Adaptive Control of Mechanical Impedance by Coactivation of Antagonist Muscles

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Abstract—**This paper examines the postulate that an important function of the activity of antagonist muscle groups is to modulate mechanical impedance. Some biomechanical modeling and analyses are presented leading to a prediction of simultaneous activation of antagonist muscles in the maintenance of upright posture of the forearm and hand. An experimental observation of antagonist coactivation in this situation is presented.**

NOMENCLATURE

b	Effective linear viscosity of a muscle
g	Gravitational constant
k	Constant
l	Distance from elbow axis to mass center of forearm
m	Mass
u	Control input
w	Purely random Gaussian process
B	Angular viscosity
C	Criterion function
$E\{\ \}$	Expectation operator; also denoted by overbar
F	Muscle force
I	Inertia
K	Angular stiffness
$\lvert\overline{M}\rvert$	Mean rectified surface myoelectric activity
P	Power
Q	Position error weighting coefficient
R	Risk function
S	Strength of purely random process
T	Torque
V	Velocity of shortening of muscle
$\delta(\tau)$	Impulse function
η	Efficiency
λ	Pooled firing rate, adjoint variable
θ	Elbow angle
σ_M	Standard deviation of surface myoelectric activity
ω	Angular velocity

Subscripts

b	Agonist
t	Antagonist
n	Net
d	Differential
o	Isometric
c	Chemical
m	Mechanical
max	Maximum

Superscripts

o	Optimal
n	Nominal
p	Perturbation

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INTRODUCTION

SKELETAL muscle is the actuator which drives natural limb movements. How it is operated by the central nervous system (CNS) to produce movement continues to be one of the fundamental questions of neurophysiological research. Over the past decade it has become clear that in pursuing this question it is not adequate to regard muscle as simply a generator of force; the mechanical impedance of muscle—the static and dynamic relation between muscle force and imposed stretch—has been shown to play an important role in the control of posture and movement [5]–[7], [18], [25], [33], [40], [47], [50].

Deafferented monkeys are capable of controlling horizontal planar movements of the forearm and hand to a visually presented target [7], [47], [54]. They can maintain posture at the target position in the presence of disturbances even in the complete absence of information about the position of the limb. Postural stability in the absence of feedback can only be achieved if, under static conditions, the muscle force changes with length in a manner similar to that of a spring. A case for the importance of the spring-like properties of the muscles was originally made by Feldman [12], [13].

When the neural pathways are intact, the response of the neuromuscular system to stretch is also spring-like [38], [39] and it has been proposed that a major role of proprioceptive reflexes may be the maintenance of muscle stiffness [43], [44]. It has been demonstrated that the action of the stretch reflex effectively compensates for the severe asymmetries and nonlinearities of areflexic electrically stimulated cat soleus muscle [10], [23], [24], [30], [43], [44].

Given the importance of the mechanical impedance of muscle, does the central nervous system modulate or control it? Manipulation of an object requires mechanical interaction with it. The mechanical impedance of the neuromuscular system determines the reaction forces on the hand in response to perturbations from the manipulated object and choosing the mechanical impedance may be one of the ways the CNS controls the behavior of the complete system, hand plus object. Studies of the response of the intact human elbow to perturbation have shown that under experimental conditions the CNS is capable of varying the total stiffness and viscosity about a joint over a considerable range [34], [57]. One goal of the work reported in this paper is to

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demonstrate the adaptive control of mechanical impedance in humans by examining a common physiological situation in which modulation of at least the neuromuscular stiffness is necessary.

A second goal of the work reported in this paper is to examine the postulate that the CNS controls impedance through the simultaneous activation of opposing muscles [25], [28], [29]. Coactivation of antagonist muscles is frequently observed under normal physiological conditions [4], [9], [37]. Because their actions oppose one another, simultaneous activation of antagonists does not contribute to the useful work output of the muscles, yet it costs input metabolic energy. Under the assumption that metabolic energy is not squandered without profit, the purpose of antagonist coactivation needs to be explained. When two or more muscles are arranged antagonistically about a joint, the torques due to the opposing muscles subtract from one another, but in contrast, the impedances due to the opposing muscles add. The net torque about a joint is predominantly determined by the difference between the activities of the agonist and antagonist muscle groups, while the net angular stiffness and viscosity about the joint is predominantly determined by the sum of their activities. Thus, within limits, the net torque and the net angular impedance about the joint can be controlled independently. In this paper a mathematical analysis is presented and a theoretical prediction of antagonist coactivation is obtained. The basic postulate underlying the analysis is that antagonist coactivation is one of the means at the disposal of the CNS for adaptively tuning the parameters of the controlled system. An experimental observation is presented in support of this postulate.

Mechanical Impedance of Muscle

The relation between the neural input to a muscle and its subsequent mechanical behavior is extremely complicated. For a given neural input the contractile force of a muscle depends on the length of the muscle, its velocity of shortening, the type of muscle, its state of fatigue, its history of exercise (or of electrical stimulation) and more. However, one fundamental observation is that the neural input to a muscle simultaneously determines the contractile force and the stiffness of the muscle (i.e., its resistance to stretch). In the case of the force increment resulting from rapid, small-amplitude stretch or release of electrically stimulated areflexic muscle, the incremental stiffness has been shown to be linearly related to the mean contractile force of the muscle [15], [16], [36], [42], [49]. This incremental or short-range stiffness is attributed to the molecular mechanism underlying muscle contraction.

The net effect of the musculature on the limbs also depends on the action of neural reflex feedback. A major consequence of the negative position feedback provided by the stretch reflex is the maintenance of muscle stiffness. In the absence of reflexes, a decerebrated cat soleus muscle stretched beyond a fraction of a millimeter exhibits pronounced nonlinear behavior, most notably a yielding or drop in muscle force [14], [15]. With reflexes present, the muscle performance is much closer to linear; the yielding is no longer observed [10], [19], [23], [30], [38], [39], [43], [44]. For the purposes of this paper, it is important to note that the stiffness of the decerebrated cat soleus increases monotonically with operating force throughout the lower half of the physiological range of muscle force [23], [24].

