comes $\Delta T_d(0, \Delta N) + K_d(IN) = \Delta T_n(0, \Delta N) + K_n(IN)$, but this is false since 87 msec $\neq$ 145 msec; the error would be 58 msec.

Briefly, the rest of the network for Becker's experiment is constructed as follows. Let r_d and r_n denote the responses to the digit and the tone, respectively. The subject was instructed to respond to the digit before responding to the tone. This constraint can be represented by a process P between r_d and r_n, having, perhaps, zero duration. We will show that $K_n(NP)$ is negative, so that N and P are in a Wheatstone bridge. Since $\Delta T_d(0, \Delta N) = \Delta N - S(N, r_d)$ and $\Delta T_n(0, \Delta N) = \Delta N - S(N, r_n)$ we have

$$\Delta T_d(0, \Delta N) - \Delta T_n(0, \Delta N) = S(N, r_n) - S(N, r_d) = -58$$

But since $S(NP) = S(N, r_d)$ (Fig. 1a),

$$S(N, r_n) - S(NP) = K_n(NP) = -58 < 0$$

Additional details are provided in (*7*).

I have sketched a method here for determining, at least in part, the schedule of processes the brain uses for a particular task. This method of analyzing reaction times considers more complicated arrangements of processes than most previous ones and yields more information. The next question, no doubt much harder, is Why does the brain choose the particular schedule it uses?

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6. Conceivably, a subject could produce constant values of $K(XY)$ through speed-accuracy tradeoffs in the various conditions, but such a strategy is unlikely to succeed. The speed-accuracy trade-off phenomenon suggests an interesting question: Given an empirically determined speed-accuracy (or time-cost) function for the entire task, characterize the class of possible speed-accuracy functions associated with the component processes.
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8. I thank B. Kantowitz, J. Neely, J. Solberg, and J. Townsend for helpful suggestions.

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Visually Induced Self-Motion Sensation Adapts Rapidly to Left-Right Visual Reversal

Abstract. *After 1 to 3 hours of active movement while wearing vision-reversing goggles, 9 of 12 (stationary) human subjects viewing a moving stripe display experienced a self-rotation illusion in the same direction as seen stripe motion, rather than in the opposite (normal) direction. This result indicates that the neural pathways which process visual self-rotation cues can undergo rapid adaptive modification.*

Visual and vestibular motion cues contribute to self-motion perception in a complementary fashion (*1*). In everyday life, head rotation results in an equal and oppositely directed angular motion of the visual scene relative to the head. This association between normal active head rotation and relative scene motion is believed to account for the phenomenon of "circularvection" (CV) (*2*): a pattern of stripes rotating around a stationary observer soon elicits a compelling sensation of self-rotation in the opposite direction (*3*).

When human subjects wear optics which either "mirror" reverse vision from left to right or invert it (rotate it by 180°), the normal relationship between left-right head rotation and relative visual scene motion is reversed. For example, head rotation to the right is accompanied by relative scene motion to the right; the seen world is no longer perceived as stationary (*4*). Spatial orientation is severely impaired. However, after an extended period of visual reversal (days to weeks), the seen world appears more stable (*4*), and subjective visual "normalcy" and coordinated movement are gradually restored (*5*). Active movement by the subject is thought to play a vital role in the adaptation process (*6*). The slow phase component of the horizontal vestibulo-ocular reflex (VOR), which contributes to perceptual stabilization under normal vision, reverses after a week or two of visual reversal (*7*). The neural pathways believed to mediate adaptive changes in the VOR have recently been explored in animals (*8*). One might also expect that the neural interpretation of visual self-rotation cues would reverse and be manifest perceptually in a reversal of the CV phenomenon. We have now demonstrated such a reversal, accompanied by only a modest reduction in vestibulo-ocular reflex gain, in 9 of 12 subjects within a brief (1- to 3-hour) period of exposure. Reversed CV could be demonstrated only when the moving stripe display size was limited to the field of view conditioned by exposure.

