

# Control of Equilibrium Position and Stiffness Through Postural Modules

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**ABSTRACT.** If muscles are viewed as spring-like torque generators, then the integral of torque with respect to joint angle is the potential energy of that muscle. An energy function for the musculoskeletal system can be defined by summing the energy contribution of each muscle and the potential energy stored in the limb. Any local minimum in this energy landscape is a possible equilibrium position for the limb. The gradient of this function with respect to joint angles is a torque field, and the task of postural control is to find a set of muscle activations to produce a desired field. We consider one technique by which this approximation may be achieved: A *postural module* is defined as a synergy of muscles that produce a class of torque functions that converge at a constant equilibrium position, but whose stiffness at this position varies as a function of activation of the postural module. For a single-joint system, we show that through control of two such modules it is possible to produce any stiffness at any desired equilibrium position.

To extend this scheme to a multijoint system, we initially derive the mechanical constraints on the shape of the restoring force field when a multijoint limb is displaced from equilibrium. Next, we consider voluntary control of the force field when the human arm is displaced from equilibrium: Mussa-Ivaldi, Hogan, and Bizzi (1985) have suggested that subjects are unable to voluntarily change the shape and orientation of the field, although they can readily scale it. This suggests existence of a limitation on the independent recruitment of arm muscles. We show, through simulation, that the inability to voluntarily control the shape and orientation of the restoring force field can be attributed to an organization of postural modules that act as local stiffness controllers. We propose that through coactivation, postural modules coarsely encode the work space and serve as an intermediate control system in the motor control hierarchy.

*Key words:* equilibrium position, force fields, mathematical model, motor control, postural modules, stiffness

From everyday experience, we know that we can control almost any aspect of our motor behavior. Brooks (1986) has suggested that the task that we wish to accomplish (or the instruction that we have been given) determines the motor variable that the brain tries to control. If the task only requires us to position our arm at some configuration, then it is not necessary to consciously set the

stiffness with which we maintain the arm, as long as it is kept above a minimum needed to ensure stability of the limb. If we are told to move our hand from one position to another, then perhaps we could pay attention to the path that the hand follows, but we can certainly choose another path if told to do so. By studying the degrees of freedom available to the controller when a task is to be performed, much has been learned about the kinds of variables that the subjects optimize to arrive at a particular solution to the ill-posed task, that is, a task for which a unique solution does not exist (cf. Hogan, 1984; or Uno, Kawato, & Suzuki, 1989). In reality, one hopes to learn about the underlying neural organization for control of the motor apparatus from the particular solution that is chosen by the subjects to the ill-posed task.

In this article, we examine a set of experiments on the control of position and stiffness in the human arm and ask what the observed constraints on the solution space tell us about the organization of the controller. We concentrate on the following findings of Mussa-Ivaldi, Hogan, and Bizzi (1985) and Shadmehr, Mussa-Ivaldi, and Bizzi (1993): When the arm was displaced from equilibrium, a restoring force field could be measured. Although the subject was not instructed to maintain any particular stiffness (the rate of change of force with respect to displacement), a consistent behavior in the shape and orientation of the restoring field was observed. Even when faced with a predictable displacement, the subjects were unable to significantly alter the shape of this field. Our goal is to show that these observations are consistent with a particular control system that organizes voluntary recruitment of arm muscles for maintenance of posture.

The idea is to view the constraints on voluntary control of arm stiffness in man in terms of the framework for con-

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trol of multijoint posture that emerges from the results of Bizzi, Mussa-Ivaldi, and Giszter (1991), who have proposed that the muscles of a multijoint limb are organized through neural *modules* in the spinal cord, and that activation of a module produces a stable postural force field. Here, we implement a computational model in which a few of these modules coarsely encode the reachable space: We begin by showing that in a single-joint system it is possible to produce an equilibrium position anywhere in the reachable space through coactivation of two modules, each with an equilibrium point at one extreme of the reachable space. We extend this work to a two-joint, six-muscle system and show that because a module has the ability only to scale a limb's stiffness, coactivation leads to fields whose stiffnesses at equilibrium are almost uniform in terms of their shape and orientation. Apparently, if the only mechanism for voluntary control of posture is through coactivation of postural modules, it would not be possible to significantly rotate the limb's stiffness. This framework provides one explanation for the constraint observed by Mussa-Ivaldi et al. (1985) in voluntary control of arm stiffness.

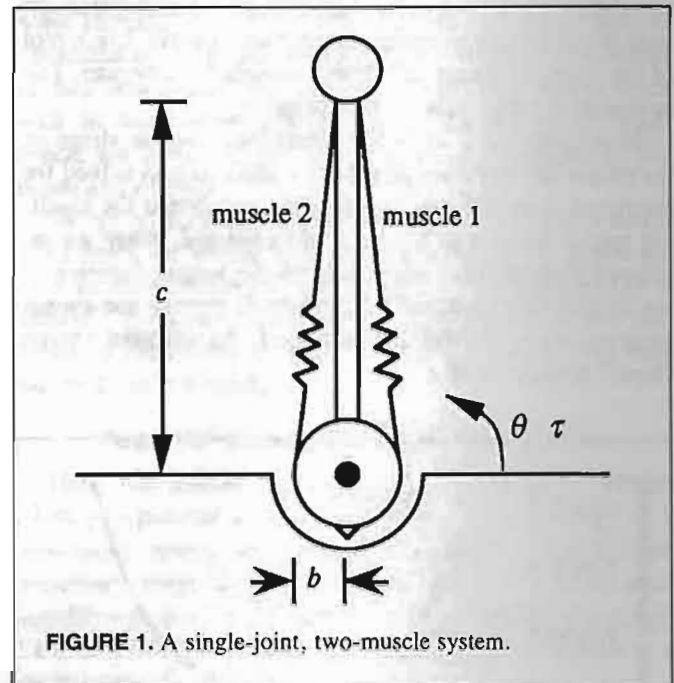
### Energy of a Spring-Like System

A characteristic of a muscle is that it tends to produce more force as it is stretched. The rate of change in muscle force with respect to muscle length is termed *stiffness*, and is related to both the mechanics of the muscle and the reflex system that is monitoring its length. For a muscle to be called a spring-like element, its force-length relationship must be integrable (Hogan, 1985), which basically means that there should be no hysteresis in its length-tension curves. In fact, it has been suggested that an important role of the spinal reflexes is to produce this spring-like property in the face of perturbing effects (Nichols & Houk, 1976).

A property of a spring-like element is that it stores potential energy when it is stretched and gives off energy when it is released. This energy is a scalar quantity and is defined as the definite integral of the force-length curve over a given displacement interval. In the case of a muscle, because force is also a function of the activation parameter, the potential energy in the muscle will be a function of this parameter as well as muscle length. It follows that the task of postural control can be seen as that of selecting muscle parameters to shape a particular potential energy function suitable for the task (cf. Kathib, 1986). Let us describe this energy for a simple system such as that of Figure 1 and select muscle parameters to produce a given behavior. For example, in the case of the muscle model introduced in Shadmehr and Arbib (1992) (which was based on the experimental results of Feldman, 1966) to account for isometric force in a reflexive muscle (i.e., a muscle that is endowed with its reflex circuitry), we have muscle force  $\phi$  as a function of length  $\lambda$ , and control parameter  $\beta$ :

$$\phi = \frac{1}{\alpha} \ln[\exp[\alpha k(\lambda - \beta)] + 1], \quad (1)$$

which is basically a nonlinear spring with a controllable



**FIGURE 1.** A single-joint, two-muscle system.

threshold length parameter  $\beta$ , where  $\lambda > \beta$ . Assuming that the joint angle is  $\theta$ , we have the torque acting on the system by muscles 1 and 2 as

$$\tau = -\frac{d\lambda_1}{d\theta}\phi_1 - \frac{d\lambda_2}{d\theta}\phi_2 - mcg \cos[\theta], \quad (2)$$

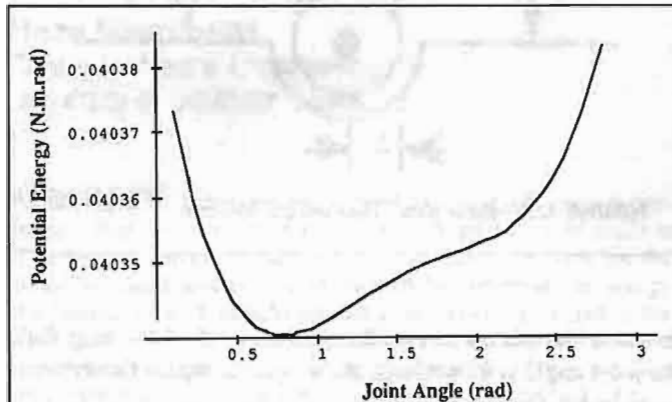
where  $m$  and  $c$  are mass and link length parameters and  $g$  is the gravitational constant. The integral of Equation 2 with respect to  $\theta$  is the potential energy of the system. Clearly, without muscle activation, that is, when  $\phi_1 = \phi_2 = 0$ , the system is unstable. To stabilize it, we can solve for muscle forces so that an equilibrium position  $\theta_E$  is held with a specified joint stiffness  $k$ , where  $k = d\tau/d\theta$ . A minimum value for this stiffness can be derived from stability analysis (Shadmehr & Arbib, 1992):  $k < -mcg \sin[\theta_E]$ . From this we can write the total torque acting on the joint when the system is near equilibrium:

$$\tau = mcg(\cos[\theta_E] - \kappa \sin[\theta_E](\theta - \theta_E) - \cos[\theta]). \quad (3)$$

