geal and gastric motility were recorded through open tubes connected to Statham pressure transducers which recorded on a dynograph; carotid pressure was recorded with the use of a Statham transducer. In 6 of the 20 dogs, stomach contractions of significant extent were seen during, or shortly after, stimulation (Fig. 1). In a number of animals which gave negative results, section of the esophagus and of the vagus nerves just above the diaphragm was followed by gastric contractions upon central-vagus stimulation. In a number of animals showing positive results, that is, gastric contractions upon central vagus stimulation, the height of the contractions increased considerably after section of the esophagus and of the vagi. There was no apparent correlation in the incidence of gastric contractions and changes in respiration or in blood pressure. In order to check the role of sympathetic impulses on gastric motility after stimulation of one or of both central ends of the vagi, the left, the right, or both stellate ganglia were excised; in two dogs, gastric contractions were abolished, while in three dogs, gastric contractions remained unaffected. It seems that pathways of impulses vary in individual animals.

The afferent and efferent pathways of this phenomenon are not known. The vagus is a mixed nerve and central stimulation travels by way of both cholinergic and adrenergic fibers. Beside nervous mechanisms, hormonal effects may play a role.

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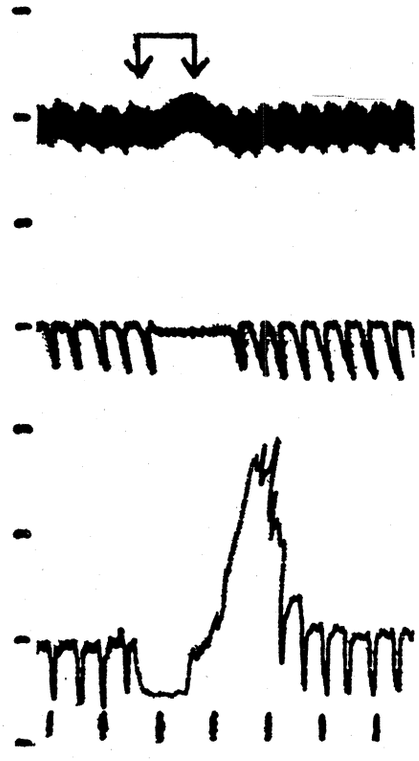

Fig. 1. Effect of stimulation of the central ends of the cut vagus nerves in the neck of a dog (cut at C_4), and esophagus and vagi cut above the diaphragm. Top, blood pressure in millimeters of mercury; center, esophageal motility, respiration is also shown; bottom, gastric motility. Arrows indicate stimulation of central ends of vagus nerves (6 v, 60 cy, 2-msec pulses). Lines on ordinate are in centimeters. Stimulation raised blood pressure 69 mm-Hg; respiration was arrested for 18 seconds; gastric tonus was depressed initially, the latent period of gastric contraction from beginning of stimulation was 18 seconds, maximum height of gastric contractions was 18.7 cm of water, and duration was approximately 20 seconds.

Note

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Phosphorylase *a* Activity in Uterine Muscle: Stimulation by Ethylenediaminetetraacetic Acid

Abstract. *Injection of ethylenediaminetetraacetic acid into spayed rats or its use in phosphorylase extraction increases the activity of glycogen-phosphorylase* a *in uterine smooth muscle tissue. Since the total phosphorylase activity is not increased, the increase in phosphorylase* a *appears to result from a conversion of inactive phosphorylase* b *to the active form of the enzyme.*

Ethylenediaminetetraacetic acid (EDTA) is used in the extraction of phosphorylase from skeletal muscle since it inhibits the formation of active phosphorylase *a* from the inactive phosphorylase *b* by chelating bivalent metallic ions reported to be essential in this conversion (*1*). Our experiments show that EDTA, either administered to rats or added to the extracting medium, increases the phosphorylase *a* activity in uterine tissue (smooth muscle). Since phosphorylase *a* is believed to be the form of the enzyme that is active in vivo, the small changes in total phosphorylase (*t*) reported below do not appear to be of biological significance.