Two experiments, which differed somewhat in procedural details, were conducted with 12 adult volunteers with no overt oculomotor or vestibular disorders. As symptoms of motion sickness often occur under vision reversal, drugs (scopolamine, 0.5 mg; or scopolamine, 0.4 mg with Dexedrine, 5.0 mg) were orally administered before the experiment. In both experiments, left-right vision reversal was achieved using prism goggles, which permitted a binocular field of vision subtending approximately 45° horizontally and 28° vertically (*9*). (Horizontal dimensions will always be presented first.)

In both experiments, CV was tested before and immediately after a period of exposure to reversed vision (preliminary and final tests). In experiment 2, CV was also tested at intervals during the exposure period. Subjects were seated in the closed, motionless cabin of a flight simulator (Link GAT-1). Vertical light and dark stripes of equal width (6.4°) moving left or right at 8° per second were back-projected onto the translucent front window, about 70 cm from the subject. During testing without the goggles before and after exposure, the shape of the moving display could be varied through the use of appropriate masks applied to the window, whereas in tests made during exposure, the shape corresponded to the field of view of the goggles. Subjects were asked to verbally report the onset and disappearance of any CV. Because the goggles transposed the visual location of the hands, subjects were asked to report their perceived direction of self-motion with respect to the direction of seen stripe motion, with respect to their left or right eyes, or both. Motion reports made according to these two different methods were always consistent.

The gain and phase of the horizontal VOR were tested in the dark before and after exposure through the use of sinusoidal simulator angular motion (0.2 Hz, 30° per second peak velocity, 6 to 8 cycles). Eye movement was measured in the dark by conventional d-c electrooculography. Subjects performed mental arithmetic tasks to maintain alertness.

During exposure, when not participating in the brief CV tests, our subjects explored their reversed visual environment

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Table 1. Latencies (in seconds) on experiment 2 CV tests. Magnitude indicators are shown in parentheses; +++, strong vection (greater than 25 percent of stripe velocity); ++, vection present; +, sustained vection beginning after simulator rotation (to trigger vection) stopped; /, no vection even with triggering rotation. In some cases, CV latency was not measured. Boldface type indicates RCV.

Subject	Preliminary testing		Testing during exposure to reversed vision: intervals					Final testing	
	PVF	CVF	1	2	3	4	5	CVF	PVF
B1	4 (+++)	(/)	4 (+++)	7 (+++)	7.5 (++)	**3 (++)**	**5.5 (+++)**	**1.5 (+++)** **1.5 (+++)**	2 (+++)
B2	7 (+++)	5.5 (+++)	5 (+++)	11 (+++)	**19.5 (++)**	**4 (+++)**	5 (+++)	**23.5 (+++)** **21 (+++)**	19 (+++)
B3	11.5 (+++)	8 (++)	5 (+++)	21 (++)	(+)	14 (++)	16.5 (+++)	**15 (++)** **9 (++)**	9 (+++)
B4	9 (++)	(/)	(/)	(/)	18 (+++)	121 (++)	5 (+++)	**(+)** **3 (+++)**	3 (+++)
B5	(+)	(+)	23 (++)	18 (++)	12.5 (++)	63 (++)	8 (+++)	(/) (+)	3.5 (+++)
B6	10 (+++)	(+)	12 (++)	(+)	12 (++)	10 (+++)	60 (+++)	(+) (+)	20.5 (+++)
B7	(+)	(/)	(/)	(+)	(/)	(/)	(/)	(/) (/)	35 (+++)

by walking through the laboratory buildings in the company of an observer. Head and body movements in the horizontal plane were encouraged.

In experiment 1 (five subjects), we first confirmed that CV direction before exposure was opposite to seen stripe motion (normal CV) through the use of a 63° by 28° rectangular stripe display. Latency of CV onset ranged from 8 to 47 seconds. Then, after the prism goggles were fitted, the subjects exposed themselves to vision reversal for 180 to 230 minutes.