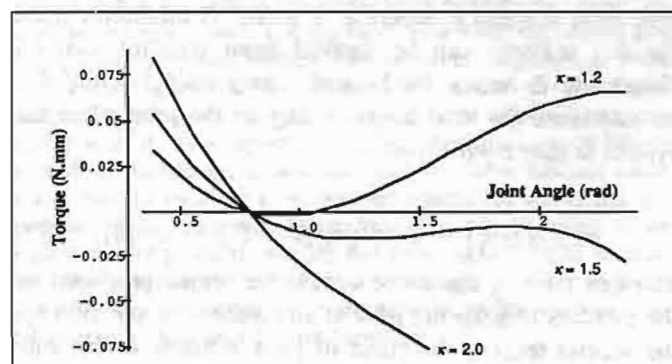
The first term in the above sum is the torque produced by the muscles to keep the joint at an equilibrium position  $\theta_E$ , the second term is the effect of joint stiffness as the limb veers from equilibrium, and the third term is the torque from the gravitational pull. The variable  $\kappa$  in Equation 3 is to indicate the actual joint stiffness as a multiple of the minimum that is prescribed by our stability analysis (therefore,  $\kappa \geq 1$ ). To maintain the limb at a specified position with a specified stiffness, we need to find the muscle parameters that in Equation 2 produce a torque function that near equilibrium ( $\theta = \theta_E$ ) is a good approximation of Equation 3. Using the parameter values for the muscle model of Equation 1 from Shadmehr and Arbib (1992), we solved for  $\beta_1$  and  $\beta_2$ , for  $\theta_E = \pi/4$  and  $\kappa = 1.5$ . These muscle param-

ters define a potential energy function that has a minimum only at the desired equilibrium position. Figure 2 is a plot of the potential energy of this musculoskeletal system. The system is globally stable at this position.

To examine the effect of joint stiffness on the shape of the torque function, we varied joint stiffness and solved for the required muscle parameters and then plotted the resulting torque function in Figure 3. As expected, when we increased joint stiffness, the slope of the torque function at the equilibrium position (i.e., where it crosses the torque axis) increased. As stiffness increased, the potential energy "bowl" became steeper.



**FIGURE 2.** Muscle control parameters are solved for so that the resulting torque function near the equilibrium position (i.e., Equation 2) approximates the torque function derived from stability analysis (i.e., Equation 3). Desired equilibrium position is 45°, and stiffness of the joint is set at 1.5 times the minimum (i.e.,  $\kappa = 1.5$ ). This figure shows the potential energy of the system defined by the resulting muscle control parameters. The actual equilibrium position is at 45°, and the system has a single basin of attraction.



**FIGURE 3.** The effect of increased joint stiffness on the torque function in Equation 5. Muscle activation parameters were solved for so that equilibrium position was maintained at  $\pi/4$  while  $\kappa$  was set at 1.2, 1.5, and 2.0 times the minimum required for stability. The equilibrium position of the system is at that joint angle where the torque function is zero. For the case where  $\kappa = 1.2$ , we see that the system has two equilibrium positions, but only one is stable (where the slope of the torque function is negative). The effect of increase in stiffness is an increase in the slope of the torque function at equilibrium.

### Modules and Encoding of the Motor Space

By approximating the torque function in Equation 3 around an equilibrium position, we have a relatively simple way of mapping a multiple of the minimum joint stiffness,  $\kappa$ , to muscle parameters,  $\beta_1$  and  $\beta_2$ , at a particular equilibrium position,  $\theta_E$ . Let us call the function that does this  $q$ :

$$q: \{\theta_E, \kappa\} \rightarrow \{\beta_1, \beta_2\}. \tag{4}$$

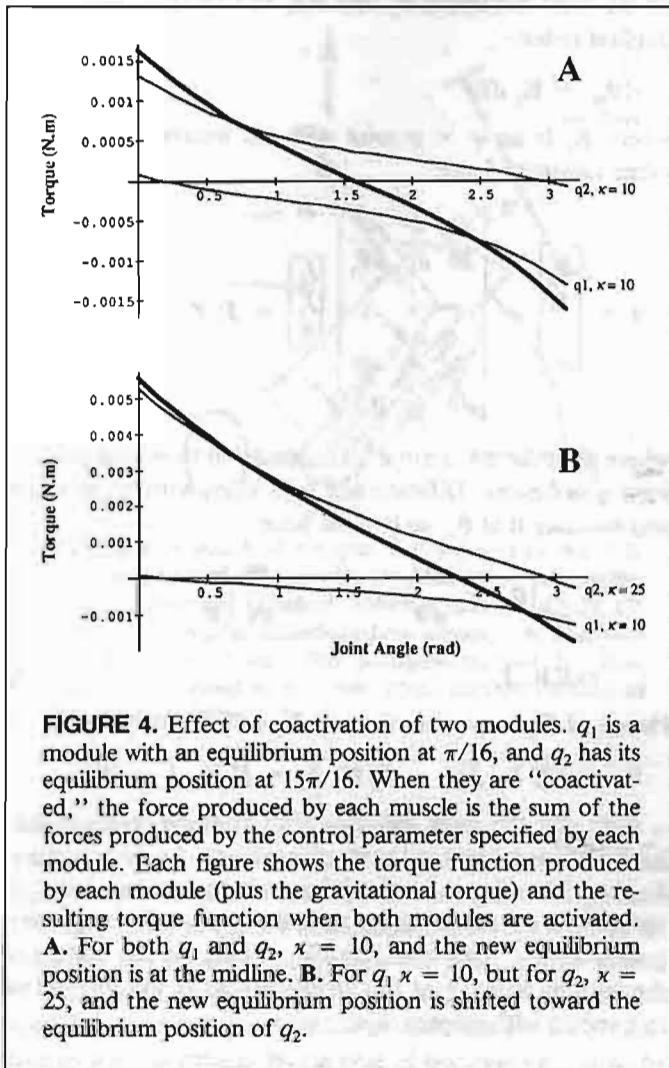
Consider two instances of the function  $q$ , where  $q_1: \kappa \rightarrow \{\beta_{11}, \beta_{21}\}$  is the mapping at  $\theta_E = \pi/16$ , and  $q_2: \kappa \rightarrow \{\beta_{12}, \beta_{22}\}$  is the mapping at  $\theta_E = 15\pi/16$ , that is, near the extremes of the work space for our single-joint system. For example, the map  $q_1$  specifies a set of muscle threshold lengths that in turn specify a set of torque functions, all of which are zero at  $\theta = \pi/16$ . Each torque function is identified by its derivative at  $\pi/16$ , where this derivative is the stiffness of the joint. We can think of  $q_1$  as a hard-wired postural module whose function is to assign muscle control parameters that enable the stiffness of the joint to be modulated without a change in the equilibrium position of the limb. This postural module is a stiffness controller: The single input to this module is the desired stiffness multiple  $\kappa$ , so the larger the input that the module receives, the more it "activates" the muscles and the stiffer the joint gets.

It is possible to devise a scheme in which we have a "row" of postural modules, each encoding an equilibrium position between the two extremes of the work space, coarsely encoding the reachable space through a finite set of stiffness controllers. But there is another way of encoding this space with only the modules in the extremes of the work space: Recall that each module is responsible for an equilibrium position, and it maps its input (the desired stiffness) to a pair of muscle parameters for this position. What happens when two modules are activated simultaneously? For a given muscle, each module would specify an activation parameter, and perhaps the activation is summed to produce the torque function for that muscle. In the case of our reflexive muscle model, where more force is generated by a reduction in the threshold length of the muscle, we simulated activation of two modules by summing the force produced by a muscle for each assigned activation parameter. For example, if for muscle 1 in Figure 1 module  $q_1$  assigns  $\beta_{11}$  and  $q_2$  assigns  $\beta_{12}$ , the force function produced by that muscle will be

$$\phi_1[\lambda_1] = \frac{1}{\alpha} \left( \ln[\exp[\alpha\kappa(\lambda_1 - \beta_{11})] + 1] + \ln[\exp[\alpha\kappa(\lambda_1 - \beta_{12})] + 1] \right).$$

This kind of segmentation of the motor space is useful because of the properties of the system when both postural modules are activated. We can illustrate this by implementing functions  $q_1$  and  $q_2$  and see where the new equilibrium position of the system will be as the input to the modules is gradually increased (i.e., as the value of their input parameter  $\kappa$  is increased). This is shown in Figure 4. Initially, we





set the input for both  $q_1$  and  $q_2$  to  $\kappa = 10$  (Figure 4A). The resulting torque function for the system will have an equilibrium position at the midline, that is, at  $\pi/2$ . In Figure 4B,  $q_2$  was further activated so that it corresponded to a  $\kappa = 25$ . This made the equilibrium position of the system shift toward the equilibrium position of  $q_2$ , that is,  $15\pi/16$ .

By varying the input to  $q_1$  and  $q_2$ , is it possible to produce an equilibrium position anywhere between the joint extremes, and can the joint stiffness at this position be controlled as well? In other words, how much control over equilibrium position and stiffness is possible when only a linear combination of torque functions produced by  $q_1$  and  $q_2$  are available? To provide an answer, we solved for the equilibrium position and stiffness of the system when the input to  $q_1$  and  $q_2$  was varied from  $\kappa = 5$  to 50 by increments of 5. Figure 5 is the description of how the two modules encoded the motor space: A point on the grid represents the equilibrium position of the system and its stiffness when input to  $q_1$  and  $q_2$  was as specified (ranging from 5 to 50, in increments of 5). For example, maximum stiffness occurred at  $\pi/2$  when the input to  $q_1$  and  $q_2$  was  $\kappa = 50$ . On the other hand, if we decreased the input to  $q_1$  and maintained the input to  $q_2$ , the equilibrium position shifted to-

ward  $15\pi/16$ , that is, the zero point for all torque function generated by  $q_2$ . Based on our assumptions regarding action of two coactivated modules, two postural modules, each with an equilibrium position at the extreme of the work space, are sufficient for production of any equilibrium position and stiffness.

