H Reflex Suppression by Thalamic Stimulation and Drug Administration*

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In 1918, Hoffman described a method of eliciting, in man, an easily identified monosynaptic response, the H reflex. Classicaly, the H reflex is initiated by electrical stimulation of a mixed nerve, such as the posterior tibial nerve in the popliteal fossa. The H reflex is recorded from the gastroc-soleus muscles by needle or overlying surface electrodes. While the posterior tibial nerve is the conventional site of stimulation, other stimulus sites are applicable. We have also used the femoral and ulnar nerves to obtain monosynaptic responses from the muscles they innervate.

In interpretation of the oscilloscope recordings (Fig. 1) evoked by stimulation of the posterior tibial nerve, it must be remembered that the stimulus affects both sensory and motor fibers. With low intensity stimulation, the large afferent fibers are the first to respond. This afferent impulse courses to the spinal cord and synapses directly with the large alpha motor neuron of the anterior horn. A monosynaptic response is evoked, and the H reflex appears in the calf following a 30-msec delay. The H reflex amplitude increases with increments in the stimulus shock. When stimulation is of sufficient magnitude, smaller fibers of higher threshold respond, producing the M wave, which results from direct stimulation of motor fibers and appears in the calf 6 msec after posterior tibial nerve stimulation. As the stimulus increases in intensity, the amplitude of the M wave increases, and that of the H reflex decreases. This decrease in the H reflex is presumably a result of antidiromic blockade.6,7,8

Study of the H reflex in patients with Parkinson's disease and its modulation by drugs known to produce a Parkinson-like state was undertaken in the hope that some correlation with previous studies in the cat and the human could be achieved. Animal experimentation has shown that stimulation of the ventrolateral nucleus of the thalamus (VL) can produce the following effects:

1. Inhibition of the contralateral knee jerk6

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* Corrected proof.
2. Inhibition of the contralateral gastrosoleus muscle spindle discharge
3. Facilitation of spinal alpha motor neurons as monitored in curarized nonnarcotized cats.

Consistent reduction or abolition of the contralateral knee jerk during and following VL stimulation was reported by Ward and Stern. Ward further noted that when inhibition of the tendon jerk did not result from VL stimulation, thalamic lesions at these stimulus sites were not as beneficial, particularly with reference to the relief of rigidity.

Past studies with chlorpromazine have shown that this drug inhibits gamma efferent and muscle spindle discharges. Chlorpromazine facilitates the monosynaptic response in the non-narcotized curarized cat.

The administration of reserpine in high dosage confers rigidity, akinesia, and tremor on rats and humans. Gamma efferent activity in the rat is abolished when the reserpine dose is sufficient to induce cogwheel rigidity. In our study, reserpine suppressed the H reflex; Arrigo, et al., have reported similar results.

Method

Stimulation studies were performed on parkinsonian patients at Parkland Memorial and St. Paul Hospitals. A monopolar stainless steel electrode with a 2 mm bare tip was placed in VL by conventional stereotaxic means. Stimulation and recording studies were performed 3 to 24 hours following electrode placement. A lesion was produced after stimulation studies were carried out. Shifting the electrode along its tract also allowed stimulation of the posterior internal capsule and the subthalamus.

H reflexes were evoked by stimulation of the posterior tibial or femoral nerves with either needle or surface electrodes. H reflexes were recorded from the gastrosoleus or quadriceps muscles with monopolar needle electrodes or with chlorided silver discs.

Pharmacological studies were made with volunteers from the neurosurgery ward at Parkland Memorial Hospital. These subjects were alert, cooperative, and without motor dysfunction. H reflexes were recorded from 25 volunteers while undergoing infusion with 5% dextrose in Ringer's lactate solution. Volunteers received either 75 mg chlorpromazine, 10 mg reserpine, or 5 mg diazepam. The drugs were administered through an intravenous tubing over a 1-min period. The H reflexes were then recorded for a subsequent 20-min period. Photographs were taken at the time of maximum H reflex suppression.

Physiological data were collected using conventional Grass amplifiers and recorded and stored on tape using a Sanborn-Ampex tape recorder. Review on the Tektronix oscilloscope model 502 was carried out and photographs obtained with a Grass camera.

Results

Most studies of the H reflex, in the past, have utilized the response recorded from the gastrosoleus muscles. In this study, it was found that the quadriceps muscle could be used as easily. H reflexes could also be recorded from the triceps muscle in accordance with Liberson's view that the H reflex can be monitored from all muscles under proper circumstances.

Figure 1 shows the characteristic M wave and H reflex. The shock artifact is followed after a short latency, by the direct muscle response, the M wave, and the H reflex appears after a latency period of 30 msec. Figure 2 depicts the basic findings of this study.