Under normal physiological conditions, evidence of an increase in muscle stiffness with muscle force may be seen in the static relation between isometric force and length or between joint torque and joint angle. For example, Vrendenbregt and Rau [55] investigated the relation between myoelectric activity of biceps, static isometric muscle-generated elbow torque, and elbow angle in normal human subjects. They found that the torque corresponding to maximum voluntary contraction was a function of angle. In addition, they found that the form of the relation between torque and myoelectric activity was independent of elbow angle if the torque was scaled by its maximum value at a given angle. This relation between torque, angle, and myoelectric activity can be written as:[1]

$$T/T_{\max}(\theta) = g(|\bar{M}|). \tag{1}$$

$T_{\max}(\theta)$ is the angle-dependent maximum torque and $g(|\bar{M}|)$ is a static function of the mean rectified value of myoelectric activity. The joint stiffness is the first partial derivative of torque with respect to angle

$$\partial T/\partial\theta = g(|\bar{M}|)\cdot \partial T_{\max}(\theta)/\partial\theta \tag{2}$$

$$= g(|\bar{M}|)\cdot T'_{\max}(\theta) \tag{3}$$

$$= \frac{T}{T_{\max}(\theta)}\cdot T'_{\max}(\theta) \tag{4}$$

$$\therefore \quad \partial T/\partial\theta = T\left[\frac{T'_{\max}(\theta)}{T_{\max}(\theta)}\right]. \tag{5}$$

The multiplicative structure of (5) implies that at any given angle, torque is linearly related to angular stiffness (or force to linear stiffness). In summary, the net static behavior of the neuromuscular system is similar to that of a variable-stiffness spring.

Just as the static relation between muscle force and length implies spring-like behavior, the static relation between muscle force and rate of shortening implies a net viscous behavior, and in general the total mechanical impedance of the muscle may be a function of neural input. Changes in the total mechanical impedance of the intact human elbow in response to small perturbations have been reported by Lanman [34] and by Zahalak *et al.* [57].

Why Modulate Impedance?

Changing the mechanical impedance of the neuromuscular system is a form of parameter-adaptive control which the CNS may use to accommodate its behavior to environmental conditions. Adaptation to the environment is probably one of the most fundamental aspects of primate motor behavior, but parameter tuning is only one of many possible forms of adaptive control. One simple but significant situation in which parameter adaptation may be distinguishable from the other possible behavioral strategies is the maintenance of the postural stability of the musculoskeletal system. The greater part of the human skeleton behaves like a series of inverted pendula stacked one on top of another. Because of this, the skeleton is statically unstable in the absence of torsional stiffness about the joints and when an object is carried, the gravitational destabilizing effect increases. Some torsional stiffness is provided by the ligaments, but this effect is small compared to the gravitational loads—if one relaxes completely one falls over. The required torsional stiffness may be provided by negative position feedback or by antagonist coactivation, or a combination of both. The main difference between the two mechanisms lies in their limitations. Feedback control is limited by transmission delays around neural feedback loops and by the limited bandwidth of open-loop muscle and sensor characteristics. As a result, the maximum feedback gain which may be used to stabilize the system is restricted and the maximum achievable stiffness of the closed-loop system is limited. In contrast, stabilization by antagonist coactivation is unaffected by neural transmission delays. However, its major limitation is that it incurs an energy cost as the opposing muscles are doing no mechanical work but are consuming metabolic energy.[2] The

[1] A list of mathematical symbols is provided in the Nomenclature.

[2] By comparison, the energy cost of feedback compensation would be small, particularly if the muscles were reciprocally activated.

central nervous system would have to compromise between postural stabilization and metabolic energy consumption. In this paper, dynamic optimization theory will be used to analyze the maintenance of upright posture of the forearm and hand and predict the modulation of impedance via antagonist coactivation.

Mathematical Modeling

In order to focus attention on a mode of control available to the CNS which has hitherto received scant attention, the mathematical modeling will assume that modulation of joint stiffness is accomplished exclusively through coactivation of antagonist muscle groups.

A simple model which characterizes the variable-stiffness behavior of muscle is shown in Fig. 1(a). At a given length, this model yields the linear relation between stiffness and force which is observed in experimental animals and intact humans. At a given level of activation, the true relation between isometric force and length is probably nonlinear [48] [see Fig. 1(b)]. However, the simple linear model of Fig. 1(a) captures the essential behavior—force increases with length—and it will be used in the interest of simplicity.

The group of muscles acting about the elbow will be modeled by two opposing spring-like muscles [see Fig. 1(c)]. The forearm and hand will be modeled as a rigid link of inertia I and mass m rotating about a fixed axis. The angle-dependent variations in the moment arms at which the muscle forces act about the joint will be ignored, an assumption valid for small changes in joint angle. The maximum flexive and extensive torques which can be generated by the muscles will be assumed equal. This assumption of symmetry simplifies the analysis and does not qualitatively affect its outcome.

In modeling the dynamic behavior of muscle, the dynamics of the excitation/contraction coupling will be ignored. The principal results of the analysis will be obtained for steady-state conditions, e.g., fixed levels of excitation, under which this assumption is justifiable. However, even at fixed excitation, muscle force depends on the velocity of contraction [31], [32], [56] [see Fig. 2(a)]. The velocity dependence will be modeled as a linear viscous element, an assumption valid for small changes in velocity. The variation of the viscous parameter b (i.e., slope of the force/velocity curve), with level of excitation will be neglected. The resulting assumed relation between net muscle torque and angular velocity of the joint is shown in Fig. 2(b). The consequences of this assumption are examined further in the discussion section.

Summarizing, the isometric muscle torques will be modeled by:

$$T_b = (T - K\theta)u_b \tag{6}$$

$$T_t = -(T + K\theta)u_t. \tag{7}$$

Subscripts b and t refer to agonist (e.g., biceps) and antagonist (e.g., triceps), respectively. The neural control is represented by u. Its relation to alphamotoneuron firing rate is described later. It is assumed to be a dimensionless number with a range from 0 to 1.

$$0 \leqslant u_b \leqslant 1; \qquad 0 \leqslant u_t \leqslant 1. \tag{8}$$

Joint angle θ is defined as zero in the vertically upright position, positive towards flexion (see Fig. 1) with a range of $\pi/2$ on either side. To represent the fact that muscles cannot push, the following inequalities will be imposed:

$$T_b \geqslant 0 \quad \text{and} \quad T_t \leqslant 0 \qquad \text{for } -\pi/2 \leqslant \theta \leqslant \pi/2. \tag{9}$$

Consequently, the bounds on the assumed value of the angular stiffness K are

$$0 \leqslant K \leqslant 2T/\pi \tag{10}$$

where T is the maximum isometric muscle torque which can be generated with the forearm in the middle position.