To apply these postural modules for control of a multi-joint limb, we would have to know how varying activation of a module changes a limb's stiffness (stiffness will be a matrix). Because muscles are spring-like, there are mechanical limitations on the form of this stiffness. We next derive these constraints:

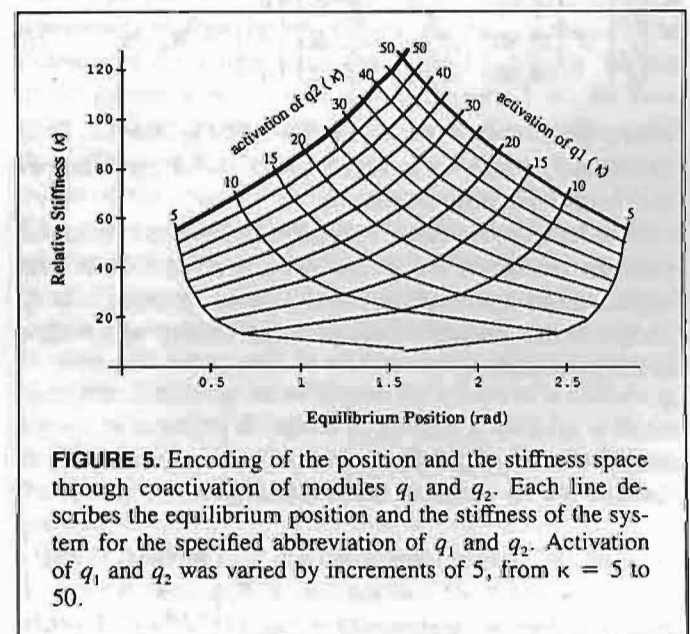
### Potential Energy of a Multijoint System

Here we follow the approach introduced by Hogan (1985) to provide a definition of the potential energy of a multijoint, spring-like system. Our goal is to derive some necessary properties of a musculoskeletal structure near equilibrium and also to introduce the nomenclature that will be useful when we extend the idea of postural modules to a multijoint system.

In the case of a limb with  $n$  degrees of freedom, the configuration of the limb is an  $n$ -dimensional vector  $\theta$ . Assuming that skeletal segments are rigid and muscles have only one point of origin and insertion, based on kinematics we can always define a unique function that maps  $\theta$  to a vector of muscle lengths  $\lambda$ , where  $\lambda$  is an  $m$ -dimensional vector. At a given limb configuration  $\theta$ , and activation vector  $\beta$ , the potential energy stored in the system is the sum of the potential energy of each element plus the gravitational energy:

$$P_{\text{tot}}[\theta, \beta] = \sum_{i=1}^m p[\lambda_i[\theta], \beta_i] + p_g[\theta]. \quad (5)$$

Rather than representing this function in joint coordinates, it is often useful to visualize it in terms of the position of



the tip of the limb. For this we need to define  $\theta$  as a function of the position of the end-effector, as denoted by a three-dimensional vector  $\mathbf{x}$ . The gradient of this function with respect to  $\mathbf{x}$  is a force field with zero curl:

$$\mathbf{F}[\mathbf{x}, \boldsymbol{\beta}] = -\nabla_{\mathbf{x}} p_{tot}[\mathbf{x}, \boldsymbol{\beta}] = \begin{bmatrix} -dp/dx \\ -dp/dy \\ -dp/dz \end{bmatrix} = \begin{bmatrix} f_x(x, y, z) \\ f_y(x, y, z) \\ f_z(x, y, z) \end{bmatrix} \quad (6)$$

$$\text{Curl } \mathbf{F} = \nabla \times \mathbf{F} = \begin{bmatrix} \frac{df_z}{dy} - \frac{df_y}{dz} \\ \frac{df_x}{dz} - \frac{df_z}{dx} \\ \frac{df_y}{dx} - \frac{df_x}{dy} \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix} \quad (7)$$

where  $\nabla = [i \, d/dx + j \, d/dy + k \, d/dz]$ ,  $\mathbf{F} = [if_x + kf_z]$ , and  $\times$  is the cross-product. Assume that the activation vector  $\boldsymbol{\beta}$  is such that there is at least one local minimum in the potential energy function of Equation 5 in the work space. Let us specify this position by the vector  $\theta_0$  in joint coordinates and  $\mathbf{x}_0$  in terms of the position of the end-effector. For some level of activation  $\boldsymbol{\beta}$ , the vector field  $\mathbf{F}(\mathbf{x})$  may be nonlinear, but at  $\mathbf{x}_0$  we can represent it by a Taylor series expansion:

$$\mathbf{F}[\mathbf{x}] = \mathbf{F}[\mathbf{x}_0] + \left. \frac{d\mathbf{F}}{d\mathbf{x}} \right|_{\mathbf{x}=\mathbf{x}_0} \Delta\mathbf{x} + \text{higher-order terms.}$$

Because  $\mathbf{x}_0$  is a local minimum,  $\mathbf{F}(\mathbf{x}_0) = 0$ . For a sufficiently small displacement from  $\mathbf{x}_0$ , the higher-order terms in the expansion may be neglected, leaving us the following relationship between force and displacement at  $\mathbf{x}_0$ :

$$\begin{bmatrix} f_x \\ f_y \\ f_z \end{bmatrix} = \begin{bmatrix} df_x/dx & df_x/dy & df_x/dz \\ df_y/dx & df_y/dy & df_y/dz \\ df_z/dx & df_z/dy & df_z/dz \end{bmatrix} \begin{bmatrix} dx \\ dy \\ dz \end{bmatrix} = \mathbf{K}_s \, d\mathbf{x}, \quad (8)$$

where  $\mathbf{K}_s$  is called the *end-point stiffness matrix*. From Equation 7, where we have the condition for zero curl, we see that  $\mathbf{K}_s$  must be symmetric.

Now let us prove that if end-point stiffness is symmetric, then the change in the torque field as a function of joint angle, called *joint stiffness*, will also be symmetric at  $\theta_0$ . Torque is the gradient of the potential energy with respect to the joint angles.

$$\boldsymbol{\tau}[\boldsymbol{\theta}] = -\nabla_{\boldsymbol{\theta}} p_{tot}[\boldsymbol{\theta}, \boldsymbol{\beta}] = \begin{bmatrix} -dp/d\theta_1 \\ \vdots \\ -dp/d\theta_n \end{bmatrix} = \begin{bmatrix} \tau_1(\theta_1, \dots, \theta_n) \\ \vdots \\ \tau_n(\theta_1, \dots, \theta_n) \end{bmatrix}$$

At the local minimum  $\theta_0$ , the  $\boldsymbol{\tau}(\boldsymbol{\theta})$  can be approximated to the first order:

$$\boldsymbol{\tau}[\boldsymbol{\theta}_0] = \mathbf{K}_j \, d\boldsymbol{\theta}, \quad (9)$$

where  $\mathbf{K}_j$  is an  $n \times n$  joint stiffness matrix. Let us now relate torque to force:

$$\boldsymbol{\tau} = \begin{bmatrix} \tau_1 \\ \tau_2 \\ \vdots \\ \tau_n \end{bmatrix} = \begin{bmatrix} dx & dy & dz \\ d\theta_1 & d\theta_1 & d\theta_1 \\ \vdots & \vdots & \vdots \\ dx & dy & dz \\ d\theta_n & d\theta_n & d\theta_n \end{bmatrix} \begin{bmatrix} f_x \\ f_y \\ f_z \end{bmatrix} = \mathbf{J}_s^T \mathbf{F}, \quad (10)$$

where  $\mathbf{J}_s$  is the differential transformation from end point to joint coordinates. Differentiate both sides with respect to  $\boldsymbol{\theta}$  and evaluate it at  $\theta_0$ , so that we have

$$\begin{aligned} \mathbf{K}_j &= \left. \frac{d\boldsymbol{\tau}}{d\boldsymbol{\theta}} \right|_{\boldsymbol{\theta}_0} = \frac{d\mathbf{J}_s^T}{d\boldsymbol{\theta}} \mathbf{F}[\mathbf{x}_0] + \mathbf{J}_s^T \frac{d\mathbf{F}}{d\mathbf{x}} \frac{d\mathbf{x}}{d\boldsymbol{\theta}} \\ &= \mathbf{J}_s^T \mathbf{K}_s \mathbf{J}_s. \end{aligned} \quad (11)$$

Hence, if  $\mathbf{K}_s$  is symmetric, then  $\mathbf{K}_j$  will be symmetric:

$$\mathbf{K}_j^T = (\mathbf{J}_s^T \mathbf{K}_s \mathbf{J}_s)^T = \mathbf{J}_s^T \mathbf{K}_s^T \mathbf{J}_s = \mathbf{J}_s^T \mathbf{K}_s \mathbf{J}_s = \mathbf{K}_j.$$

Note that the joint and end-point stiffness matrices will have only negative (and real) eigenvalues: As the system is displaced from  $\mathbf{x}_0$  (a local minimum in the potential energy function) by a small amount  $d\mathbf{x}$ , the force field is approximately  $\mathbf{F}(\mathbf{x}_0 + d\mathbf{x}) = \mathbf{K}_s \, d\mathbf{x}$ . The change in the energy of the system because of this displacement is the integral of this vector with respect to  $\mathbf{x}$ :

