sleep than during time-equivalent eu-
thermic periods.

Torpor was usually terminated by in-
creased wakefulness soon followed by
an increase in $T_{br}$, but REM sleep was
not present. Despite the suppression of
REM sleep during torpor, compensatory
REM rebounds did not occur in the 24-
hour period after the return to euthem-
ia (9) (Fig. 1) (Table 1). The occurrence of
shallow torpor did not significantly affect
the amounts of total sleep or SWS during
subsequent euthermia when compared
with pretorpid euthermia.

Daily shallow torpor occurring in
some small mammals such as pocket
mice is sometimes interpreted as an ex-
tension or magnification of the euthemic
circadian rhythm of $T_{br}$. However, in this
study some bouts of torpor occurred in-
dependently at times remote from the
minor circadian decreases of $T_{br}$. There-
fore, the decreased $T_{br}$ of torpor appears
to be associated more closely with the
sleep state than with a preestablished
circadian variation of $T_{br}$ independent of
sleep.

These results also point to the physio-
logical identity of estivation and hiberna-
tion. Sleep patterns during shallow tor-
por in desert ground squirrels were qualit-
itively and quantitatively identical to
those of shallow hibernation in alpine
ground squirrels (10). Moreover, changes in EKG, EMG, and EEG activi-
y during bouts of shallow torpor were
typical to those of shallow hibernation.

The function of sleep is typically re-
garded as the restoration of one or more
physiological processes degraded during
prior wakefulness, in spite of a lack of
concrete empirical support for such an
interpretation (11). An alternative, but
not necessarily exclusive hypothesis is
that SWS evolved as an adaptation for
energy conservation that partially offset
the high costs of endothermy (12–14). Evidence for this hypothesis was until
recently confined to correlative studies
and included (i) strong positive correla-
tions between metabolic rate and amount
of SWS in mammals (15), (ii) the parallel
ontogeny of SWS and thermoregulation
(14), and (iii) the absence of SWS in ecto-
therms (15). The finding of a regulated
decrease in $T_{br}$ during SWS indicative of
reduced metabolism provided direct sup-
port for this hypothesis (5). Since the
electrophysiological patterns of SWS
and shallow torpor are temporally con-
tinuous and essentially isomorphic, it
is probable that thermoregulatory adju-
ments while entering torpor are an exten-
sion of those initiated during SWS.

There can be little doubt about the bioen-
thetic activation value of torpor, and the
physiological homologies between tor-
por and sleep described point to a uni-
itary primordial function for both states.

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7. The initial weight of this animal was 143 g. After
12 days of food deprivation it weighed 108 g and
after 26 days, 85 g.

8. The first instance of torpor for three animals
(torpor entrances at 10:00, 10:10, and 10:28
p.m.) and the second for the fourth animal (9:50
p.m.) served as the data base. Sleep-wakeful-
ess patterns were determined for time-equiva-
 lent euthemic periods before the first period of
torpor and after torpor. Comparisons between
pretorpid, torpor, and post-torpor and in this
subsequent comparisons were made by one-way
analysis of variance for repeated measurements.
Duncan’s multiple-range test was used to com-
pare specific means.

9. Euthemic sleep-wakefulness measures were
determined in the 24-hour period (8 p.m. to 8
p.m.) prior to the first period of torpor. Although
individual animals had experienced differing
diet deprivation with the varying times of oc-
currence of the first torpor, sleep-wakefulness
measures were as homeoegenous as and within
the range of normal data reported for another
species of squirrel (4). Sleep-wakefulness pat-
terns were measured in the 24-hour period that
torpor occurred (same periods of torpor as in
(8)) and in the subsequent 24-hour euthemic
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Membrane Potential Changes During Chemokinetics in
Paramecium

Abstract. Intracellular recordings show that (i) paramecia hyperpolarize slightly in
attractants and depolarize in repellents that depend on the avoiding reaction (an
abrupt change of swimming direction), and (ii) paramecia more strongly hyper-
polarize in repellents and more strongly depolarize in attractants that depend on
changes of swimming velocity. These membrane potential changes are in agreement
with a hypothesis of membrane potential control of chemokinesis in Paramecium.