After the exposure period, the prism goggles were removed and CV was tested with a 30° by 19° display, so as to stimulate only the visual field previously exposed to the goggles. With a latency of between 10 and 42 seconds, all five subjects reported an unequivocal sensation of motion in the same direction as the seen stripe motion. Subjects usually likened the sensation to being "pulled along with the stripes." We refer to this perception as "reversed circularvection" (RCV). Four of the five subjects experienced compelling, sustained RCV until, some 30 to 60 seconds later, we suddenly increased the display size to 63° by 27°. Subjects then reported that RCV immediately ceased and, after several seconds, that normal (unreversed) CV appeared. Changing back to the narrow field abolished normal CV, and RCV was reestablished after several seconds. This sequence could be demonstrated repeatedly over several minutes. Active head movements (eyes open in light) appeared to abolish RCV. Our fifth subject experienced an initial 17-second period of compelling RCV, followed by periods of normal CV and RCV, even though the display area remained narrow.

To more systematically define the time course of CV adaptation, seven additional subjects participated in experiment 2, in which CV was systematically tested every 30 to 40 minutes during a 190-minute exposure period. To more clearly demonstrate the dependency of CV direction on the region of the visual field stimulated, CV was measured both before and after exposure with a rectangular central visual-field display (CVF) (29° by 17°) and with a peripheral visual-field display (PVF) (180° by 23°, with a dark mask of the CVF display area). Before exposure, the CVF test was preceded by a PVF test. After exposure, two CVF tests (one with each direction of stripe movement) were followed by a PVF test, and the head was immobilized in an effort to avoid readaptation. Subjects were asked to verbally report the first appearance of CV, its direction, and their sensation of self-rotation in 45° increments. In order to compare subjects, we converted these reports to a four-bin velocity scale (Table 1). When vection could not be demonstrated through the use of the display alone, we attempted to trigger it with a brief rotation of the simulator cab, thus providing a transient semicircular canal motion cue (present during normal head rotation, but absent using conventional CV test procedures). Four subjects demonstrated RCV (Table 1). The first RCV episode occurred after 95 to 190 minutes of exposure.

The VOR slow phase velocity gain and phase with respect to trainer velocity were measured in both experiments prior to the first CV test before exposure and also after the exposure, just before the goggles were removed. Data from two subjects in experiment 1 could not be analyzed because of technical problems. In the remaining ten subjects, VOR gain decreased from 0.72 [standard error (S.E.) = 0.35] before to 0.55 ± 0.29 after exposure (paired-sample t-test, $P < .001$). Among those seven subjects who experienced RCV, gain decreased from 0.80 ± 0.39 to 0.64 ± 0.31 ($P < .005$). Slow phase eye velocity of the ten subjects lagged trainer velocity by 180° ± 7° before exposure and by 176° ± 11° after exposure. As confirmed by direct inspection of the eye-movement records, no subject showed any evidence of reversal of the VOR slow phase component.

Our subjects showed a 17 percent reduction in VOR slow phase gain over their brief exposure period. This short-term reduction in gain could be due to uncontrolled changes in alertness caused by motion sickness or fatigue. Also, scopolamine depresses oculomotor responses (*10*). Alternatively, the reduction may represent the early stages of the adaptive VOR change described by Gonshor and Jones (*7*). Their subjects, who did not use drugs, showed similar changes in gain during the first day of exposure.

The RCV cannot simply be explained as a negative aftereffect (*11*), since RCV was demonstrated without preceding unreversed CV. Similarly, RCV is distinctly different from the brief episodes of inverted self-motion perception reported during prolonged (4- to 12-minute) optokinetic stimulation (*12*): RCV could be demonstrated after distinctly shorter latencies (on the order of seconds) with no preceding normal vection, was sustained for periods greater than 5 seconds (usually 20 seconds to more than 1 minute), and could be quickly and consistently manipulated by alter-

ing the display size. Rather, our results imply that vision reversal reversed the interpretation of visual information from the exposed portion of the visual field. This change was adaptive, in that it appeared to be directed toward the goal of veridical self-motion perception. The RCV could be demonstrated only when the field of view was limited to or less than the field of view of the goggles. With a wide field stimulus (as in experiment 1), RCV appeared to be overwhelmed by normal vection. In experiment 2, normal CV response to PVF stripe motion was, if anything, enhanced by exposure of the central field to visual reversal. Our findings suggest that visual information from the exposed central visual field is transmitted centrally along pathways separate from those carrying information from the peripheral visual field and that each of these pathways may be separately modified as a result of sensory experience. The occasional presence of alternating normal CV and RCV during early tests during exposure suggests that RCV may not develop gradually. Instead, it is as if a second competitive mechanism develops.