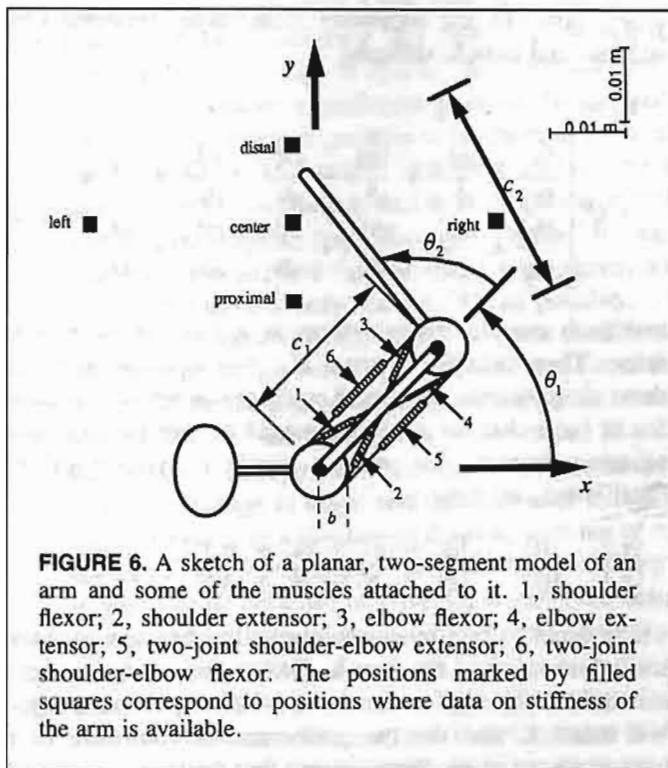
$$\Delta p_{tot} = -\int \mathbf{F}^T \, d\mathbf{x} = -\frac{1}{2} d\mathbf{x}^T \mathbf{K}_s \, d\mathbf{x}.$$

Because, by definition,  $\mathbf{x}_0$  is a local minimum in  $p_{tot}$ ,  $\Delta p_{tot}$  is a positive number, that is, the system gains energy because of this displacement; therefore,  $d\mathbf{x}^T \mathbf{K}_s \, d\mathbf{x} < 0$  and  $\mathbf{K}_s$  is a negative definite (n.d.) matrix. Because  $\mathbf{K}_s$  is symmetric, all of its eigenvalues will be real. Furthermore, because  $\mathbf{K}_s$  is a symmetric n.d. matrix, all of its eigenvalues will be negative. The same procedure can be used to show that  $\mathbf{K}_j$  is a symmetric n.d. matrix.

These derived properties of the restoring force field are based on the assumption that the neuromuscular system is spring-like. In the next section, we look at results of experiments in which this field has been measured.

### Voluntary Control of Stiffness in Man

Consider the problem of how to control the six muscles of our two-joint system in Figure 6 so that a certain set of torques are produced about the joints. In this perspective, single-joint muscles are sufficient to generate all the necessary torques, and multijoint muscles appear to be a redundancy in the system. Gielen and Van Zuylen (1986) proposed that the redundancies can be dealt with by defining a tensor whose components depend on how the direction of



torque for each muscle correlates with the direction of torque of other muscles (a kind of covariance matrix). The eigenvectors of this tensor are the *principal components* (Jolliffe, 1986) of the six-dimensional space spanned by the muscles, and it has been proposed that a linear combination of the muscle torque vectors along each principal direction specifies the relative activation of each muscle for production of a given torque. In the case of our two-joint arm, the procedure would be to define a two-dimensional space with an orthogonal coordinate system that represents torques in the flexion/extension directions for each joint. For muscle  $i$ , its activation leads to a torque vector  $\tau^i$  whose components are the torque contributions in the two directions. Next, define a symmetric matrix  $D$ :

$$D_{ij} = |\tau^i| |\tau^j| \cos [\psi_{ij}] \quad i, j = 1 \text{ to } 6,$$

where  $\psi_{ij}$  is the angle between  $\tau^j$  and  $\tau^i$ . This matrix will have two non-zero eigenvalues. Call the associated eigenvectors  $V_1$  and  $V_2$ .  $V_1$  is meant to represent the relative torque production in the six muscles so that only a unit torque about the shoulder joint (joint 1) is produced (similarly for  $V_2$ ). To produce some desired torque  $\tau_1^d$ , simply produce muscle torques as specified by  $\tau_1^i V_i$  (cf. Pellionisz & Peterson, 1988).

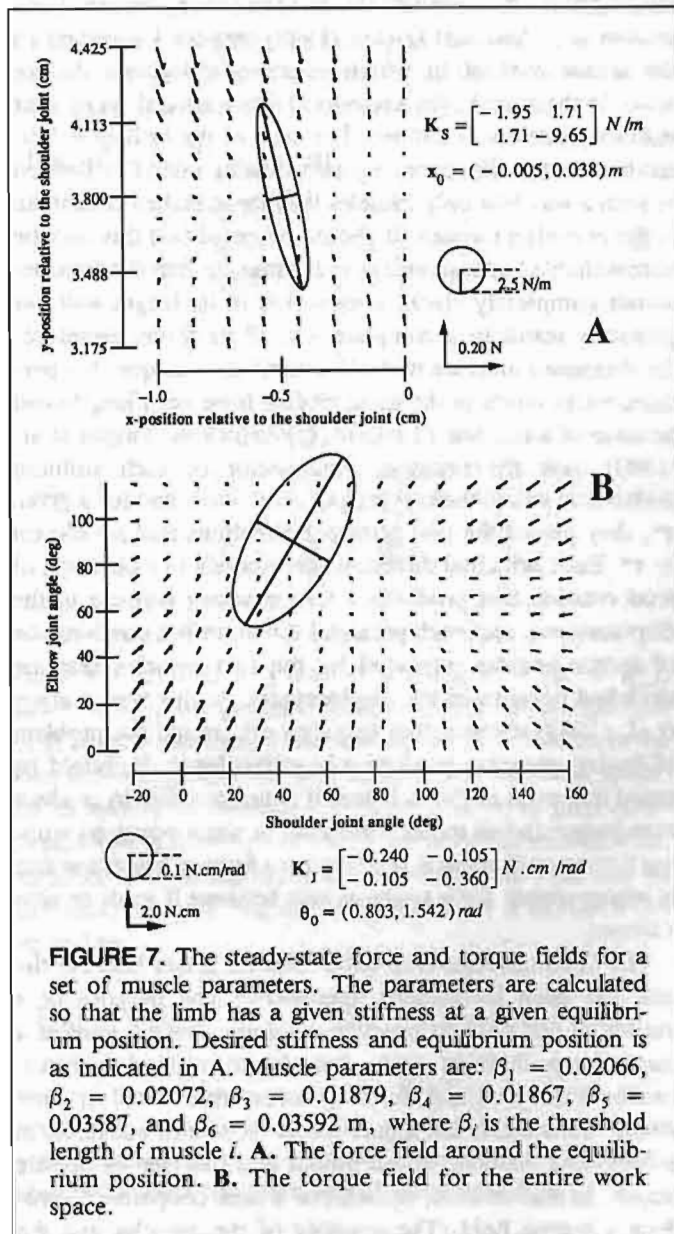
There are two problems with this approach: (a) Components of an eigenvector will sometimes have a sign that is opposite to the torque direction that is produced when the corresponding muscle is activated, forcing the muscle to produce a negative force; and (b) to keep the joint in equilibrium, that is, to produce zero net torque, this approach would specify zero torque for each muscle, leading to zero stiffness (likely an unstable situation) and no way to vary

this stiffness. To overcome the first limitation, Jongen, Denier van der Gon, and Gielen (1989) proposed a variant of the tensor method in which negative activations do not arise. In their work, for a two-joint arm a special set of joint stiffness matrices is defined: For each of the orthogonal directions in the  $d\theta$  space, a joint stiffness matrix is defined in such a way that only muscles that are stretched contribute to the restoring torques. It should be noted that this may be unreasonable because unless every muscle that shortens becomes completely slack, a reduction in its length will not generally result in a complete loss of its force; therefore, the shortened muscles will still contribute a torque, but perhaps not as much as the muscles that have been lengthened because of a rotation of a joint. Nevertheless, Jongen et al. (1989) took the principal eigenvector of each stiffness matrix and called these *principal directions*, and for a given  $\tau^d$ , they pieced the two principal directions that are closest to  $\tau^d$ . Each principal direction corresponds to a direction of joint rotation that produces a torque vector colinear to the displacement, and each principal direction is a combination of torque vectors produced by the two muscles that are stretched because of the displacement. In this way, Jongen et al. (1989) showed that they can get around the problem of having muscles produce a negative force. It should be noted that even in this scheme, it is not possible to produce zero torque (so as to keep the joint at some position) without having each muscle produce zero force, a condition that is inappropriate for a postural task because it leads to zero stiffness.

The dilemma regarding extra muscles arises because the task has been formulated improperly: The purpose of a muscle is not only to produce a torque about a joint at a particular position in space, but also to respond to disturbances if the joint is affected by an external force. In other words, a muscle is not a pure torque generator but rather is a compliant element whose output is a function of muscle length. In this context, muscles of a limb cooperate to produce a torque field. The quantity of the muscles and the complexity of their length-tension curves contribute to the richness of the torque fields that can be produced. In case of the human arm, the two "extra" muscles (i.e., the two-joint muscles), in principle, allow the nervous system to set the stiffness of the limb in a way that matches the requirements of the task that is being performed (Hogan, 1985): By varying the relative activation of the muscles, the shape and orientation of a force field can be rotated while its equilibrium point is kept constant. The arm might be made compliant in one direction (to accommodate an external kinematic constraint such as an unyielding surface) and stiff in another direction (to minimize the effects of a disturbing force), or it might be useful to present a uniform stiffness in all directions. In effect, these conditions would require the ability to manipulate all the elements of the stiffness matrix  $K_S$ .

