Conditioning of the Spinal Stretch Reflex: Implications for Rehabilitation

The purpose of this article is to describe a new technique that can potentially be applied to patients with hyperactive spinal stretch reflexes (SSRs). The progression of clinical research from conditioning of individual muscles or muscle groups (electromyographic biofeedback) to conditioning SSRs is explained. Research data from subhuman primates in addition to the first human experiments are reviewed. Potential applications of SSR conditioning are discussed, as are the issues requiring further delineation and research before the specificity of a training effect can be ascertained. [Wolf SL, Segal RL. Conditioning of the spinal stretch reflex: implications for rehabilitation. Phys Ther. 1990;70:652-656.]

Key Words: Biofeedback, Electromyography, Motor activity, Stretch reflex, Stroke.

During the past two decades, physical therapists have come to understand that the term biofeedback means the use of instrumentation to make individuals aware of one or more covert physiological processes through continuous presentation of visual and auditory representations of that (those) process(es). If one believes that biofeedback has a unique effect on enhancing physiological function, then certainly the speed information is conveyed to the patient and the specificity of that information, as defined by the placement of transducers, must account in part for improvement when biofeedback is compared with control or conventional therapeutic interventions. Indeed, numerous controlled studies governing return of functional capabilities following stroke or other central nervous system pathologies have been reported (for detailed reviews, see works by Wolf and Fischer-Williams or Krebs). Controlled clinical studies have also demonstrated that muscle biofeedback can facilitate recovery from musculoskeletal trauma following anterior cruciate ligament repair or meniscectomy, repair of hand injury, surgery for shoulder instability, and episodes of back pain. Collectively, existing data seem to imply that biofeedback is a valuable treatment adjunct with potential to augment function, particularly when proprioception or other components required for maximal sensorimotor integration are not disrupted.

If one now permits a more dynamic perspective on how muscles can be retrained using feedback paradigms, an alternative to conventional muscle biofeedback emerges. The typical interface of machine to patient necessitates the placement of reference electrodes over a specific muscle or muscle group with the intent of providing visual and auditory cues regarding the behavior of the target muscle(s). This arrangement offers little definitive information regarding the relative excitability of the spinal motoneuron population to that muscle or to that of any synergists because the recording site picks up activity representative of only subsets of motor units contributing to overall muscle activation. Furthermore, uncertainty exists about the specificity of location from which processed electromyographic (EMG) signals originate, especially when one realizes that the content of surface EMG signals is governed by such factors as...
interelectrode separation, subcutaneous tissue density, and the cutoff frequencies used to filter the EMG signals. Therefore, the question arises about how to better access the nervous system to condition muscles. One option is to evoke spinal stretch reflexes (SSRs) and attempt to voluntarily alter the magnitude of such responses.

The Spinal Stretch Reflex

The SSR is induced by a sudden stretch to a muscle or muscle group that acts synergistically about a joint. The muscle can be at rest or can be perturbed; that is, the muscle can be stretched (lengthened) as it contracts. Because stretch to a voluntarily contracted muscle usually elicits a threephase response, only the earliest, or shortest-latency, response engages spinal mechanisms exclusively. The SSR is thought to be mediated primarily by a two-neuron monosynaptic arc, although an oligosynaptic component may also contribute to the SSR. The afferent limb of the arc consists of Ia fibers from muscle spindles that respond to the stretch of their parent muscle. The Ia fibers then excite alpha motoneurons to activate the efferent phase of the arc. Surface electrodes are also used to record the SSR. The recorded response when SSRs are elicited presumably represents a population of motoneurons synchronously activated by the monosynaptic drive onto them. In this regard, the recording is unlike surface EMG monitoring during voluntary movement, which continuously records asynchronous muscle activity.

The electrical analogue of the SSR, the H-reflex, has been used in clinical neurophysiological studies for years to gather an index of motoneuronal excitability. Thus, for example, if the posterior tibial nerve is excited through electrical stimulation, an orthodromic direct response (M-response) can be recorded from surface electrodes placed over the soleus muscle. A longer-latency Hoffmann reflex (H-reflex), representing orthodromically conducted, low-threshold, spindle-afferent input to the spinal cord causing monosynaptic activation of alpha motoneuron, is subsequently recorded at the same soleus muscle site. If H-reflex responses to constant posterior tibial nerve stimulation yield low variability, then changes in amplitude of the H-reflex to various clinical interventions (eg, stretch, vibration, topical anesthetics) will presumably indicate changes in either motoneuron excitation thresholds or reflex gains to these interventions.

Conditioning the Spinal Stretch Reflex—Nonhuman Findings

Over the past 8 years, Wolpaw and colleagues have examined the capability of nonhuman primates to alter the magnitude of the SSR as part of a systematic study of the anatomical and physiological substrates governing memory. If monkeys could be trained to alter the size of an SSR without changing prestretch muscle length or the amount of supraspinal drive necessary to maintain a constant contraction before the stretch perturbation, then some innate “learning” at the spinal level could be demonstrated.