The net isometric muscle torque T_n is the sum of the antagonist muscle torques

$$T_n = T(u_b - u_t) - K(u_b + u_t)\theta. \tag{11}$$

Thus, at any given angle the torque about the joint and the stiffness about the joint can be controlled independently via the sum and difference of the input activities, respectively [Fig. 1(d)].

As the limb moves in a vertical plane, the gravitational torque about the joint is given by

$$T_g = mgl \sin\theta \tag{12}$$

where g is the acceleration due to gravity and l the distance from the center of gravity to the axis of rotation.

Including the assumed viscous effects of the muscles and the inertial effects of the limb, the dynamic model equations are as follows:

$$\dot{\theta} = \omega \tag{13}$$

$$I\dot{\omega} = T(u_b - u_t) - K(u_b + u_t)\theta + mgl \sin\theta - B\omega \tag{14}$$

where B is the viscous coefficient and ω is the angular velocity of the limb. Equations (13) and (14) and inequalities (8)–(10) represent the dominant mechanical behavior of the limb in response to neural inputs.

To obtain a prediction of antagonist coactivation, dynamic optimization theory will be used to minimize a criterion function representing the task of maintaining upright posture. To model the tradeoff between energy consumption and postural stabilization, the criterion function to be minimized will be the time integral of the instantaneous power consumed by the muscle plus the square of deviation from the desired posture. An expression for the metabolic energy consumption of muscle is required. For simplicity, it will be assumed that to a reasonable approximation the *input* metabolic power or energy rate is independent of the output mechanical states of the muscle and depends only on the neural input. It will be assumed that the *output* mechanical power depends upon the muscle state variables in a manner which is adequately characterized by the force–velocity relation for the muscle. To provide a qualitative check on adequacy of these assumptions, the relation between muscle efficiency η and relative muscle force can be computed for a given level of neural excitation as follows: by assumption, the force/velocity relation for a single muscle is

$$F = F_o - bV. \tag{15}$$

F_o is the isometric muscle force. V is the velocity of shortening. Rearranging

$$V = \frac{F_o}{b}\left(1 - \frac{F}{F_o}\right). \tag{16}$$

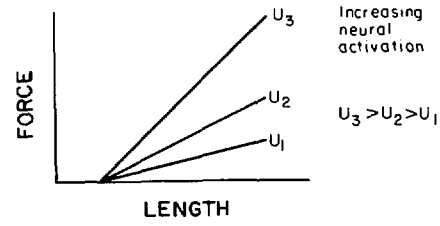
Mechanical output power P_m is the product of force and velocity

$$P_m = \frac{F_o^2}{b}\left(1 - \frac{F}{F_o}\right)\frac{F}{F_o}. \tag{17}$$

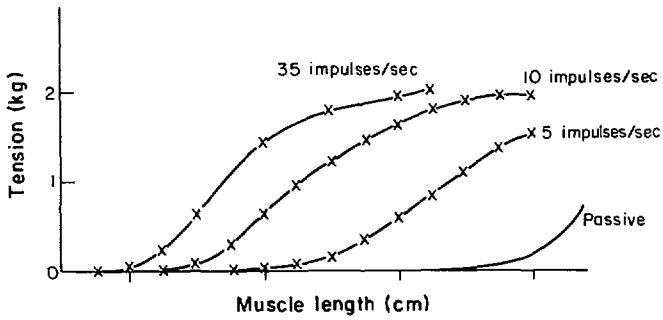
By assumption, at fixed excitation the input chemical power consumption is constant at P_c and the efficiency η is given by

$$\eta = \frac{P_m}{P_c} = \frac{F_o^2}{P_c b}\left(1 - \frac{F}{F_o}\right)\frac{F}{F_o}. \tag{18}$$

Fig. 3 shows a plot of efficiency versus relative isometric muscle force. Data from Hill [21], [22] for relative muscle forces



(a)



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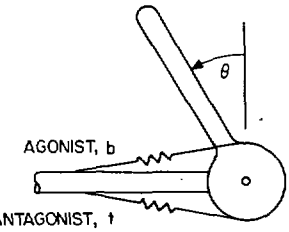

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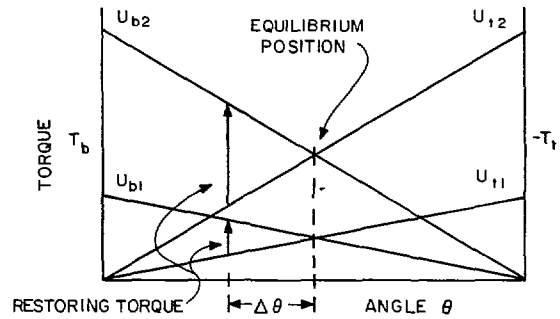
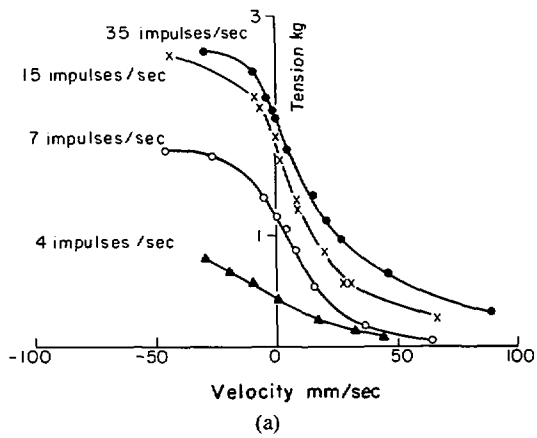


(d)

Fig. 1. (a) A simple model which characterizes the variable-stiffness behavior of muscle. This model captures the essential static behavior but ignores known nonlinear behavior, such as that shown in (b) obtained from electrically stimulated areflexic cat soleus muscle under isometric conditions (data redrawn from [32]). (c) The entire group of muscles acting about the elbow are modeled by two opposing muscles with characteristics as in (a) acting at fixed moment arms about the joint axis. (d) If the opposing muscles are active simultaneously, the net torque about the joint and the stiffness about the joint may be controlled independently via the sum and difference of the muscle activities, respectively. If external torques on the joint are zero, antagonist coactivation defines an equilibrium position for the joint.