Let us describe how one can control stiffness while maintaining the same equilibrium position for the two-joint system of Figure 6. The task is to maintain the limb at  $\theta = \theta_0$





with an end-point stiffness specified by  $K_s$ . Based on our discussion in the last section, we know that due to the spring-like properties of the system,  $K_s$  will be symmetric and negative definite. Using Equation 11,  $K_s$  can be mapped to  $K_J$ , which in turn can be mapped to a muscle stiffness matrix. Using these kinematic relationships (Mussa-Ivaldi, Morasso, & Zaccaria 1988), we can solve for muscle forces so as to produce a desired torque vector and a desired end-point stiffness. For the system of muscles in Figure 6, we have the following kinematics:

$$\begin{aligned} \lambda_1 &= (\pi/2 - \theta_1)b + c_1/2, \\ \lambda_2 &= (\theta_1 + \pi/2)b + c_1/2, \\ \lambda_3 &= (\pi/2 - \theta_2)b + c_1/2, \\ \lambda_4 &= (\theta_2 + \pi/2)b + c_1/2, \\ \lambda_5 &= (\theta_1 + \pi/2)b + (\theta_2 + \pi/2)b + c_1, \text{ and} \\ \lambda_6 &= (\pi/2 - \theta_1)b + (\pi/2 - \theta_2)b + c_1, \end{aligned}$$

which gives us the following relationship between joint stiffness and muscle stiffness:

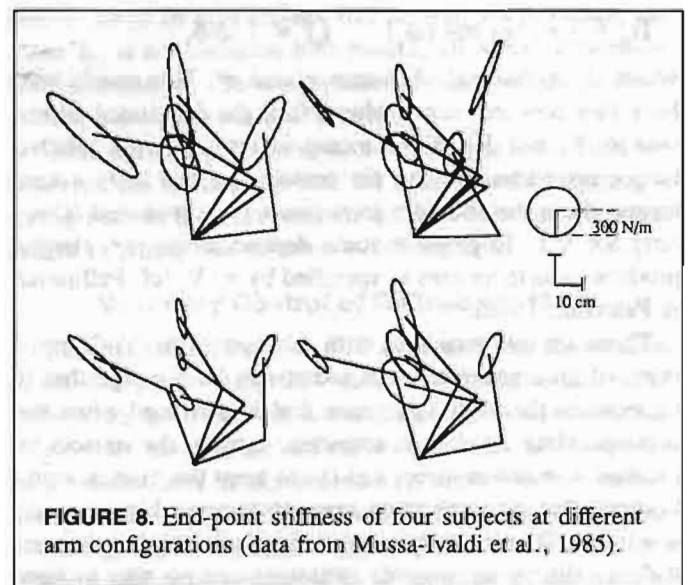
$$K_J = -b^2 \begin{bmatrix} \frac{d\phi_1}{d\lambda_1} + \frac{d\phi_2}{d\lambda_2} + \frac{d\phi_5}{d\lambda_5} + \frac{d\phi_6}{d\lambda_6} & \frac{d\phi_5}{d\lambda_5} + \frac{d\phi_6}{d\lambda_6} \\ \frac{d\phi_5}{d\lambda_5} + \frac{d\phi_6}{d\lambda_6} & \frac{d\phi_3}{d\lambda_3} + \frac{d\phi_4}{d\lambda_4} + \frac{d\phi_5}{d\lambda_5} + \frac{d\phi_6}{d\lambda_6} \end{bmatrix}, \quad (12)$$

and each muscle stiffness term is a function of muscle force. Therefore, for a given  $K_J$ , this equation gives us three simultaneous, nonlinear equations in terms of muscle forces (note that we get three instead of four simultaneous equations because the stiffness matrix is symmetric). At equilibrium, we have

$$\begin{bmatrix} \tau_1 \\ \tau_2 \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \end{bmatrix} = b \begin{bmatrix} \phi_1 - \phi_2 - \phi_5 + \phi_6 \\ \phi_3 - \phi_4 - \phi_5 + \phi_6 \end{bmatrix},$$

which gives us two more equations. But because we have six unknowns (the six muscle forces), we need one more constraint. Note that in the above we have pairs of antagonist muscles, and the two-joint muscles contribute to a torque at both joints. If we assume that the torque produced by each pair of antagonist muscles (e.g., the single-joint shoulder flexor and shoulder extensor) is zero at equilibrium, then we can split the above relation into three simultaneous equations. This, along with the stiffness constraint, gives us six equations to solve for muscle force.

As an example, assume that we wish the arm in Figure 6 to produce end-point stiffness  $K_{xx} = -1.9$ ,  $K_{yy} = K_{yx} = 1.7$ , and  $K_{yy} = -9.6$  N/m at the center position in that figure. Converting the end-point stiffness to joint stiffness gives us  $K_{J11} = -2.4$ ,  $K_{J12} = K_{J21} = -1.05$ , and  $K_{J22} = -3.6$  N.mm/rad. Next, we solved the resulting six simultaneous equations for muscle force and then solved for the control parameter  $\beta$  for each muscle so that it produced this force at the desired limb configuration. Figure 7A shows



the resulting steady-state force field as a function of the position of the end-effector (the coordinate system is centered at the shoulder).<sup>1</sup> It can be seen that the field converges to the desired equilibrium position. The desired stiffness of the field at this position is represented by an ellipse (Mussa-Ivaldi et al., 1985). This field can also be represented in joint coordinates, and in this case our muscle parameters describe a torque field. In Figure 7B, we have drawn the torque field for the entire work space, and we can see that there is only one equilibrium position.

When the end-point stiffness of a number of subjects was measured by Mussa-Ivaldi et al. (1985), they found that the shape and orientation of the stiffness varied systematically with location of the hand in the work space. The data of four subjects is reproduced in Figure 8. Remarkably, the pattern of change in shape and orientation of the end-point stiffness matrix as a function of the configuration of the arm appears to fall into a specific pattern: The orientation of  $\mathbf{K}_s$  is such that its principal eigenvector is along the radial axis of a polar coordinate system centered at the shoulder of the subject (Flash, 1987). The shape of  $\mathbf{K}_s$  is such that the ellipse becomes elongated as the hand approaches the distal boundary of the reachable space. It was reported that shape and orientation of these ellipses remained quite repeatable over time (Mussa-Ivaldi et al., 1985, Shadmehr et al., 1993). In fact, the only variable that changed over repeated measurements of stiffness was the size of the ellipse (determinant of  $\mathbf{K}_s$ ), which the subjects could vary at will.

To account for the systematic changes in the shape and orientation of  $\mathbf{K}_s$ , Mussa-Ivaldi et al. (1985) hypothesized that for the limited work space in which the experiments were conducted, the changes were due to the geometric transformation from joint to end-point coordinates. The idea was that the subjects were keeping shape and orientation of joint stiffness  $\mathbf{K}_j$  constant, and the changes in the shape and orientation of  $\mathbf{K}_s$  were due to the configuration-dependent relationship between the two. Mussa-Ivaldi et al. (1985) and Hogan, Bizzi, Mussa-Ivaldi, and Flash (1987) have reported that their simulations show that this hypothesis essentially agrees with the available data. To see how close the predictions of this hypothesis match the measured results, in Figure 9A we have drawn the measured stiffness of a subject from Mussa-Ivaldi et al. (1985) (Subject A) at five positions. Figure 9B is the plot of the expected end-point stiffness, assuming that the subject was maintaining a constant joint stiffness (joint stiffness was computed from the subject's end-point stiffness matrix at the *center* position). The joint stiffness was then converted to end-point stiffness at other arm configurations by inverting Equation 11 and solving for  $\mathbf{K}_s$ .

By comparing the experimental data in Figure 9A and the predictions in Figure 9B, we see that although the match was not exact (at the position marked *left*, the predicted stiffness is more elongated than the observed stiffness; cf. Flash & Mussa-Ivaldi, 1990), the basic pattern of variations can be attributed to a system that is maintaining a more or less constant joint stiffness. To illustrate this further, in Fig-

