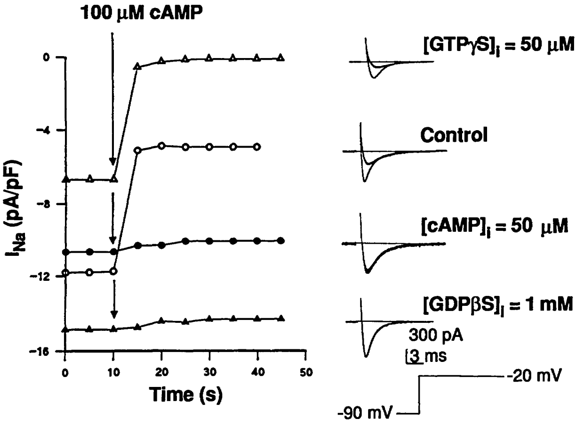

Fig. 4. The effect of extracellular cAMP on I_{Na} in frog ventricular myocytes dialyzed with cAMP, GTPγS, or GDPβS. Left, time course of suppression of I_{Na} by 100 μM cAMP applied 1 to 2 s after the third control measurement in cells dialyzed with the following: 50 μM GTPγS (△), control solutions with no intracellular cAMP (○), 50 μM intracellular cAMP (●), or 1 mM GDPβS (▲). Right, superimposed tracings of I_{Na} in the four indicated conditions activated by 15-ms depolarizing pulses to −20 mV from a holding potential of −90 mV. Solutions used in myocytes dialyzed with control (○) or 50 μM cAMP (●): all external solutions (*12*) contained 10 mM NaCl and, in the case of GTPγS and GDPβS, 1 μM nitrendipine. The internal solution (*12*) was standard except for the changes in the concentration of cAMP or replacement of cAMP with GTPγS or GDPβS. Cell capacitances were 79 to 99 pF.

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Autoassociation and Novelty Detection by Neuromechanics

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Many biomechanical systems contain ball joints with several elastic actuators (muscles) obliquely attached to the links. The problem of calculating the optimum actuator commands to achieve a desired link orientation is a difficult one for any control system; however, the elasticity of the actuators may be part of the solution. Mechanoreceptors such as those found in muscles and tendons are capable of performing operations that can be regarded as autoassociation and novelty detection, respectively, by minimization of potential energy. The information provided by such sensors may then be exploited for optimization of muscle coordination.

JOINTS IN BIOMECHANICAL SYSTEMS are usually moved by muscles attached to the links. Such muscles are elastic, so a given motor command does not result in a unique muscle length or torque. The arrangement of muscles in space is oblique, so the direction of movement generated by one muscle is affected by the activation of another muscle, and the number of muscles is always greater than the number of degrees of freedom of the joint, so there is a continuum and hence an infinite number of possible motor commands for achieving a given movement of a link. As a result, it seems difficult to find a procedure that yields an optimal pattern of motor commands for any desired orientation of the link. The information provided by sensors located in the elastic actuators can help to solve this coordination problem, if the system incorporates operations similar to autoassociation and novelty detection.

Autoassociation has been shown to exist in multiunit systems that allow for "multilateral" or long-range interactions, such as simulated neural networks interacting through synapses (*1*), spin glasses interacting through electromagnetic fields (*2*), electronic circuits interacting through connections (*3*), or optical systems interacting through photons (*4*). I will show that the interactions between elastic actuators, such as muscles, acting on a common rigid body, combined with suitable receptors, such as mechanoreceptors, provide the basis for autoassociation and novelty detection. Among vertebrate muscle systems, the muscles rotating the eyeball expose these properties particularly clearly: there are six springlike muscles attached to a common rigid body, each muscle richly endowed with muscle receptors (*5*) and tendon receptors (*6*) that increase their activity upon mechanical stretch of the "parent" muscle.