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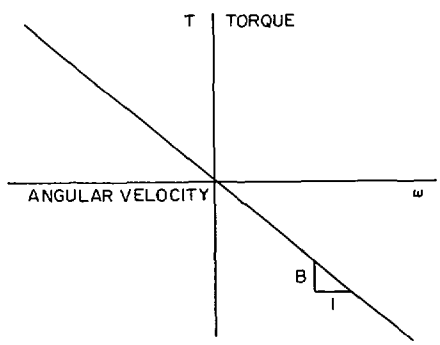

(b)

Fig. 2. The force generated by a muscle depends on the velocity of contraction. (a) Data obtained from electrically stimulated areflexic cat soleus muscle under isotonic conditions (data redrawn from [31]). To model the change in net muscle torque as angular viscosity varies about zero, the variation in the slope of the force velocity curves is neglected and the relation assumed to be linear as shown in (b).

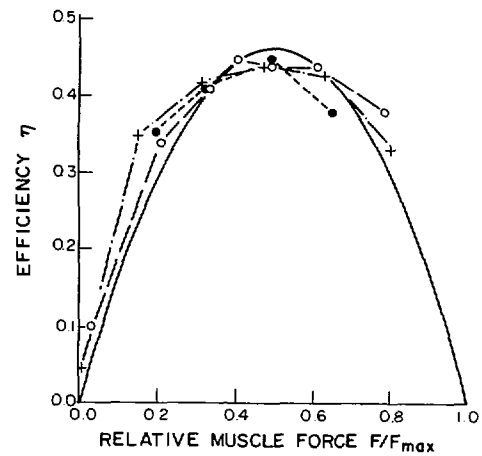

Fig. 3. The modeled relation between the efficiency η of a muscle and the ratio of muscle force to its maximum value is shown by the continuous line. Peak efficiency is $F_0^2/4bP_c$. Experimental data (redrawn from [22]) is also shown. The ratio of model parameters is chosen to fit the experimental data. Up to 80 percent of maximum contraction of the agreement is adequate.

ranging from zero to 80 percent of maximum contraction are also shown. Throughout this range, the agreement between the data and the shape of the curve derived from the simple model of (18) is adequate, indicating that the assumptions are consistent with the dominant thermodynamic behavior of the muscle.

For the analysis, a relation between the neural excitation into a muscle and the energy consumption of the muscle is required. It will be modeled as follows: the total muscle activity is the sum of individual motor unit activities. Incoming nerve impulses are distributed across space (different motor units) and time. The pooled firing rate λ of the motor nerve will be defined as the sum of all nerve impulses arriving at the muscle per unit time. It is a single parameter which may be used to summarize the overall neural excitation.

The total metabolic energy consumed in a muscle per unit time

is the simple linear sum of the energy consumed in each motor unit. For simplicity, it will be assumed that the energetic cost of firing a motor unit is the same for all motor units.[3] Consequently, the total metabolic energy consumed by a muscle per unit time will be proportional to the pooled firing rate

$$P_c = k_1 \lambda. \tag{19}$$

A relation between neural excitation and the mechanical output of a muscle is also required for the analysis. This is deduced from the relations between the electrical activity of muscle and its mechanical output and neural input, respectively, as follows: the incoming nerve impulses result in the firing of individual motor units and the depolarizations of the sarcolemma membranes of the muscle fibers sum to form the bulk of the gross electrical activity of the muscle [45]. Theoretical considerations have shown that the total variance or power of the myoelectric signal is directly proportional to the pooled firing rate

$$\sigma_M^2 = k_2 \lambda. \tag{20}$$

The amplitude distribution of surface myoelectric activity is well described as Gaussian with zero mean [51]. As a result, the standard deviation of surface myoelectric activity is proportional to its mean rectified value

$$\sigma_M = k_3 |\overline{M}|. \tag{21}$$

The total isometric contraction force of the muscle is a combination of the individual tension twitches of the active motor unit, but unlike energy, forces do not superimpose linearly [46], [53]. The relation between isometric muscle force and mean rectified surface myoelectric activity of biceps brachii has been investigated extensively [35], [41], [55]. Up to at least 30 percent of maximum voluntary contraction, the relation is linear (see Fig. 4).

$$|\overline{M}| = k_4 \frac{F}{F_o}. \tag{22}$$

Combining (20)–(22) and rearranging yields

$$\frac{F}{F_o} = \frac{\sqrt{k_2}}{k_3 k_4} \sqrt{\lambda}. \tag{23}$$

That is, relative muscle force is proportional to the square root of pooled firing rate. The most commonly quoted data in the literature on muscle force versus neural firing rate is that of Joyce, Rack, and Westbury [32]. Their data show a sigmoidal form which is not modeled by (23). However, their data were obtained from electrically stimulated muscle and the authors point out that the sigmoidal form may be an artifact of their experimental procedure. In contrast, the data of Fig. 4 upon which (23) is based were obtained from intact humans under physiological conditions and exhibit no sigmoidal form.

At this point, for notational convenience, the neural control input u is defined as the square root of the pooled firing rate scaled by its maximum value

$$u \triangleq \sqrt{\frac{\lambda}{\lambda_{\max}}}. \tag{24}$$

Equations (19) and (23) may now be rewritten as

$$P_c = P \cdot u^2 \tag{25}$$

$$F = F_o \cdot u. \tag{26}$$

[3]In fact, motor units recruited at higher contraction levels have higher energetic cost. This will make the relation between input metabolic power and pooled firing rate more than linear. The existence of a solution to optimization problem is not affected.