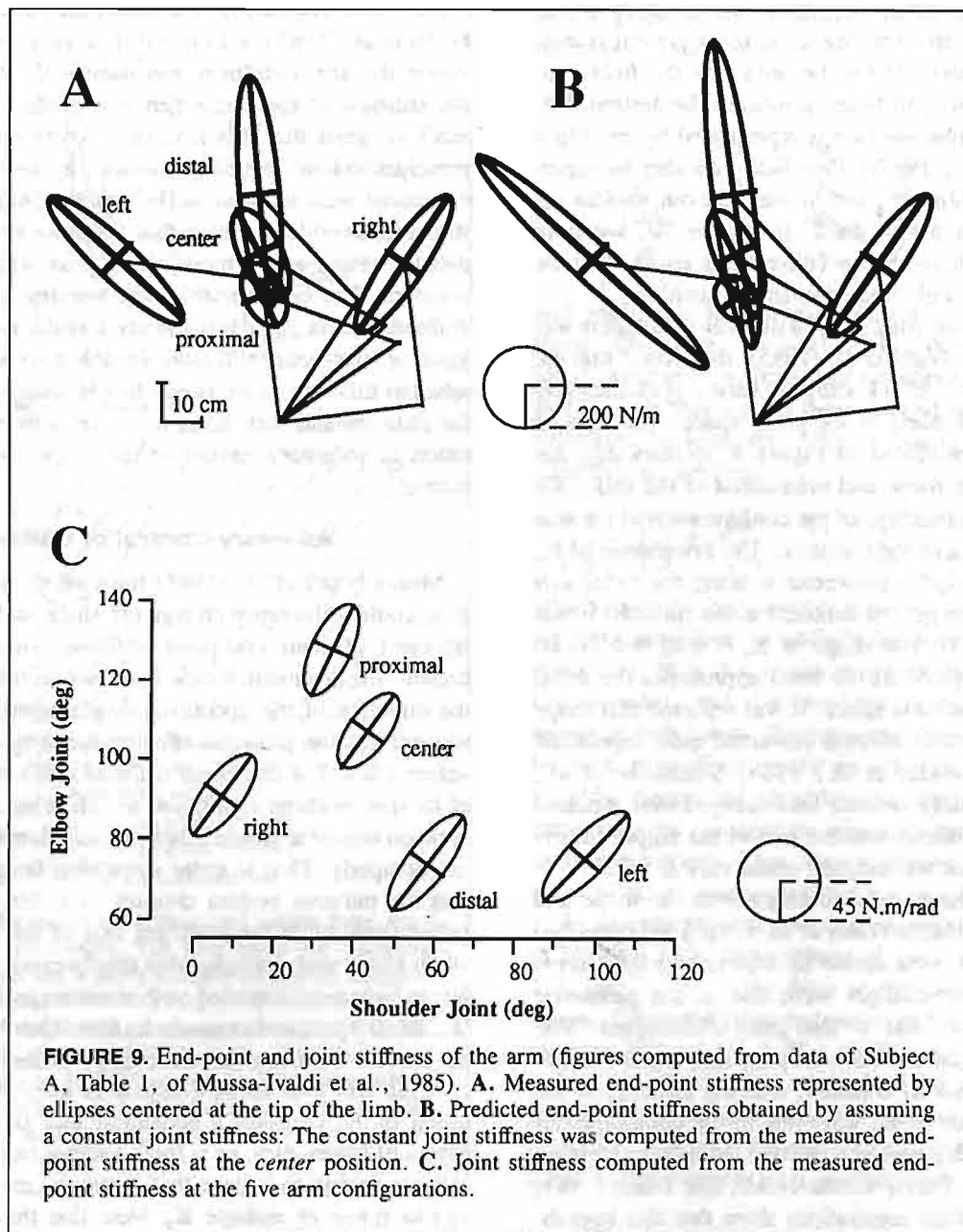
ure 9C we computed joint stiffness for Subject A of Mussa-Ivaldi et al. (1985) and plotted it at the joint configurations where the arm's stiffness was measured. The similarity of the stiffness of the arm when it is plotted in joint coordinates suggests that it is invariant to arm configuration (the principal axis of the ellipse rotates by less than  $10^\circ$  when compared with stiffness at the *center* position). Therefore, it is a reasonable assertion that for these experimental conditions, shape and orientation of joint stiffness were kept constant. The only variable that remains is the size of the stiffness matrix, which is merely a scalar multiple of either joint or end-point stiffness. In the next section, we ask whether this invariance is due to a mechanical limitation on the muscles and their kinematics, or is there a neural limitation in voluntary control of the shape and orientation of stiffness.

### Voluntary Control of Stiffness

Mussa-Ivaldi et al. (1985) have asked whether the subjects could voluntarily change the shape and orientation parameters of their end-point stiffness when faced with a known displacement vector (i.e., when the subjects knew the direction of the upcoming displacement). Their results suggest that the principal effect induced by attempts at voluntary control of end-point stiffness result only in a change of its size, without significant modification of shape or orientation (i.e., the whole matrix is scaled, rather than rotated or reshaped). This is quite surprising because it suggests that the nervous system chooses to stiffen the whole arm rather than rotate the principal axis of the stiffness ellipse when faced with a predictable displacement. These experiments have been repeated with electromyographic feedback (E. Bizzi, personal communication, October, 1989), and the basic conclusions have remained unchallenged.

Is the fact that subjects appear to keep shape and orientation of  $\mathbf{K}_j$  constant a constraint that is rooted in some aspect of kinematics, or is there another explanation? There is some reason to believe that it may be mechanically difficult to rotate or reshape  $\mathbf{K}_j$ : Note that the stiffness of the two-joint muscles contributes to all elements of  $\mathbf{K}_j$ . Perhaps, then, one can hypothesize that voluntary control of arm stiffness is accomplished by modulation of the stiffness of the two-joint muscles, and because these terms appear in all of the elements of  $\mathbf{K}_j$ , an increase in the stiffness of the two-joint muscles merely scales  $\mathbf{K}_j$ . It is a simple matter to show that this hypothesis is false: In Figure 10, we have plotted the form of the end-point stiffness  $\mathbf{K}_s$  as a function of the relative stiffness of the single- and double-joint muscles while the arm was maintained at the *center* position of Figure 9A. It can be seen that only when the stiffness of the double-joint muscles was increased relative to that of the single-joint muscles was there a significant change in the orientation of the end-point stiffness ellipse (compare 1 and 5 in Figure 10). This figure also shows what kind of control of end-point stiffness would be possible if the arm were composed of only single-joint muscles: Compare figures numbered 1, 2, 3, and 4 in Figure 10. Increased stiff-





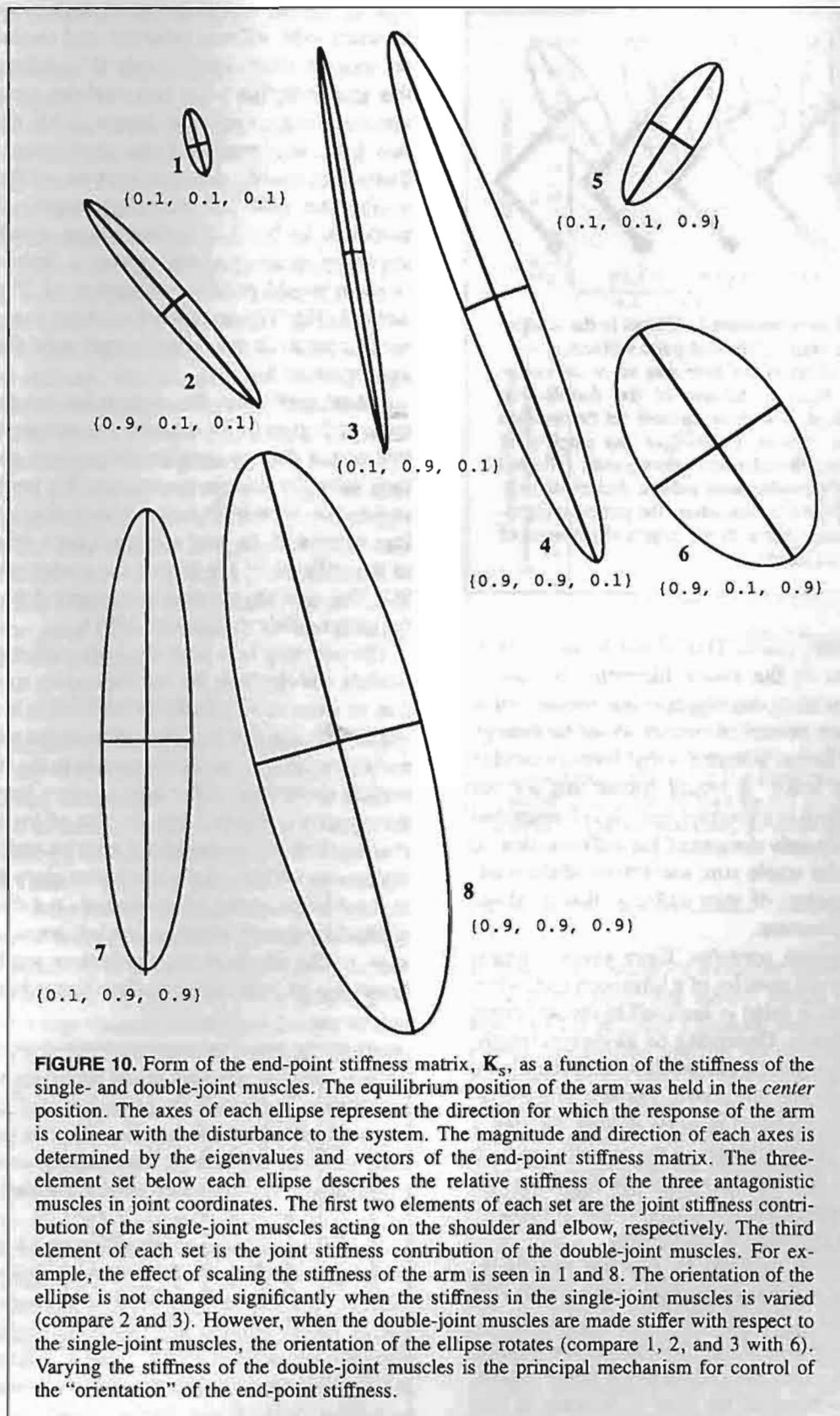
ness in the single-joint muscles did not significantly rotate the end-point stiffness. However, increased stiffness in the two-joint muscles (as in number 5 in Figure 10) rotated the principal axis of the end-point stiffness.