It is not surprising that the RCV velocity magnitudes reported were modest, given the narrow field of view of the goggles, as normal CV can be more effectively elicited if peripheral retinal areas are exposed to the moving display (*13*). Whether a subject demonstrated RCV within the allowed exposure period appeared to be correlated with CV strength produced with a narrow field stimulus. In experiment 2, subjects B1 through B4 showed strong normal CV at least once, either in CVF tests before exposure or during the first four sessions during exposure; they were the only subjects in this experiment to show RCV. Test scheduling constraints limited exposure to 190 minutes in experiment 2. Had the exposure for subjects B5 through B7 been extended, it is conceivable that they, too, would have shown RCV. The exposure duration in experiment 1 was not so constrained, and all five subjects experienced RCV.

In animals, convergence of visual and vestibular head rotation information occurs in neurons of the vestibular nucleus (*14*), which are thought to determine the slow phase velocity of vestibular and optokonetic nystagmus under many conditions. The extent to which vestibular nucleus neurons contribute to rotation perception is unknown, although there seems to be a close relationship between the time course of normal CV in humans (*15*) and the response pattern of some vestibular nucleus neurons in animals (*14, 16*). It would be interesting to know if a reversal in visual sensitivity of vestibular nucleus neurons can be demonstrated in animals after several hours of active exposure to vision reversal. It may be that both CV and VOR (*8*) adaptation are mediated by a common (possibly transcerebellar) mechanism that provides a reversed drive to neurons of the vestibular nucleus. The long latency (1 to 2 weeks) of VOR reversal (*7*) (when contrasted with the rapid CV reversal we found) may result from the presence of a direct, competitive input from primary semicircular canal afferents.

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Ethanol Specifically Potentiates GABA-Mediated Neurotransmission in Feline Cerebral Cortex

Abstract. *Ethanol (ethyl alcohol) potentiates the inhibition of cortical neurons by γ-aminobutyric acid. This effect is specific, since ethanol does not potentiate inhibition by glycine, serotonin, or dopamine. These results have implications for alcoholism because (i) γ-aminobutyric acid mediates anxiolytic mechanisms, and (ii) anxiety is implicated in the etiology of alcoholism.*

Ethanol (ethyl alcohol) is the oldest and most commonly used psychoactive agent. Most people consume alcoholic beverages without apparent harm, but an estimated 9 million Americans suffer from alcoholism (*1*). Ethanol shares many pharmacological effects with benzodiazepines and barbiturates (*2*), drugs which are known to potentiate neurotransmission mediated by γ-aminobutyric acid (GABA) (*3*). Furthermore, ethanol potentiates spinal presynaptic inhibition (*4*), which is also mediated by GABA (*5*). Finally, bicuculline (a specific GABA antagonist) diminishes the behavioral manifestations of ethanol intoxication, whereas amino-oxyacetic acid (an inhibitor of GABA catabolism) markedly increases these behavioral manifestations (*6*). I have therefore studied the interactions of ethanol with various hypothesized synaptic transmitters acting on single feline cortical neurons; ethanol specifically potentiated the inhibitory effects of GABA, but not the inhibitory effects of glycine, serotonin, or dopamine. Ethanol also potentiated the inhibition of single cortical neurons by electrical stimulation of the surface of the cerebral cortex; that inhibition is believed to be mediated by endogenous GABA (*7*).

The experiments were carried out on cats (2.5 to 4 kg, of either sex) anesthetized with Fluothane or methoxyflurane. The same results were obtained in five control experiments with the "isolated cerebrum," unanesthetized preparation (*8*). Multibarreled glass microelectrodes were used to make extracellular recordings from single neurons of the pericruciate cortex (*9*). The recorded action potentials (spikes) were amplified, gated, electronically counted, processed through a peristimulus histogram ana-

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