VL Stimulation. In Fig. 2, A-1 shows single-frame H reflex control recordings from a needle electrode in right gastrocnemius muscle of a patient with Parkinson's disease. An electrode had been placed in the contralateral ventrolateral nucleus of the thalamus 3 hours before the recording. A-2 demonstrates single-frame H reflexes in this same patient, suppressed by simultaneous stimulation of the left ventrolateral nucleus of the thalamus with pulses of 5 V, at 20/sec, of 1 msec duration. We found suppression of H reflexes during VL stimulation in five different parkinsonian patients.

Chlorpromazine. B-1 shows single-frame H reflex control recordings in one of the volunteers without motor dysfunction; B-2 demonstrates suppression of H reflexes in the same subject by chlorpromazine. An intravenous dose of 75 mg was given over a 1-min period, and the photographs were taken at the time of maximum H reflex suppression which occurred 4 min after in-
H Reflex Suppression

Fig. 2A. Thalamic stimulation. A-1. Single-frame H reflex control recordings produced by monopolar needle electrode stimulation of posterior tibial nerve with 11 V, 1.5 msec pulses, at 1/sec. Recorded by needle electrode in lateral belly of muscle. A-2. Suppression of H reflex produced by stimulation of left VL with 15 V, 2/sec, 2 msec duration pulses. Brain stimulus artifact is seen following the diminished H reflex. Tremor was evoked by this stimulation of VL.

Fig. 2B. Chlorpromazine administration. B-1. Single-frame H reflex control recordings produced by left posterior tibial nerve stimulation with 130 V, 4/sec, 1 msec pulses. Silver skin surface electrodes for recordings and stimulation. B-2. Suppression of the H reflex following intravenous administration of 75 mg chlorpromazine. Photograph taken 4 min after injection.

Fig. 2C. Reserpine administration. C-1. Single-frame H reflex control recordings produced by left posterior tibial nerve stimulation with 95 V, 0.5/sec, 0.1 msec pulses. Silver skin surface electrodes for recording and stimulation. C-2. Suppression of the H reflex following intravenous administration of 19 mg reserpine. Photograph taken 5 min after injection.

Fig. 2D. Diazepam administration. D-1. Single-frame H reflex control recordings produced by left posterior tibial nerve stimulation with 115 V 0.5/sec, 0.1 msec pulses. Silver skin surface electrodes for recording and stimulation. D-2. Suppression of H reflex following intravenous administration of 10 mg diazepam. Photograph taken 4 min after injection.

We have performed this experiment on 10 different volunteers, using doses of chlorpromazine varying between 25–75 mg, and found maximal suppression with 75 mg.

Reserpine. C-1 again shows single-frame H reflex control recordings in a different volunteer: C-2 demonstrates suppression of H reflexes in the same subject following reserpine. An intravenous dose of 10 mg was given over a 1-min period. Photographs were taken 5 min after injection at the time of maximum H reflex suppression. Consistent results were obtained in five other volunteers using the same dosage of reserpine.

Diazepam. D-1 shows single-frame H reflex control recordings in still a different vol-
unteer; D-2 demonstrates suppression of H reflexes by diazepam. An intravenous dose of 5 mg was given over a 1-min period, and the photographs were taken 4 min after injection at the time of maximum H reflex suppression. Consistent results were obtained in five other volunteers using the same dosage of diazepam.

Of six parkinsonian patients subjected to this study, five had excellent relief of rigidity and tremor. These same five had H reflex suppression evoked by VL stimulation. Stimulation also elicited tremor, especially in the arm. The sixth patient, who did not demonstrate H reflex suppression with VL stimulation, had relief of tremor without relief of rigidity.

Discussion
As a monosynaptic response, the H reflex is subject to the modulating influences converging on the alpha motor neuron pool from local, segmental, intersegmental, and supraspinal levels. It has been postulated that parkinsonian rigidity is an expression of abnormal influences operating on this segmental alpha motor neuron pool. Suppression of the H reflex by VL stimulation and administration of chlorpromazine and reserpine correlates well with previous experimental studies reported elsewhere.

We believe that this H reflex suppression is probably a function of inhibition of gamma motor activity (the efferent input to the muscle spindle) with resultant diminished tone of the muscle spindle, decreased spindle afferent discharge, and consequent suppression of H reflexes and tendon jerks. We agree with Steg and with Angel and Hofmann that experimental evidence does not lend great support to the hypothesis that parkinsonian rigidity stems from gamma motor neuron hyperactivity or increased spindle afferent activity driving the alpha motor neurons.

We further believe that study of the H reflex will prove of great value in the understanding of both normal and pathological states of spinal cord function in the intact human subject.

Summary
Studies in man indicate the following:
1. Stimulation of the ventro-lateral (VL) nucleus of the thalamus suppresses the H reflex.
2. Chlorpromazine, reserpine, and diazepam suppress the H reflex.
3. The effectiveness of lesions of VL in reducing parkinsonian rigidity correlates well with H reflex suppression in response to prior stimulation of the lesion site.

References