The two-joint muscles give the nervous system the ability to rotate the restoring force field at equilibrium. To illustrate this point, in Figure 11 we have plotted the effect of increase in the relative stiffness of the double-joint muscles while the equilibrium position of the arm was maintained at the *center* position. Increased stiffness of the double-joint muscles rotated the principal eigenvector of the end-point stiffness matrix. From these results we concluded that if the subjects are able to control the stiffness of their double-joint muscles independently of the stiffness of their single-joint muscles, then they should be able to significantly change the orientation of the restoring force field. Is their inability

a manifestation of a neural constraint, that is, is it evidence for existence of an intermediate control system that organizes supraspinal access to the muscles? In the following, we forward a conjecture that accounts for the observed constraints in voluntary control of stiffness through a model of postural modules in a two-joint system.

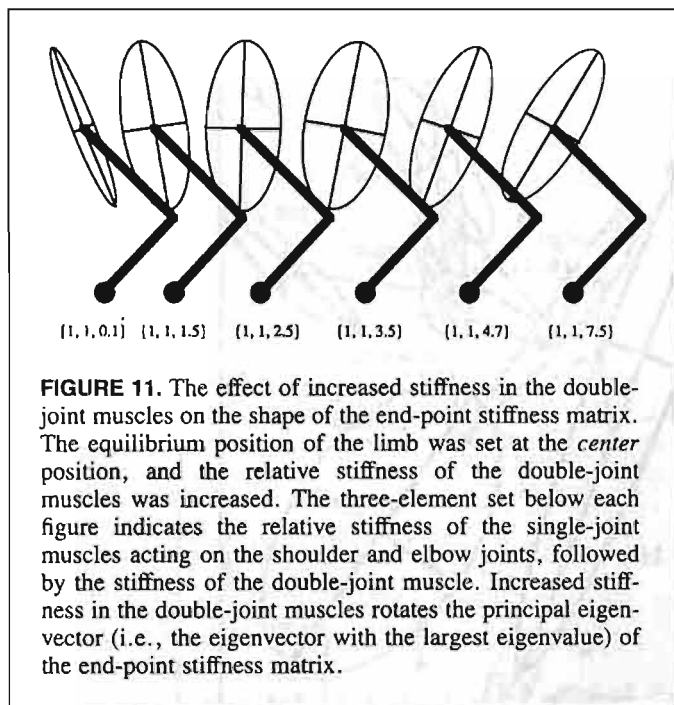
### Postural Modules in the Two-Joint System

A *postural module* is a stiffness controller that specializes in recruiting muscles so that the limb assumes a particular equilibrium position (Mussa-Ivaldi & Giszter, 1993). The constraint of a postural module is that its sole controllable parameter is the stiffness of the limb at this position. Mathematically, each module is a map from input stiffness (which represents the "activation" of the module) to muscle control parameters at a specific limb equilibrium position.



When these values are assigned to the muscle control parameters, a force field is generated that converges at the specified equilibrium position and has the specified stiffness for small displacements about this position. These modules are useful entities because when they are coactivated, the

force fields that they produce add to produce a field that converges to a new equilibrium position. The idea is that the modules should provide us with a mechanism to encode the entire motor space (reachable space and stiffness space) in terms of activation of a few modules situated at the ex-



tremes of the reachable space. This would be an intermediate control system in the motor hierarchy because it would alleviate the need to directly calculate the activation of each muscle; rather, control of posture would be through activation of a set of neural assemblies that form a spanning set of the reachable space. It would appear that for our framework, (a) the input to a postural module is constrained so that one can specify only the size of the stiffness, that is, one can stiffen only the whole arm; and (b) for all the modules, the other parameters of joint stiffness, that is, shape and orientation, are constant.

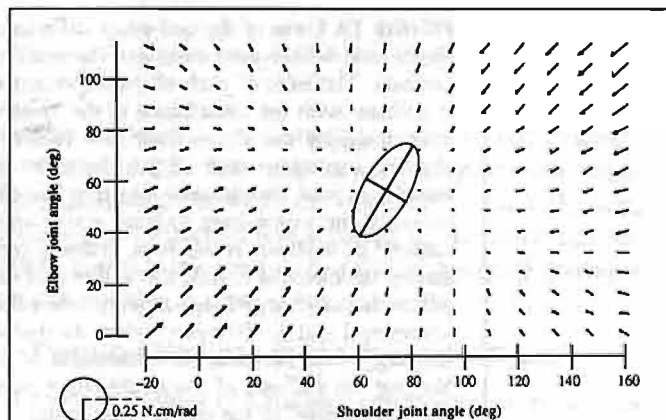
Consider the following scenario: There exists a neural organization that recruits muscles of a limb such that, when this system is excited, a value is assigned to the activation parameter of each muscle. Depending on its current length, a certain elastic energy can be attributed to each muscle by virtue of the activation that it receives. The sum of the elastic energies of all the activated muscles defines the elastic energy of the limb. The mechanics of the muscles are such that they will guide the limb to a joint configuration that produces the minimum in this energy function. This represents an equilibrium position for the system.

Varying the excitation received by a neural module allows us to change the stiffness of the limb without changing its equilibrium position: As we increase the excitation to this neural organization (we represented this by the variable  $\kappa$  in the section on modules and coding of the motor space), we would like the stiffness of the limb to increase in size (i.e., the joint stiffness matrix should be scaled without being rotated or reshaped), but the equilibrium configuration of the limb should remain the same. Now consider a small set of these postural modules, each module of which recruits the muscles, so that when the set is activated it produces a converging torque field at another limb config-

uration. The constraint is that all such modules produce an invariant joint stiffness behavior, and modulation of the input to each module scales only this stiffness. A quality of this system is that when two modules are activated simultaneously, their respective torque fields add to produce a new field, with possibly a new equilibrium position for the limb. Presumably, one can increase stiffness at this new equilibrium point by increasing coactivation of the two modules. To be able to produce an equilibrium position anywhere in the reachable space, a limb of  $m$  degrees of freedom would require a minimum of  $2^m$  modules (Shadmehr, 1991). The number of modules is equal to the number of corners in the  $n$ -dimensional solid that represents the joint space of the limb.

Let us implement this system for the two-joint mechanism of Figure 6. To describe the action of a module, we first pick a desired equilibrium position and stiffness, and then solve for muscle parameters. We performed this procedure for an equilibrium position situated at one of the four corners of the joint space and for a joint stiffness equal to the stiffness of the arm at the *center* position of Figure 9C. The resulting muscle parameters define a torque field for each module (Shadmehr, 1991).

The next step is to scale the joint stiffness matrix for each module and describe the corresponding muscle parameters that produce this stiffness at the module's equilibrium position. The scale of the joint stiffness for module  $i$  is specified by variable  $\kappa_i$ , and corresponds to the activation of that module (because a stiffer field requires larger activation of the muscles). Figure 12 is the plot of the torque field that results when the two modules with an equilibrium position at opposite corners of the reachable space (the first module at shoulder-extended, elbow-flexed, and the second module at shoulder-flexed, elbow-extended) are equally activated at  $\kappa = 1$ . The ellipse at the equilibrium position of this field represents its joint stiffness. Note that it has the same shape



**FIGURE 12.** The torque field produced when two postural modules are activated. In this case, the modules corresponding to equilibrium positions (shoulder-min, elbow-max) and (shoulder-max, elbow-min) are activated equally. The resulting stiffness at the new equilibrium position is plotted.



and orientation as the ellipses at the center position of Figure 9C.

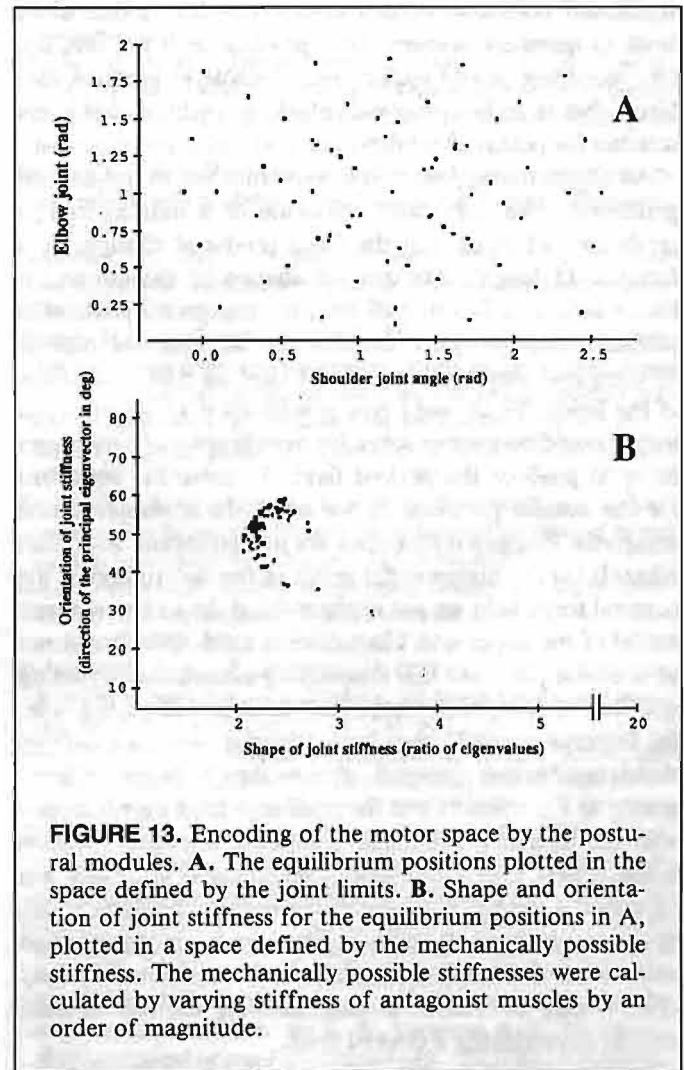
Through coactivation of the modules, it is possible to encode the joint space. To show this, we calculated the equilibrium position and stiffness of the resulting torque field when the four modules are each activated at three levels:  $\kappa = 0.2, 2.2,$  and  $5.2$  times the magnitude of stiffness as measured by Mussa-Ivaldi et al. (1985). The equilibrium space spanned by activation of the modules is plotted in Figure 13A. In Figure 13B, the shape and orientation parameters of each field are plotted as a function of the mechanically possible space for that parameter. The change in activation of the modules results in a change in the size of the resulting joint stiffness, which spans two orders of magnitude. From these simulations, we can make the following conclusions: (a) The more a module is activated, the more the equilibrium position of the system moves toward the equilibrium position encoded by that module. It can be seen that the entire reachable space was encoded. (b) Although the activation of the four modules is varied by more than twenty-fold, the shape (ratio of the eigenvalues of the joint stiffness matrix) and orientation (direction of the principal eigenvector of the joint stiffness matrix) of the resulting field remain relatively constant.