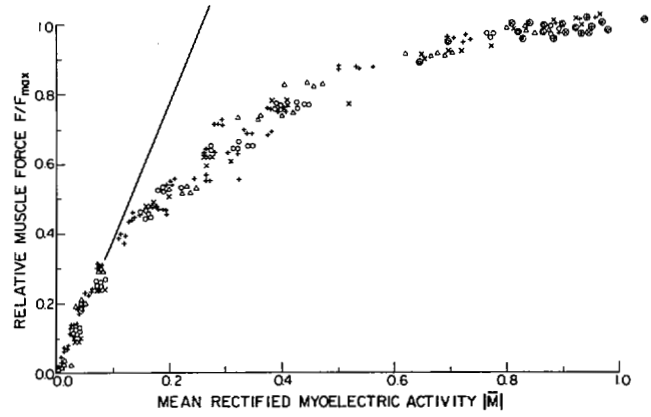

Fig. 4. The modeled relation between isometric muscle force and mean rectified surface myoelectric activity is shown by the straight line. Up to at least 30 percent of maximum voluntary contraction the experimentally observed relation is also linear (data redrawn from [55]).

P is a scaling constant and represents the metabolic power consumption of the muscle at maximum excitation. F_o is the isometric force at maximum excitation. According to these equations, the relation between input metabolic power and relative isometric muscle force is nonlinear. On the basis of much more detailed considerations, Hatze [20] also arrived at a nonlinear relation.

Note that the dependence of isometric force on muscle length is embodied in F_o. Multiplying (26) by the moment arm about the axis of the joint and representing the length-dependence explicitly as a dependence of isometric torque on joint angle yields (6) and (7).

Dynamic Optimization

The problem of maintaining upright posture while minimizing energy consumption is modeled as the problem of finding the neural control inputs u_b and u_t which minimize the criterion function C

$$C = \int_0^t \left(P u_b^2 + P u_t^2 + Q\theta^2 \right) dt \tag{27}$$

where Q is a coefficient penalizing deviation from upright posture ($\theta = 0$), subject to the constraints on the control inputs [inequality (8)] and the constraining dynamic equations (13), (14). Analysis yields the following conditions:

$$u_b = \frac{-\lambda_2 (T - K\theta)}{2PI} \qquad \text{if } 0 \leqslant u_b \leqslant 1 \tag{28}$$

$$u_t = \frac{\lambda_2 (T + K\theta)}{2PI} \qquad \text{if } 0 \leqslant u_t \leqslant 1. \tag{29}$$

The adjoint variable λ_2 determines the optimum values of u_b and u_t. Using inequality (9) yields the following:

$$\left.\begin{aligned} T - K\theta \geqslant 0 \\ T + K\theta \geqslant 0 \end{aligned}\right\} \qquad \text{if } -\pi/2 \leqslant \theta \leqslant \pi/2. \tag{30}$$

As a result, the optimum solution (by this analysis) is reciprocal activation of the antagonist muscles:

$$\lambda_2 > 0 \qquad u_b = 0 \qquad u_t = \lambda_2 (T + K\theta)/2PI \tag{31}$$

$$\lambda_2 = 0 \qquad u_b = 0 \qquad u_t = 0 \tag{32}$$

$$\lambda_2 < 0 \qquad u_t = 0 \qquad u_b = -\lambda_2 (T - K\theta)/2PI. \tag{33}$$

The steady-state optimum solution may be deduced by noting that at equilibrium in the upright position, the net torque about

the joint is zero and the only admissible solution is

$$u_b = u_t = 0. \tag{34}$$

According to this analysis the optimum solution for minimum energy maintenance of upright posture is complete relaxation. However, with the muscles relaxed, the limb is unstable.

This result is obtained because the open-loop dynamic optimization techniques used in the analysis do not take account of stability [8]. (It will be recalled that feedback was deliberately omitted from the modeling and analysis to focus attention on antagonist coactivation as a means of postural stabilization.)

To ensure that the analysis takes account of the instability of the system, an infinitesimal unpredictable perturbation $w(t)$ is added to the dynamic model. For convenience, $w(t)$ is assumed to be a zero mean, Gaussian, purely random process of strength S.

$$E[w(t)] = 0 \tag{35}$$

$$E[w(t)w(t+\tau)] = S\delta(\tau). \tag{36}$$

Once the solution to the resulting stochastic optimization problem is obtained, the limit as S approaches zero will be taken. The model equations are now:

$$\dot{\theta} = \omega \tag{37}$$

$$I\dot{\omega} = T(u_b - u_t) - K(u_b + u_t)\theta + mgl\sin\theta - B\omega + w. \tag{38}$$

Because of the influence of the random perturbation, the criterion function is now a random variable. The optimum control is found by minimizing the expected cost per unit time

$$R = E\left[\frac{1}{t}\int_0^t \left[Pu_b^2 + Pu_t^2 + Q\theta^2\right]\right] dt \tag{39}$$

$$R = \frac{1}{t}\int_0^t \left[Pu_b^2 + Pu_t^2 + Q\overline{\theta^2}\right] dt. \tag{40}$$

The dynamic equations constraining this minimization problems are the nonlinear, time-varying, stochastic differential equations for the evolution of the mean squared deviation from upright posture $\overline{\theta^2}$. An approximate solution to this type of problem may be obtained by first solving the nonlinear deterministic problem of minimizing the criterion function (27) subject to the deterministic constraining dynamic equations. The resulting control and state trajectories are referred to as the nominal control $\boldsymbol{u}^n$ and nominal state $\boldsymbol{\theta}^n$, respectively. The steady-state solution to this problem was obtained above and is

$$\boldsymbol{u}^n = \begin{bmatrix} u_b^n \\ u_t^n \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \end{bmatrix}; \qquad \boldsymbol{\theta}^n = \begin{bmatrix} \theta^n \\ \omega^n \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \end{bmatrix}. \tag{41}$$

The nonlinear stochastic system equations (37) and (38) are then linearized about the nominal state trajectory and a set of linearized perturbation covariance equations are derived. The risk function (40) is minimized subject to the linearized covariance equations to obtain a perturbation control $\boldsymbol{u}^p$, which will keep the system close to the nominal state trajectory. The approximate optimal control $\boldsymbol{u}^o$ is obtained by adding the nominal (deterministic) control and the (linearized) perturbation control

$$\boldsymbol{u}^o = \boldsymbol{u}^n + \boldsymbol{u}^p. \tag{42}$$

This approach is similar to the "perturbation control" method [3].