The uteri of adult spayed female rats were removed and assayed for active phosphorylase *a* and total phosphorylase (*2*). In the experimental groups 60 μmole of EDTA, the calcium or magnesium salt of EDTA, Ca, or Mg per gram of tissue were added to the 0.1*M* NaF solution used in homogenization. The free-acid form of EDTA was used; the *p*H of each solution was adjusted with NaOH to that of the glucose-1-phosphate used in the reaction (*p*H 6.1). Control uteri were homogenized in 0.1*M* NaF.

Table 1. Activity of uterine phosphorylase *a* and *t* after additions to the homogenate of 60 μmole of the materials indicated per gram of tissue, reported as micrograms of P formed from glucose-l-phosphate by 100 mg of tissue in 10 minutes at 37°C. The values given are the mean of at least four determinations with ± S.E. of mean.

Phosphorylase activity (μg P)		Ratio (a/t) × 100
a	*t*	
	Control	
85 ± 9	332 ± 9	26.1 ± 3.0
	Mg or Ca	
83 ± 8	320 ± 4	26.0 ± 6.5
	EDTA	
200* ± 11	298† ± 7	66.7* ± 1.7
	Mg or Ca salt of EDTA	
188* ± 7	298† ± 12	63.4* ± 4.0

* $P < .001$; † $P < .02$.

The effect of the different additions to the homogenate on phosphorylase activity is shown in Table 1. The phosphorylase *a* activity and the *a* to *t* ratio were significantly increased by EDTA. When the calcium or magnesium salt of EDTA was used, the percentage change in phosphorylase activity (both *a* and *t*) was the same as that produced by EDTA alone and the values are combined in Table 1. No effect on uterine phosphorylase activity was observed if $CaCl_2$ or $MgCl_2$ was added in the same concentration; these values are also combined in Table 1.

When the tissue was extracted in a medium containing 0.1*M* NaF and 60μ*M* EDTA and then dialyzed against 0.1*M* NaF, the phosphorylase *a* activity expressed as micrograms of P liberated was 115 as compared with 47 in the controls (average of two determinations). When homogenates of uteri prepared in 0.1*M* NaF were dialyzed against 0.1*M* NaF, subsequent addition of EDTA also increased phosphorylase *a* activity from 50 to 87 μg of P (average of four determinations, $P < .01$). Thus, the increase in phosphorylase *a* activity with EDTA is not dependent upon any dialyzable substance.

The blood concentration of EDTA reaches a maximum 1 hour after an intraperitoneal injection of an EDTA

Table 2. Activity of uterine phosphorylase *a* and *t* after intraperitoneal injections of 0.6 millimole of EDTA or its calcium salt per kilogram of body weight. Reported in micrograms of P as in Table 1. The values given are the mean of at least four determinations with ± S.E. of mean.

Phosphorylase activity (μg P)		Ratio $(a/t) \times 100$
a	*t*	
	Control	
83 ± 12	308 ± 21	26.7 ± 2.9
	EDTA	
165* ± 8	258 ± 9	64.2* ± 3.1
	Ca salt of EDTA	
212* ± 8	305 ± 6	69.9* ± 1.8
	NaCl†	
115 ± 11	342 ± 7	33.2 ± 2.8

* $P < .001$; † Same osmotic pressure as the Ca salt of EDTA.

solution (*3*). Therefore we injected EDTA and its calcium salt (0.6 mmole/kg of body weight in 1 ml volume at *p*H 7.3) intraperitoneally into spayed female rats and determined the uterine-phosphorylase activity. As a control, enzyme determinations were also made on uteri from rats injected with a solution of NaCl equiosmotic with the solution of the calcium salt of EDTA. The results (Table 2) show that there was a significant increase in phosphorylase *a* activity and the *a/t* ratio with both EDTA and its calcium salt but not with NaCl.