### Discussion

Because a spring-like system stores potential energy as it is stretched, for any combination of muscles and links an energy function can be defined: The integral of muscle force with respect to muscle length is the energy stored in the muscle. The energy stored in the limb is the sum of the energies of each muscle, plus the potential energy of the mass in the links. Using kinematic transformations, this energy function can be written in terms of joint angles of the limb. Any local minimum in this energy landscape is a possible equilibrium position for the limb. The object of postural control is to assign muscle control parameters so that this energy landscape has a minimum at a desired joint configuration, and a particular shape near this position. Equivalently, the task is to approximate a desired torque/force field with a set of compliant elements, that is, the muscles. Muscle parameters are assigned so that the resulting field approximates a torque function appropriate for the stiffness near equilibrium.

An alternative to torque function approximation is field construction through a system of modules that coarsely encode the motor space: A postural module is a stiffness controller for a particular equilibrium position (Bizzi et al., 1991, Mussa-Ivaldi & Giszter, 1993). It maps joint stiffness to muscle parameters for a given equilibrium position. A single-joint system can be controlled effectively with two modules positioned near the extremes of the reachable space: By varying the input to each module, it is possible to produce an equilibrium position with a desired stiffness anywhere between the joint extremes.

Because the stiffness of a multijoint system is a matrix (rather than a scalar, as was the case for the single-joint



**FIGURE 13.** Encoding of the motor space by the postural modules. **A.** The equilibrium positions plotted in the space defined by the joint limits. **B.** Shape and orientation of joint stiffness for the equilibrium positions in **A**, plotted in a space defined by the mechanically possible stiffness. The mechanically possible stiffnesses were calculated by varying stiffness of antagonist muscles by an order of magnitude.

system), the concept of a module could not be directly applied to the multijoint system unless we specified which aspect of the stiffness a module was supposed to control at a given equilibrium position. To get insight into this matter, we looked at the data of Mussa-Ivaldi et al. (1985), who measured the stiffness of the human arm at various positions and asked the subjects to change this stiffness voluntarily. By displacing the human arm from an equilibrium position, they had measured the restoring force field and showed it to be essentially curl free, as would be expected from a spring-like neuromuscular system. What was not expected, however, was the observation that subjects were unable to voluntarily change the shape and orientation of the restoring field, although they could readily scale it. The reason why this was surprising is related to the question of actuator redundancy and the role of double-joint muscles: If muscles are viewed only as pure torque generators, then two-joint muscles are redundant, because a given joint torque vector may be produced via activation of only the single-joint muscles on each joint. In previous work (Jongen et al., 1989), the procedure has been to construct principal components of the torque space and project each individual muscle's torque vector onto this vector. A

significant limitation of this work, however, is that if we wish to maintain posture (i.e., produce zero net torque), this procedure would require each muscle to produce zero force, that is, to be completely slack, a condition that is not suitable for postural stability.

An alternative approach is to view muscles as torque field generators: Not only does activation of a muscle lead to production of force, but the force produced changes as a function of length. The rate of change of the torque (or force) field as a function of joint (or end-point) position is stiffness, and two-joint muscles are an essential part of forming and shaping this stiffness (and therefore the shape of the field). We showed how specification of stiffness and torque could be used to solve for muscle control parameters so as to produce the desired field. To solve the equations for the muscle parameters, we made the assumption that antagonist single-joint muscles are in equilibrium with each other. If (a) the higher-order terms in the description of the postural force field are not neglected and (b) a more realistic model of the upper-arm kinematics is used, then it may not be necessary to make this simplifying assumption regarding coupling of antagonist single-joint muscles. In such a case, the force/torque field may be sampled at various positions about equilibrium (instead of assuming a linear stiffness matrix in Equation 8) and the nonlinear field approximated with the available nonlinear compliant elements (Mussa-Ivaldi, 1993). Our recent work (Shadmehr et al., 1993) has described a method for quantifying postural fields as a sum of nonlinear basis functions. This work, in conjunction with detailed kinematic models such as that of Dorney (1991), may be fruitful in understanding the role of each muscle in producing a desired field.

It appears that the two-joint muscles give the nervous system the mechanical ability to orient the principal axis of the force field (where there is most resistance to a displacement). Surprisingly, data of Mussa-Ivaldi et al. (1985) suggested that subjects maintained a relatively constant shape and orientation of their joint stiffness as a function of arm configuration, even when the direction of displacement was known. This may be an important piece of evidence in support of the hypothesis that there exists an intermediate control system that organizes supraspinal access to the muscles of the arm. This organization was represented by a set of postural modules, where each module was a stiffness controller with the ability to recruit muscles to produce a converging torque field with a specific equilibrium position. Activation of this module led to recruitment of muscles so that the field was scaled without being rotated. It was shown that the motor space of a two-joint, six-muscle system could be encoded with four modules, each with an equilibrium position at one of the corners of the rectangle defined by the joint limits. Simulations were done to measure the stiffness of the field that results when two or more modules were activated. It was shown that the shape and orientation of the resulting field at its equilibrium position varied by a small amount, although the size of the field scaled significantly (the determinant of the stiffness matrix

changed by two orders of magnitude). This kind of system, in which stiffness controllers (modules) are coactivated, would account for the kind of limitations that were observed by Mussa-Ivaldi et al. (1985) regarding voluntary control of stiffness: Coactivation of the modules produced fields that greatly varied in size, but relatively little in terms of shape or orientation.

The framework of postural modules was inspired by the neurophysiological data from the Bizzi laboratory (Bizzi et al., 1991) regarding organization of the premotor regions in the spinal cord of the frog. The basic property of a postural module is its ability to coordinate activity of a set of muscles so that they define an equilibrium position for the limb. Implicitly, this means that both agonist and antagonist muscles need to be recruited to produce a stable equilibrium somewhere in the limb's work space. This ability to produce an equilibrium posture has been illustrated in the spinal frog, whose spinal cord was surgically disconnected from the brainstem (Bizzi et al., 1991): The gray matter of the frog's lumbar spinal cord was stimulated, and isometric forces at the ankle were measured while the frog's leg was held in a variety of postures. It was found that the spatial distribution of the forces induced by the stimulation was structured in a well-defined pattern: The flow of the force vectors converged toward a single equilibrium position. Giszter, Mussa-Ivaldi, and Bizzi (1991) have found four spinal regions from which four distinct convergent fields are elicited (measuring at the ankle when movement is constrained to a plane). Within each region, the stimulation of the premotor layers of the spinal gray matter generates a similar pattern of force vectors. A crucial characteristic of the circuitry of each such module is that the interneurons that constitute a module make synaptic connections with different motor pools, suggesting a synergy of muscles that include both flexors and extensors.

The basic idea of modules is that they form a spanning set of the motor space: Through coactivation, any point in the reachable space can be reached. Pairs of these modules may form an orthogonal set of axes of an abstract coordinate system for control of posture in complex musculoskeletal organisms. There is evidence suggesting that this may be a general characteristic of the intermediate stages in motor control hierarchy: In the barn owl, Masino and Knudsen (1990) have shown that head movements are encoded in terms of an abstract, orthogonal coordinate system whose components are controlled by four functionally distinct neural circuits. In cats and monkeys, electrical stimulation of several brainstem tegmental regions elicits either primarily horizontal or vertical head movements (Fukushima, 1987, Westheimer & Blair, 1975). Psychophysical experiments suggest that human arm movements may also be encoded in terms of their azimuthal and elevational components (Lacquaniti, 1989; Soechting & Flanders, 1989). In the frog, it has been shown (Masino & Grobstein, 1989) that a transformation from a retinocentric to a "parcellated" form of spatial representation occurs: Visually triggered orienting behavior of frogs following lesions in the caudal



medulla was absent for all stimulus locations in the ipsilateral visual hemifield, whereas responses to stimuli in the contralateral visual field were normal.

In summary, this work has linked limitations on voluntary control of stiffness in the human arm to a framework in which postural modules serve as an intermediate control system through which relatively high-level commands regarding desired position and stiffness are translated into the muscle commands needed to execute a postural task.

### ACKNOWLEDGMENT

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### NOTE

1. Recall that stiffness is a measure of only the gradient of the torque field at near-equilibrium. Once muscle parameters are assigned, the torque field will in general vary nonlinearly as a function of joint or end-effector position (because muscle force is a nonlinear function of muscle length). The torque and force fields in this article are the actual fields produced by the nonlinear muscle model and not a linear approximation derived from the stiffness relationship.

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