Assuming a steady-state solution exists, it is obtained by setting all rates of change to zero, which results in the following set of conditions defining the perturbation control:

$$u_b^p = \frac{\lambda_2 K\overline{\theta^2}}{2PI} = u^p \tag{43}$$

$$u_t^p = \frac{\lambda_2 K\overline{\theta^2}}{2PI} = u^p. \tag{44}$$

The adjoint variable λ_2 is defined by

$$\lambda_2 = \frac{QI}{K(u_b^p + u_t^p) - mgl}. \tag{45}$$

As the nominal control $\boldsymbol{u}^n$ was zero, the perturbation control is equal to the optimal control. The optimal control is to activate the antagonists equally by an amount given by the following cubic in u^o:

$$\frac{u^o}{K}[2Ku^o - mgl]^2 = \frac{QS}{4PB}. \tag{46}$$

The elements of the covariance matrix are given by the following equations:

$$\overline{\theta^2} = \frac{S}{2B[2Ku^o - mgl]} \tag{47}$$

$$\overline{\theta\omega} = 0 \tag{48}$$

$$\overline{\omega^2} = \frac{S}{2BI}. \tag{49}$$

Note that the mean square velocity is unaffected by the antagonist coactivation. This is because the model assumed that only the stiffness was modulated by coactivation, i.e., that the viscosity was constant.

Equation (47) can be rearranged to express the tradeoff between optimal coactivation and the mean squared error

$$u^o = \frac{mgl}{2K} + \frac{S}{4BK\overline{\theta^2}}. \tag{50}$$

Thus, as the mean squared error is allowed to become large, or the strength of the random perturbation becomes small, the optimum level of muscle activity decreases to a limiting value of $mgl/2K$. This is the minimum value for which the limb is stable in the upright position.

The perturbing noise process was included solely to ensure that stability was factored into the analysis. Taking the limit as the strength S of the perturbation goes to zero yields the following:

$$\lim_{S\to 0} u^o = \frac{mgl}{2K} \tag{51}$$

$$\lim_{S\to 0} \overline{\theta^2} = 0 \tag{52}$$

$$\lim_{S\to 0} \overline{\omega^2} = 0. \tag{53}$$

In the limit, the power consumption of the muscles is given by

$$P_c = \frac{P(mgl)^2}{2K^2}. \tag{54}$$

As expected, modulation of stiffness by antagonist coactivation places a continuous power drain on the muscles.

Experimental Observation

The neuromuscular system must participate actively in the maintenance of upright posture of the forearm as passive tissue effects are insufficient. When the destabilizing effects of gravity are increased by carrying an object, the total joint stiffness must increase to preserve postural stability. If stiffness is controlled

solely by coactivation of antagonists the above analysis predicts a constant, nonzero level of muscle activity which increases with the magnitude *mgl* of the gravitational term. Consequently, one simple test of the postulated control of impedance through antagonist coactivation is to observe antagonist muscle activity during maintenance of upright posture of the forearm as gravitational loads are increased. If there is no significant increase in antagonist muscle activity, then the necessary increase in stiffness cannot be due to antagonist coactivation and must be accomplished solely through feedback control. A significant increase in antagonist activity would be evidence in support of impedance control through antagonist coactivation, although a contribution from the feedback loops could not be ruled out.

To test this prediction a simple experiment was performed. Surface myoelectric activity of forearm flexor and extensor muscles (biceps and triceps) was recorded from two human subjects while they maintained a series of postures of the upper extremity. The myoelectric activity was obtained using pairs of dry stainless-steel disk electrodes with the first stage differential preamplifier (Motion Control, Inc., UT) mounted directly on the electrodes. The preamplifier has a passband of 5 Hz to 1.7 kHz with a midrange gain of 300. The common-mode rejection ratio is typically 100 dB up to 1 kHz. The performance of this instrumentation is discussed in detail by Hogan and Mann [26], [27]. For present purposes it is important to note that in that paper the cross-correlation between the output of different electrodes pairs was shown to decrease rapidly with the separation of the pairs and was below 0.5 at a separation of 3 cm. In the experiment reported here a single pair of electrodes was placed on biceps and a pair was placed on triceps. The two pairs were on opposite sides of the upper arm and as a result the cross correlation between their activities was close to zero.

Myoelectric activity was recorded while the upper arm was maintained in a series of stable postures. In the first series the upper arm hung vertically downwards and the forearm was held in the saggittal plane at angles of approximately 0°, 45°, 90°, and 135° with respect to the vertically downward direction [see Fig. 5(a)]. In the second series the upper arm was rested comfortably on a stable support so that it was in a horizontal position in the saggittal plane. The forearm was held upright in the saggittal plane at angles of approximately 0°, 45°, 90°, and 135° with respect to the horizontal [see Fig. 5(b)]. In all cases the wrist was held in the supine position. The subject was instructed to relax while maintaining posture.

In a third series of observations the postures of the first series were repeated, but this time the subject held a 5 lb weight in the hand [see Fig. 5(c)]. The wrist was again supine. A fourth series of observations was obtained while the subject held the 5 lb weight while maintaining the postures of the second series [see Fig. 5(d)].

Representative results for each of the 16 cases are shown in Fig. 5. 1.5 s segments of raw (unprocessed) surface myoelectric activity of biceps and triceps are shown corresponding to each of the 16 cases described above. The extensive gravitational torque in position 6 is approximately equal to the flexive gravitational torque in position 8. To facilitate comparison of biceps and triceps activity, the gain of the recording instrumentation was adjusted so that the magnitude of biceps myoelectric activity in position 6 was approximately equal to the magnitude of triceps myoelectric activity in position 8.

In positions 1 and 5 in both the loaded and unloaded cases the muscles were relaxed. Consequently, the myoelectric activity of the muscles is effectively zero (see Fig. 5). In position 3 in the unloaded case [see Fig. 5(a)] biceps is seen to be active. This level of myoelectric activity is required to maintain the horizontal posture of the forearm against gravity.