Paramecia accumulate in or disperse from the vicinity of certain chemical stim-
uli (1). The animals do this by either of two mechanisms: modulation of fre-
quency of changing swimming direction (the avoiding reaction) or modulation of
forward swimming speed (2-4). Neither mechanism seems to involve orientation
toward or away from the stimulus, and therefore the behavior is termed chemok-
inesis rather than chemotaxis (5, 6). Components of both of the mechanisms
are under electrical control at the cell membrane: (i) each avoiding reaction
corresponds to the generation of a calcium action potential, and (ii) the fre-
quency and angle of ciliary beating, which determine swimming speed, are
controlled by changes from the resting membrane potential (7, 8). Therefore, it
is of interest to determine the electro-
physiological correlates of the com-
plex behavior of chemokinesis. I have
made intracellular recordings from cells in
attractants and repellents. The mem-
brane potential ($E_m$) values from these
recordings are presented here and are in
agreement with a hypothesis of mem-
brane potential control of chemokinesis
(2, 3).

Accumulation of organisms is associ-
ated with decreased frequency of avoid-
ing reaction ($F_{AR}$) or with decreased speed ($V$) in the area of attractant (5, 9).

Conversely, dispersal is associated with
increased $F_{AR}$ or with increased $V$ in the

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area of repellent (5, 9). However, in *Paramecium* one cannot separately affect the $F_{AR}$ and the frequency of ciliary beating that determines $V$ because both are under control at the cell membrane. For example, a small hyperpolarization will bring the membrane potential away from threshold and decrease $F_{AR}$ and will also increase the frequency of ciliary beating and angle, increasing forward $V$. Note that the decreased $F_{AR}$ accompanies accumulation, and increased speed accompanies dispersal of the animals. Therefore, not only are the components of chemokinesis inseparable, they appear to be opposed by the classical mechanisms outlined above.

Mutations and conditions that eliminate avoiding reaction have been used in determining the contributions of $F_{AR}$ and $V$ to chemokinesis (2, 3, 10, 11). Elimination of the avoiding reaction abolishes responses to only some attractants and repellents. In this manner, I have distinguished two groups of chemicals that cause chemokinesis by two different mechanisms, I and II (2, 3, 10). In mechanism I, attraction and repulsion are correlated with decreased and increased $F_{AR}$. The associated increase and decrease in velocity seem to be unimportant in determining net effect. Moreover, in "paws" [mutants with no avoiding reaction (12)] agents of group I do not cause appreciable attraction and repulsion. In mechanisms II, it is the response swimming velocity that predominates. Repulsion is associated with increased $V$ and a decrease of $F_{AR}$ to zero, and attraction is associated with decreased $V$ due to slow swimming and time spent in frequent turning in the avoiding reaction. Paws are attracted and repelled by agents of group II. Attractants I and repellents II cause the same qualitative changes in behavior (decreased $F_{AR}$ and increased $V$) but result in opposite chemokinesis behavior. Likewise, repellents I and attractants II cause qualitatively similar changes (increased $F_{AR}$ and decreased $V$) but have opposite chemokinesis results. The attractants and repellents used for intracellular recordings were organized in these groups.

The $E_m$'s of cells in attractants I, such as acetate (OAc), and repellents II, such as OH$^-$, measured by intracellular recording (Fig. 1) were more negative than those of the same cells in control solutions. The $E_m$'s of cells in repellents I, such as quinidine-HCl, and attractants II, such as BaCl$_2$, were more positive than the $E_m$'s in control solutions, and both repellents I and attractants II elicited frequent action potentials (Fig. 1). The membrane potentials of cells in several attractants and repellents and of controls are given in Table 1 along with measurements of the strengths of the attractants and repellents.

Variations in the measurements of $E_m$ arise from differences in resting $E_m$ between cells and possibly from gradual changes of electrode properties that alter apparent resting $E_m$. However, relative changes of $E_m$ upon changing solutions were consistent and in the same direction in all cells. To demonstrate this constancy, the net changes of $E_m$ upon changing solution from control to test and back were measured (Table 2). The changes of potential in attractants I and repellents II were always hyperpolarizing and were about twice as large for repellents II as for the strongest attractant I. The changes in repellents I attractant II were consistently depolarizations, and the depolarizations in attractant II were greater than those in any repellent I.