The increase in phosphorylase *a* activity with EDTA is not a universal property of smooth muscle since the phosphorylase *a* activity of taenia coli muscle from guinea pigs, homogenized in NaF and EDTA, was 220 μg of P per 100 mg of tissue while the activity of those homogenized in NaF alone was 400 μg of P (average of four assays, $P < .001$).

Since EDTA did not increase the total phosphorylase activity, the activation appears to result from a conversion of the inactive form to the active form of the enzyme. However, the mechanism of activation of uterine phosphorylase by EDTA is unknown at present and it appears to be unlike that reported for any other tissue (*4*).

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Adaptation to Displaced Vision: Visual, Motor, or Proprioceptive Change?

Abstract. *After seeing his hand through wedge prisms, a subject points incorrectly with that hand at auditory as well as visual targets. The other hand is virtually unaffected. Thus the change cannot be solely visuo-motor or visual. Other evidence suggests that it is a change in felt hand location, rather than motor learning. When the subject's adapted hand feels as if it is pointing straight ahead, for example, it is actually pointing off to one side.*

When the images on a person's retinas are inverted, reversed, or displaced from their normal positions, the person at first misses when he reaches for objects. After some practice, he "adapts" and reaches more accurately (*1*).

Despite widespread interest in this adaptation and its implications, there has been no general agreement on the nature of the adaptation—on how the person who has adapted differs from one who has not. Even careful introspection provides no clear-cut answer: the detailed diary that Stratton kept while adapting to inverted vision (*2*) has sometimes been quoted to show that he experienced a shift in visual perception, sometimes to show that the only change was behavioral.

As Stratton found, inverting the retinal image leads to extreme disruption of visually guided behavior. This disruption disappears only after days of practice. But if the image is merely displaced, considerable adaptation may occur within minutes (*3*).

In the present experiment, people adapted to a sideways displacement produced by wedge prisms. Before putting on the prisms, and after taking them off, they pointed at visual targets, at sounds, and "straight ahead." The differences between the responses made before adaptation and those made afterward served as a measure of the adaptation.

Throughout the experiment, the subject sat at a table whose transparent top was slightly below his eye level. His head was held immobile by a bite-board. His task was to point (with his arm extended under the tabletop) at one of five rods sticking up from the tabletop, or at the sound of a clicker, or "straight ahead." The rods were spaced 4 inches apart in a row perpendicular to the subject's line of sight, with the central rod directly in front of his nose and 24 inches away from it. The clicker was held immediately behind one of the three middle rods, about 2 inches above the tabletop.

Before adapting, the subject pointed three times with each hand at each of the five visual targets, five times with each hand at each of the three auditory targets, and six times "straight ahead" with each hand. He did the same after adapting. Each subject pointed at the targets in a different mixed order determined by the experimenter. While pointing straight ahead or at the sound, the subject kept his eyes closed. When he pointed at the visual targets, an opaque cloth thrown over the table kept him from seeing his hand and arm.

While the subject was adapting, the opaque cloth was removed so that he could see his hand. He adapted for 3 minutes by pointing at the central visual target 90 times while wearing prisms that displaced his retinal images about 11° to the right or left. No fixation point was designated. Two subjects pointed only with their right hands while wearing the prisms base-left; two with right hands, prisms base-right; and two with each of the other two hand-prism combinations. The pointing motion was stereotyped: the subject started with his hand on a crossbar above his lap, from which he shot it forward and up, hitting the underside of the tabletop with the top of his forefinger. All subjects at first missed the target, but quickly became more accurate.

Table 1 presents the mean differences between measurements taken before and after adaptation (*4*). The measure was the point of intersection of the row of visual targets with a line from the subject's nose through his fingertip, as seen on photographs taken by an overhead camera.

It is clear that the adaptation effect transfers to all targets, regardless of their modality, but there is little or no transfer to the unadapted hand (*5*). Other investigators have also found that the effect does not transfer from the adapted to the unadapted hand in human beings (*6*, *7*) and in normal and "split-brain" monkeys (*6*).