Position 7 corresponds to the posture for which the analytical predictions were derived. In the loaded case simultaneous myoelectric activity of approximately equal magnitude can be seen in

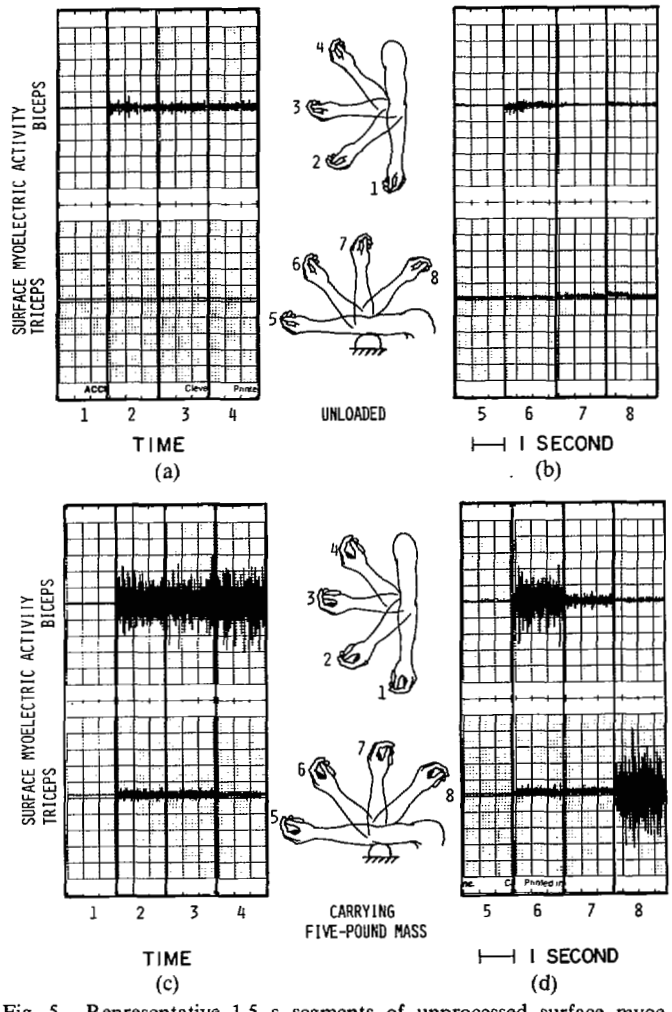

Fig. 5. Representative 1.5 s segments of unprocessed surface myoelectric activity of biceps and triceps recorded from a normal human subject maintaining a series of stable postures. (a) The upper arm hung vertically downward, the forearm was held in the saggital plane in the positions shown. (b) The upper arm was rested comfortably on a stable support so that it was in a horizontal position in the saggital plane, the forearm was held upright in the positions shown. (c) The postures of (a) were repeated while the subject held a 5 lb weight. (d) The postures of (b) were repeated while the subject held a 5 lb weight. The wrist was supine in all cases. Simultaneous activity of the antagonist muscles is clearly evident.

both biceps and triceps [Fig. 5(d)]. The strength of this contraction can be estimated by noting that the magnitude of the myoelectric activity of biceps in position 7 in the loaded case is comparable to the magnitude of its activity in position 3 in the unloaded case. That is, to maintain upright posture while carrying a 5 lb weight, biceps generates a net flexive torque comparable to that required to hold the unloaded forearm in a horizontal position against gravity.

Although no analytical predictions were made for the other positions, it is interesting to note that in the loaded condition, simultaneous activity of agonist muscles is seen at all positions in which posture cannot be maintained by relaxing completely. In position 3 the gravitational torque is at its maximum, yet the antagonist is active at a level approximately comparable to the level of agonist activity required to hold the unloaded forearm in the same posture.

Discussion

The simple experiment described above was designed to answer two questions: Are significant levels of simultaneous activation of

agonist and antagonist muscles observed under normal physiological conditions? And does the level of antagonist coactivation increase as gravitational torques increase? The answer to both questions is unequivocally affirmative. The simultaneous activity seen in Fig. 5(d) cannot be attributed to any artifact of the recording procedure such as crosstalk between electrodes. In the first place it has been shown that crosstalk between electrodes decreases very rapidly with separation of electrode pairs [27]. In the second place any postulated crosstalk would have to be a consistently observed phenomenon. If the recorded activity of triceps in position 7 in the loaded case [Fig. 5(d)] were due to crosstalk from the biceps, it should be observed every time biceps is active. It can be seen in positions 2, 3, and 4 in the unloaded case [Fig. 5(a)] that this is not the case, even though the level of biceps activity is comparable. Conversely, if the recorded activity of biceps in position 7, loaded case, were due to crosstalk from the triceps, then when triceps activity increased as it did in position 8, loaded case [Fig. 5(d)], the level of recorded biceps activity should also increase. In fact, in this case biceps activity decreases. In short, the recorded activity represents a real phenomenon; antagonist muscles are observed to be active simultaneously under physiological conditions [4], [9], [34], [37].

The increase in antagonist coactivation is consistent with the hypothesis that it is a vehicle for modulating the impedance of the musculoskeletal system, but it does not exclude the possibility that the observed coactivation may serve some other purpose or arise from some other cause. For example, rapid reciprocal activation of the antagonist muscles cannot be ruled out without further analysis and experimentation. However, any other postulated cause or purpose for antagonist coactivation would have to account for the observed increase with added load. The hypothesis presented in this paper offers a simple explanation for the increased coactivation: the joint stiffness must increase to offset gravitational destabilization.

It might be argued that the observed coactivation is simply a consequence of holding the weight. In the experiment, the weight was held by the subject (rather than attaching it to the wrist, for example) so that experimental conditions would match normal physiological conditions as closely as possible. It is commonly observed that gripping an object or making a fist results in coactivation of muscle groups of the forearm, arm, shoulder, and trunk. This, in fact, strengthens the case for impedance modulation through antagonist coactivation. To grip an object is to establish a mechanical coupling between hand and object. A "firm grip" corresponds to a high mechanical impedance for the hand and, by hypothesis, would require increased antagonist coactivation. As the hand, forearm, and trunk are in series, a high mechanical impedance of the coupling between object and hand would be of little value in providing support for the object if it were not accompanied by a corresponding high impedance between hand and forearm, forearm and arm, arm and shoulder, and so on. The hypothesis that antagonist coactivation is used to modulate impedance is completely consistent with observed patterns of global muscle activity.

