VOR	vestibulo-ocular reflex

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