To keep the treatment as simple as possible and to arrive at a linear characterization of the mappings, I used a first-order approximation (*7*) of the system behavior in the vicinity of a particular operating point. Thus an orientation change of the rigid body can be represented by a three-dimensional (3-D) vector δe, the direction of which is the axis of rotation and the length of which is a measure of the angle of rotation (*8*). The angular elongations δp_i of the n individual muscles are obtained from the scalar product of the direction of orientation change δe with the unit directions of muscle action, and they may be written

$$\delta p = -M^T \delta e \qquad (1)$$

where M is a 3 × n matrix, the columns of which are formed by the n unit directions of rotations that could be caused by individual muscles, and the superscript T denotes the

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matrix transpose. The negative sign results, because muscles are shortened when the rigid body is turned in their direction. If the change of torque δf_i exerted by the ith muscle is assumed to be the sum of an active component and a passive component, a linear approximation reads

$$\delta f = Z\delta m + A\delta p \tag{2}$$

where δm_i are the motor command changes to the muscles and A and Z are diagonal $n \times n$ matrices that contain the coefficients of elastic stiffness and neuromuscular transmission, respectively. The potential energy change $\Delta\epsilon$ of a muscular system in the absence of external torques is given by the sum of two scalar products

$$\Delta\epsilon = (-M\delta f)^T\delta e + (L\delta e)^T\delta e \tag{3}$$

where L represents an elastic load, a tensor expressed in terms of a 3×3 matrix (*9*). In a stable equilibrium of the neuromechanical system, the condition of vanishing gradient of ϵ yields

$$M\delta f = L\delta e \tag{4}$$

Solving the coupled set of vector Eqs. 1, 2, and 4 for δe as a function of δm yields

$$\delta e = (MAM^T + L)^{-1}MZ\delta m \tag{5}$$

The introduction of two more simplifying assumptions clarifies the meaning of this expression. First, the rotatory stiffness coefficients of the muscles are assumed to be approximately equal, so the diagonal matrix becomes proportional to the identity matrix, $A = \alpha I$. Second, it is assumed that in all directions the stiffness of the muscles is considerably larger than the stiffness of nonmuscular tissue, so the load tensor can be approximated by a zero matrix, $L = 0$. Under these conditions, Eq. 5 simplifies to

$$\delta e = \alpha^{-1}(M^T)^+Z\delta m \tag{6}$$

Here the superscript "plus" denotes the Moore-Penrose generalized inverse (*10*) (MPGI). The MPGI exists for any matrix and is identical to the proper inverse, if the latter exists.

In the derivation of the dependences of receptor signals on the motor commands it is important to note that two classes of receptors have been observed: the length receptors located parallel to the muscles that monitor the individual elongations (muscle receptors) and the tension receptors located in series to the muscles that monitor the individual torques (tendon organs). Combining the respective transduction coefficients of these receptors into diagonal matrices X and Y yields

$$\delta w = X\delta f \tag{7}$$

$$\delta u = Y\delta p \tag{8}$$

for tension receptor signals δw and length receptor signals δu.

	l.r.	m.r.	s.r.	i.r.	s.o.	i.o.
l.r.	0.457	-0.456	-0.132	-0.148	0.015	0.016
m.r.	-0.456	0.457	0.147	0.133	-0.016	-0.014
s.r.	-0.132	0.147	0.549	-0.456	0.015	0.014
i.r.	-0.148	0.133	-0.456	0.535	-0.023	-0.024
s.o.	0.015	-0.016	0.015	-0.023	0.504	-0.499
i.o.	0.016	-0.014	0.014	-0.024	-0.499	0.499

	l.r.	m.r.	s.r.	i.r.	s.o.	i.o.
l.r.	0.543	0.456	0.132	0.148	-0.015	-0.016
m.r.	0.456	0.543	-0.147	-0.133	0.016	0.014
s.r.	0.132	-0.147	0.451	0.456	-0.015	-0.014
i.r.	0.148	-0.133	0.456	0.465	0.023	0.024
s.o.	-0.015	0.016	-0.015	0.023	0.496	0.499
i.o.	-0.016	0.014	-0.014	0.024	0.499	0.501

Fig. 1. Projections implemented by the neuromechanics of the human eye. The upper matrix describes the autoassociation produced by length receptors; the lower matrix describes the novelty detection produced by tension receptors in the extraocular muscles. Measurements of the spatial arrangement of extraocular muscles were taken from Volkmann (*14*) (l.r., lateral rectus; m.r., medial rectus; s.r., superior rectus; i.r., inferior rectus; s.o., superior oblique; and i.o., inferior oblique).