The modeling and analysis presented in this paper considered an extreme case as the possible use of afferent feedback for stabilizing limb posture was ignored. To some extent this can be justified by the growing body of experimental results which show that many aspects of motor control previously thought to be due to peripheral feedback modulation of descending motor commands can in fact be observed in the complete absence of peripheral feedback [5], [6], [47], [54]. However, it seems unlikely, to say the least, that the central nervous system would completely ignore available peripheral feedback. For example, the dominant role played by vestibular feedback in the maintenance of upright posture can hardly be questioned. Instead, it seem likely that the central nervous system takes advantage of all available methods of controlling posture and movement, exploiting the strengths of each as the task dictates. Feedback control is energetically efficient, but is necessarily limited by transmission delays around the feedback loop and by the dynamics of the sensors and actuators. On the other hand, stabilization by antagonist coactivation is unaffected by loop transmission delays, but incurs a heavy metabolic energy cost. Neither of the two strategies is superior in all respects but a combination of the two may be superior to either one alone under a wider range of conditions.

The model presented above is a considerable simplification of the true situation. It was carefully chosen to be the simplest model which would exhibit all of the essential mechanical behavior of the limb. The object was to develop analytical techniques and to explore the idea of impedance modulation by antagonist coactivation. The complexity required for a more accurate model would have impeded progress toward this goal. Almost certainly, it would have required numerical solution rather than the closed-form algebraic solution obtained. The algebraic solution obtained provides a gestalt, an overview of the essential behavior which could not be obtained from single numerical solutions.

In the analysis, the Gaussian, purely-random perturbation was included solely for the purpose of considering stability in the open-loop case (modulation of impedance by coactivation) and consequently its strength was taken to zero in the limit. It is possible that in the real physiological situation a perturbation exists due, for example, to the nature of muscle contraction. However, the steady-state mean-square position error was not measured directly, but it is typically close to zero—human subjects have no difficulty maintaining upright posture of the forearm and hand. As a result the analytical step of taking the limit as the strength of the perturbation goes to zero is a reasonable approximation to the physiological situation.

The mathematical model of muscle behavior used in this paper neglected the variability of the effective viscosity of mucle. As a result, the predicted steady-state mean-square velocity error was not affected by coactivation of antagonist muscles. In reality the effective viscosity about the joint would be modulated by antagonist coactivation along with the stiffness [34], [57]. Increasing the viscosity would reduce both the mean-square velocity error and the mean-square position error [see (47), (49)] and for a perturbation with a nonzero strength the optimum level of coactivation would be reduced. However, as the strength of the perturbation approaches zero, the limiting value of the required antagonist coactivation is unchanged.

The analytical technique used in this paper of separating the optimization problem into a nonlinear deterministic part and a linearized stochastic part is an approximation which closely resembles the highly successful perturbation control approach. As Athans [3] has pointed out, minimizing a quadratic criterion function acts to ensure the accuracy of the linear approximation. In this paper, the nonlinearities encountered were rather simple, but a more general detailed model of muscle would almost certainly include significant nonlinearities. The analytical technique used in this paper may be applied without modification to more general problems.

In this paper, postural stabilization was chosen as an example of one situation in which the need to modulate musculoskeletal impedance is clear. The analysis, simple as it is, yielded a prediction of antagonist coactivation which is consistent with experimental observation and indicates that antagonist coactivation may be an important means of modulating mechanical impedance. Contribution to postural stabilization is only one of the possible functions of an ability to modulate mechanical impedance [25]. Another important function is the control of the mechanical and dynamic coupling between the hand and a held object such as a tool [11], [52]. In some cases tight dynamic coupling is called for: better "grip" means higher mechanical

impedance. In other cases low mechanical impedance may be required to prevent undesirable transmission of shock and vibration to the rest of the musculoskeletal system. Any movement against an external kinematic constraint is simplified by the choice of an appropriate value of the mechanical impedance of the limb. The concept has application to locomotion as well as manipulation [1], [17]. The lower limbs can be regarded as a combination of a propulsion system and an adaptive, tunable suspension system whose properties can be adjusted to match environmental conditions [18], [40]. However, the importance of these adaptive capabilities remains a task for further investigation. As adaption implies the modulation of behavior to meet some criterion of performance, the optimization techniques presented in this paper may prove useful.

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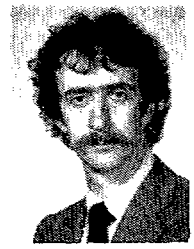

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Guaranteed Robustness Properties of Multivariable Nonlinear Stochastic Optimal Regulators

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Abstract—**We study the robustness of optimal regulators for nonlinear, deterministic and stochastic multiinput dynamical systems, under the assumption that all state variables can be measured. We show that, under mild assumptions, such nonlinear regulators have a guaranteed infinite gain margin; moreover, they have a guaranteed 50 percent gain reduction margin and a 60 degree phase margin in each feedback channel, provided that the system is linear in the control and the penalty to the control is quadratic, thus extending the well-known properties of LQ regulators to nonlinear optimal designs. These results are also valid for infinite horizon, average cost, stochastic optimal control problems.**

I. INTRODUCTION

REGULATOR design for dynamical systems is usually performed on the basis of a nominal model of the plant to be controlled. Modeling errors are unavoidable and, in fact, often desirable because they may result in simpler designs. It is therefore essential that the regulator based on the nominal model is robust; that is, it preserves its qualitative properties (namely, the stability of the closed-loop system) in the face of modeling errors.

The robustness and sensitivity to modeling errors of controlled linear systems has been extensively studied in the past [2], [6]. The robustness (stability margins) of regulators has been traditionally described in terms of gain and phase margins, although more recent approaches [3], [9], [12] focus on the singular values of the return difference or of the inverse return difference matrix.

One of the most appealing features of optimal linear quadratic (LQ) regulators are their guaranteed stability margins. Namely, LQ regulators remain stable when the control gains are multiplied by any number greater than 1/2. They also have guaranteed phase margins of 60 degrees [1], [13], [14], [16]. These results can be obtained directly by appropriately manipulating the associated Riccati equation [13].

A recent paper by Glad [5] has shown that gain margins of optimal regulators for nonlinear systems can be derived from the associated Hamilton–Jacobi–Bellman (HJB) equation, under suitable assumptions. This result ties nicely with the results on LQ regulators because the Riccati equation is a direct consequence of the HJB equation associated with LQ problems. However, the results of [5] are only applicable to single-input, deterministic systems, perturbed by memoryless nonlinearities, thus allowing only derivation of gain margin results; no phase margin results were derived in [5].

In this paper we derive general robustness margins of optimal

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