There are apparent contradictions in the chemokinesis behavior of animals in solutions that cause qualitatively similar changes in $F_{AR}$ and $V$ but cause opposite accumulation and dispersal results. For example, attractants I and repellents II both decrease $F_{AR}$ and increase $V$ but have opposite chemokinesis results (2, 3). A new mechanism of behavior control can be invoked for chemokinesis, or the observed behavior can be used to infer electrical events during chemokinesis based on the established membrane electrical control of *Paramecium* ciliary motion (7). I have taken the latter approach (2, 3). The resulting simple hypothesis of $E_m$ control of chemokinesis (Fig. 2) predicts that attractants I will cause a slight hyperpolarization, causing the characteristic decrease in $F_{AR}$ and small increase in $V$ (Fig. 2a). As attractants I more strongly hyperpolarize the potential, the $F_{AR}$ will drop toward zero, making the attractants less effective for accumulating animals. As the membrane potential is more strongly hyperpolarized, $F_{AR}$ drops to zero and the velocity increases become important and cause repulsion. Hence, repellents II should strongly hyperpolarize the $E_m$. In this way, the same qualitative change of $E_m$ and of behavior components can lead to two different chemooaccumulation results, depending on the magnitude of the membrane potential change.

The hypothesis also predicts a small positive shift in potential for cells in repellents I (Fig. 2a). The positive shift increases $F_{AR}$ and decreases $V$. As the

**Fig. 1.** Intracellular recordings from *Paramecium tetraurelia* in (a) 5 mM KCl control and 5 mM K acetate solutions; (b) 1 mM KCl (pH 7.0) control and 1 mM KOH (pH 8.7) solutions; (c) 0.1 mM KCl control and 0.1 mM quinidine-HCl solutions; and (d) 2 mM KCl control and 1 mM BaCl$_2$ solutions. Dashed lines indicate 0 mV. Horizonal lines in each panel are 2-second scales; vertical lines are 20-mV scales.

**Fig. 2. (a)** Graphical description of membrane potential control of chemokinesis. Change of membrane potential ($\Delta E_m$) from control (at origin) is plotted against the index of chemokinesis; $I_{the} > 0.5$ indicates attraction; < 0.5 indicates repulsion (see Table 1 legend). As chemical stimuli change $E_m$ relative to control, animals will be attracted or repelled, depending on the magnitude and direction of the $E_m$ change. (b) Data from Tables 1 and 2 plotted as $\Delta E_m$ produced by the attractant or repellents versus $I_{the}$. Scale of $\Delta E_m$ is different for depolarizing and hyperpolarizing stimuli.
membrane potential is even more depolarized relative to control, the change in \( F_{AR} \) increases and \( V \) decreases until the animals no longer escape by changing direction, but instead are trapped by very slow movement and accumulate. Therefore, attractants II are expected to more strongly depolarize the membrane than repellents I.

The membrane potentials measured in attractants and repellents are in agreement with the hypothesis (Fig. 2b). Strong attractants I hyperpolarized the membrane slightly, about 8 to 10 mV. A weaker attractant, potassium folate, hyperpolarized the membrane even more, presumably to a point near the transition between the mechanisms of attraction I and repulsion II. The repellent II, \( OH^- \), caused a stronger negative shift in potential than attractants I (\(-16 \text{ mV}\)), which was well correlated with the decrease in \( F_{AR} \) to zero and the repulsion by the resulting increased \( V \). Repellents I depolarized the \( E_m \) by up to +25 mV, while the attractant II depolarized even more (+31 mV).

Several observations can be made about the \( E_m \) data. Absolute \( E_m \) is probably not the determining factor in attraction and repulsion, but rather the magnitude of change of \( E_m \) from control determines attraction and repulsion (Fig. 2b). These changes of \( E_m \) are reversible (Table 2), and the strengths of attractants and repellents are not simply proportional to the \( E_m \) changes they produce (Fig. 2b).

Cells change \( E_m \) and behavior in response to new ionic environments. With time in the new solution, the cells accommodate their resting \( E_m \), a new threshold for action potentials is established relative to the new \( E_m \), frequency and angle of ciliary beating return to a resting level (13, 14), and \( F_{AR} \) and \( V \) return to basal levels (2, 10) despite the shift in \( E_m \) that has occurred. Although the eventual accommodation of electrical properties and accompanying adaptation of behavior are necessary for the classical mechanisms of chemokinesis (5, 15) and for the hypothesis of membrane potential control of chemokinesis (2, 3), the initial responses of the animals determine the direction (accumulation or dispersal) and strength of the response. Therefore, only initial membrane potential responses to attractants and repellents are reported at present.