Wolpaw operantly conditioned the biceps brachii muscle SSR in monkeys by placing the forearm in a manipulandum and stretching the biceps brachii muscle only when the elbow joint angle and the biceps brachii muscle’s EMG activity met selected criteria. Light-emitting diodes (LEDs) indicated to the subject that the elbow was in the correct position and that the rectified EMG signal was neither too high nor too low. Furthermore, the time of perturbation after these criteria were met was randomized to prevent the development of expectation levels. If the correct response was made, that is, increasing or decreasing the size of the SSR above or below a set criterion, the monkey received a juice reward, and the criterion level was then changed to help “shape” future responses in the appropriate direction (much like the level detector or threshold in a muscle biofeedback machine is used to shape subsequent patient efforts).

Indeed, Wolpaw and colleagues could increase (uptrain) or decrease (downtrain) the magnitude of the SSRs, and Wolpaw could even train animals to reverse the changed response, thereby demonstrating an adaptive plasticity. Monkeys would often undergo several thousand muscle stretches a day and in the process demonstrated a two-phase learning curve. Appropriate change in stretch reflex behavior was seen in the first several days, followed by a second appropriate, but slower, change in SSR amplitude over several months. Both phases of training were shown to persist after the conclusion of training to change either SSR or H-reflex activity. Perhaps the most remarkable aspect of the Wolpaw conditioning paradigm, however, was the realization that, following total spinal cord transection, animals previously subjected to H-reflex training, but now unable to exert control over the H-reflex, showed SSRs at levels attained prior to the lesion, thus suggesting evidence for memory circuits within the spinal cord neural network itself. Thus, whether conditioning was achieved through mechanical stretches or electrical stimulation of peripheral nerve, both persistence and “memory” of changes in responses appeared to occur within the spinal cord itself.

Conditioning the Spinal Stretch Reflex—Human Findings

From a clinical perspective, the questions of interest become whether human SSRs can be conditioned and, if so, might hyperactive SSRs be successfully downtrained or flaccid muscles be uptrained with less effort and in a more timely manner than demonstrated with nonhuman primates. Preliminary studies by Neilson and Lance suggested that elements of the response to muscle stretch would be attenuated in clients with cerebral palsy.

We have embarked on a series of experiments designed to address these and other questions. Figure 1A shows EMG, torque, and joint-
for mean peak-to-peak-amplitude SSRs under the control condition, and the other bar graph showed the value of the last response in relation to the control responses. The subject had to either increase or decrease the SSR. For every four correct responses (below or above control bar graph values), the control level was moved 1% further in the appropriate direction, that is, made taller in the uptraining condition and shorter in the downtraining condition. The example presented in Figure 3 shows that this downtraining subject had already made eight responses below the criterion level (ie, 98% of the criterion level) and that the last response (right bar) was at 90% of the criterion level. In this manner, SSR behavior was "shaped" similarly to the use of the threshold levels or goal-setting of an EMG biofeedback machine.

Human subjects can tolerate about 400 muscle stretches over a 1-hour period and, unlike monkeys, need not work for thousands of responses. In preliminary studies conducted by Evatt and colleagues,25 four uptrained subjects demonstrated a median increase in SSR amplitude of 65.7% over nine training sessions, whereas five downtrained subjects demonstrated a median reduction in SSRs of 21.7% over nine training sessions. Follow-up control-condition evaluation approximately 3 weeks later showed that the directionality of the change had persisted over that time;

**Figure 1.** Example of raw electromyographic (EMG) data illustrating biceps brachii muscle stretch reflex response (arrow), torque generated by 40-msec pulse, and displacement of manipulandum for single (A) and multiple (B) stretches.

displacement recordings resulting from the elicitation of a biceps brachii muscle SSR in a healthy subject. The arrow indicates the onset of the SSR. This reflex is readily reproducible, as demonstrated in Figure 1B, which shows four superimpositions of the same response. The biceps brachii muscle SSR was produced through a stretch of approximately 20 degrees with the elbow positioned and maintained at 90 degrees (SD = 5). The subject was required to produce a biceps brachii muscle contraction of approximately 15% of maximal voluntary contraction. Appropriate background EMG levels were monitored through use of a window discriminator set to actuate an LED when EMG criteria were met. The LEDs monitoring elbow joint position signaled when the elbow was positioned at 90 degrees (SD = 5). In this manner, background supraspinal drive and joint position were always controlled. At a random time 1 to 5 seconds after the criteria were met, a computer activated a torque motor, which produced a 40-msec pulsed stretch of the biceps brachii muscle, shown as displacements on the lowest traces of Figure 1.