Combination of Eqs. 1, 6, and 8 leads to

$$\delta u = -\alpha^{-1}YM^T(M^T)^+Z\delta m \tag{9}$$

The term between the diagonal matrices Y and Z is an orthogonal projection operator on the space spanned by the columns of M^T. This result is important for two reasons. First, because M^T is identical to $[(M^T)^+]^+$, the image space happens to be the space of "optimal" input vectors to the motor system, as described by Eq. 6, where "optimal" means that the activity changes have minimal norm, and thus motor command components that cancel each other are avoided (*11*). Second, such orthogonal projection is equivalent to a linear autoassociative mapping, which has been discussed extensively by Kohonen (*12*). In other words, the physical structure of the actuator system seems to allow the set of optimal motor commands to be stored in a neuromechanical autoassociator, which automatically suppresses all components of the signal that are not optimal, associates a motor command pattern optimally suited to generating the desired orientation change, and makes the signal available to the nervous system through the length receptors.

On the other hand, combining Eqs. 1, 2, 6, and 7 yields

$$\delta w = X[I - M^T(M^T)^+]Z\delta m \tag{10}$$

Here the term between the two diagonal matrices (*13*) X and Z is an orthogonal projection operator onto the subspace orthogonal to the space of optimal motor commands. This projection is equivalent to a linear novelty filter (*12*) with respect to the same "stored" vectors as those of the autoassociator. Hence the same neuromechanical system is capable of suppressing all components of the signal that are optimal, isolating the "novel" components that deviate from the optimal ones, and making these novel components available to the nervous system through the tension receptors.

The matrices describing autoassociation and novelty detection in the neuromechanical system depend only on the number and the spatial arrangement of the muscles. Consequently, on the average, the matrices can be considered as constants for a given species. As an example, measurements (*14*) of the spatial arrangement of the six external human eye muscles have been evaluated. The characteristic matrices (*15*) for length receptors and tension receptors are given in Fig. 1.

The operations performed by the neuromechanical system solve a nontrivial task: for any number of muscles and for an arbitrarily oblique arrangement in the 3-D space, the optimal motor commands and the deviations from these commands are continuously found in real time. Solving this task with a computer involves at least a lengthy procedure such as calculating the generalized inverse of a matrix; in the nonlinear case the task can only be solved numerically by means of iterative algorithms. In a neuromechanical system, this function is achieved by minimization of a potential energy change $\Delta\epsilon$ in the n-dimensional signal space; replacing the 3-D torque and orientation changes in Eq. 3 by the sum of the n individual components yields

$$\Delta\epsilon = (Z\delta m)^T\delta p + (A\delta p)^T\delta p + (L\delta e)^T\delta e \tag{11}$$

The shape of the potential function close to the minimum is mainly determined by the stiffness term quadratic in δp, but the location of the minimum is shifted by the activation term dependent on δm. Thus the calculatory power of the system is due to a mechanism analogous to the autoassociation mechanisms found in other systems, which are also based on the minimization of either quadratic energy functions (for example, spin glasses) or error functions (for example, neural networks). However, the neuromechanical system is unique in one aspect:

whereas in other systems meaningful patterns can only be stored when a procedure, or learning rule, exists to establish the correct interactions, in the neuromechanical system the interactions are built into the mechanics and happen to store meaningful patterns.

As an example of the relation between the signals, consider the oculomotor system. When the oculomotor system has just begun to acquire the capability to generate functionally correct movements, such as to compensate for head movements in 3-D space, it is very likely that the coordination of the six extraocular muscles is still far from optimal. The two classes of stretch receptors in the extraocular muscles provide two types of signals that may help to teach the control system how to generate the same (functionally correct) eye movement with an optimal trajectory in the six-dimensional actuator space; the length receptors produce by autoassociation a sign-inverted optimal teach trajectory, and the tension receptors produce by novelty detection a vectorial corrective time function. These signals are available in several adaptive structures of the brain, for example, in the flocculus of the cerebellum (*16*).

Because the error-correcting properties of the operations are evolutionary advantageous, it is possible that the computational power of neuromechanics is exploited for the development of biological control systems. A feedback mechanism suitable for improving the motor commands by means of the novelty detector signals requires only sign inversion and linear summation on a fast time scale. Linear summation in the control center does not suffice for the exploitation of the autoassociation signals in a feedback loop because a feedback of positive sensor signals leads to instabilities, whereas a feedback of negative sensor signals simply counteracts all movements. However, a nonlinear mechanism that uses the optimal signals as teachers for adaptation on a slower time scale would allow a fast optimizing feedforward controller to develop. Such nonlinear mechanisms are known as learning rules in the field of artificial neural networks, and these mechanisms have been shown to be effective (*17*). Thus neuromechanics may play an important role in the organization of control of biological and unconventional artificial manipulators.

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