Figure 2 provides an example of a typical response in a subject being trained to dampen (downtrain) his biceps brachii muscle SSRs, using the identical criteria previously noted. Knowledge of results was provided immediately through bar graphs displayed on the computer screen (Fig. 3). One bar graph depicted the value

**Figure 2.** Example of raw electromyographic (EMG) data illustrating occurrence of biceps brachii muscle spinal stretch reflex (arrow) in subject undergoing downtraining. Torque generated by 40-msec pulse, and displacement of manipulandum also shown.
Baseline, control, and training sessions consisted of 250 random stretches, which occurred 1 to 5 seconds after maintaining the elbow joint at 90 degrees (SD = 5). Biceps brachii muscle EMG activity was maintained at a level (typically 100 μV) necessary to resist a small extension torque. The window about this EMG level was variable because of high background EMG responses across subjects. However, the window for each subject was kept constant across sessions.

There was no sequence effect (that is, control followed by training, or vice versa). We then pooled the control data from both groups and the training data from both groups. Figure 4 shows that all pooled median SSR magnitudes during experimental (treatment) sessions were below the baseline level. On the other hand, median SSR magnitudes during control sessions were more variable, with some above and some below the baseline level. These differences between control and treatment sessions were significant ($P < .007$, repeated-measures analysis of variance). The average reduction in amplitude during the training interval was 35.3% (SD = 37.0%, range = decrease of 66.0% to increase of 32.0%). Therefore, these preliminary findings suggest that the biceps brachii muscle SSRs of stroke patients may be downtrained.

Further studies are needed to document whether certain lesion sites allow successful training and other lesion sites prohibit successful training. Moreover, functional correlates of successful training need to be demonstrated before this technique is widely used.

**Clinical Applications**

If long-term studies with appropriate follow-up indicate that the hyperactive biceps brachii muscle SSRs can be dampered, then this technique may become clinically useful. In addition, studies enhancing hypoactive muscle responses may hold promise. A pri-

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**Figure 3.** Example of visual feedback provided to subject following elicitation of a spinal stretch reflex (SSR). Horizontal line is present criterion value for biceps brachii muscle electromyographic (EMG) activity. Left bar is initial criterion EMG value for session. Right bar is representation of SSR, expressed as percentage of initial criterion value.

that is, downtrained subjects demonstrated reduced SSRs and uptrained subjects demonstrated increased SSRs, even though there was no training (or feedback of stretch amplitude) in the follow-up session. Because these were single follow-up visits, no effort was made to test the significance of these maintained changes.

Clearly, these data suggest that the human nervous system can be conditioned by monitoring and feedback of the SSR. One critical question then becomes whether heightened SSRs can be downtrained. Preliminary data on stroke patients subjected to this treatment procedure using identical criteria to those established for healthy subjects, but fewer trials, appear encouraging. We have examined the ability of six stroke patients (age [in years]: $\bar{X} = 58$, SD = 5, range = 49–64; time post-lesion [in years]: $\bar{X} = 5.3$, SD = 2.0, range = 2.0–7.5) to downtrain hyperactive biceps muscle SSRs. Each subject participated in two baseline sessions without feedback of SSR amplitude. Subjects then participated in nine additional baseline (control) sessions, followed by nine SSR downtraining sessions, or vice versa.

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**Figure 4.** Percentages of change from baseline of data pooled from both study sequences. Notice that all experimental (treatment) sessions (hatched bars) are below baseline, whereas control sessions (plain bars) are above or below.
mary concern in any clinical application would be the computer interface to a torque motor and the expense of the motor itself. The combined cost of these two items, however, is less than half the cost of most computer-interfaced exercise equipment capable of monitoring isokinetic or eccentric muscle contractions. More importantly, the entire training paradigm can be computer controlled and directed, thereby not placing excessive demands on the clinician.

Before this procedure can be considered for clinical adoption, however, numerous issues must be resolved, including whether the downtraining effect persists, especially in the absence of pharmacological agents to manage spasticity; synergistic muscles are also engaged and downtrained; and functional improvement, in terms of speed or fluidity of movement, accompanies SSR conditioning. As one might anticipate, SSR retraining may have profound influences on reshaping movement control in patients with acute or chronic spinal cord injury and may even provide clues regarding synaptic reorganization within the spinal cord following trauma or surgical interventions to repair injured spinal cord tissue.

If SSR conditioning produces intrinsic changes within spinal cord elements, as has been suggested by some clinical and laboratory data\(^{28-30}\) then the possibility that motoneurons may change their responsiveness to both excitatory and inhibitory presynaptic events exists. Accordingly, whether enhanced movement or reaction time is linked to uptraining of SSRs can also be evaluated. If this relationship is demonstrated, then the implications for SSR conditioning to enhance hypoactive motor function, sports performance, or responses to aversive or threatening behaviors are obvious.

The availability of high-technology instrumentation to enhance rehabilitative efforts is present and growing at an unimaginable rate. The capability to condition responses within the central nervous system, whether they are elicited through SSRs or through evoked potentials anywhere within the neuraxis, is now possible. The extent to which such conditioning techniques affect physical restorative processes will inevitably become an important topic for exploration and application within the last decade of this millennium.

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References