Membrane electrical change may be a mechanism common to most chemosensory systems, while the nature of the change varies between systems: slime molds and macrophages hyperpolarize in response to attractants (16, 17), bacteria may hyperpolarize while adapting to either attractants or repellents (18) and require a Ca\(^{2+}\) flux across the plasma membrane for repulsion (19), and insects and vertebrates respond to food extracts and odors with increased frequency or bursts of membrane electrical activity (20–23). As more measurements of \( E_m \) in attractants and repellents are made, I would expect that ratios of \( F_{AR} \) or \( V \) in test and control solutions will modify the simple idea that changes in \( E_m \) control chemokinesis behavior.

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Table 1. Membrane potentials (\( E_m \)) of cells in test and control solutions. Cells were bathed in control or test solution of pH 7 (see Fig. 1 legend) except in acetic acid (pH 5.3) and KOH solution (pH 8.7). The \( E_m \) data are averages of \( N \) measurements ± 1 standard error (S.E.). The \( E_m \)'s were measured 6 minutes after the bath solution was changed because potentials were again stable by then. An exception was the \( E_m \) when the solution was changed from quinidine-HCl to KCl, in which case cells could take more than 8 minutes to recover a stable potential. The \( E_m \)'s in quinidine were difficult to measure accurately because cells in quinidine sometimes showed action potentials with plateaus prolonged for 2 to 3 minutes. The response of paramecia to chemicals was measured by a T-maze assay designed to present a test and control solution to a population of animals (24). The index of chemokinesis (\( I_{che} \)) is defined as the number of animals swimming into the arm of the T containing test solution divided by the number of animals swimming into both arms, containing test and control solutions. An \( I_{che} > 0.5 \) indicates attraction to the test solution; \( I_{che} < 0.5 \) indicates repulsion from the test into the control solution. The \( I_{che} \) data are averages of four or more experiments ± 1 standard deviation.

<table>
<thead>
<tr>
<th>Attractants I</th>
<th>Test solution</th>
<th>( E_m ) (mV)</th>
<th>( N )</th>
<th>( I_{che} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>KCl, 5 mM</td>
<td>K acetate, 5 mM</td>
<td>-31.3 ± 0.8</td>
<td>50, 16</td>
<td>0.84 ± 0.07</td>
</tr>
<tr>
<td>NaCl, 5 mM</td>
<td>Na lactate, 5 mM</td>
<td>-31.3 ± 0.8</td>
<td>50, 16</td>
<td>0.83 ± 0.06</td>
</tr>
<tr>
<td>NaCl, 5 mM</td>
<td>Na acetate, 5 mM</td>
<td>-27.0 ± 0.8</td>
<td>22, 19</td>
<td>0.75 ± 0.11</td>
</tr>
<tr>
<td>NaCl, 5 mM</td>
<td>NH(_4)Cl, 5 mM</td>
<td>-27.0 ± 0.8</td>
<td>22, 10</td>
<td>0.86 ± 0.11</td>
</tr>
<tr>
<td>KCl, 2 mM</td>
<td>K(_2) folate, 1 mM</td>
<td>-33.8 ± 0.9</td>
<td>10, 6</td>
<td>0.61 ± 0.06</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Repellents I</th>
<th>Test solution</th>
<th>( E_m ) (mV)</th>
<th>( N )</th>
<th>( I_{che} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>KCl, 5 mM</td>
<td>NaCl, 5 mM</td>
<td>-31.3 ± 0.8</td>
<td>50, 50</td>
<td>0.27 ± 0.13</td>
</tr>
<tr>
<td>KCl, 0.1 mM</td>
<td>Quinidine-HCl, 0.1 mM</td>
<td>-44.1 ± 1.3</td>
<td>7, 12</td>
<td>0.08 ± 0.04</td>
</tr>
</tbody>
</table>

Table 2. Net changes of \( E_m \) (\( \Delta E_m \)) when the solution around the cell was changed from control to test solution or vice versa. Values are averages of \( N \) measurements ± 1 S.E. The \( \Delta E_m \) was measured as the difference between the \( E_m \) of the cell in control solution just before the solution change and the \( E_m \) of the cell 6 minutes after the solution change, except when the solution was changed from quinidine to KCl (see Table 1 legend). The \( E_m \) changes were measured after a change of solution that should have simulated the diffusion gradients in the T-mazes. Gradients were not identical in the electrical recording and T-maze, and therefore the time course of \( \Delta E_m \) was not analyzed.

<table>
<thead>
<tr>
<th>Control solution</th>
<th>Test solution</th>
<th>( \Delta E_m ) change from control to test solution (mV)</th>
<th>( N )</th>
<th>( \Delta E_m ) change from test to control solution (mV)</th>
<th>( N )</th>
</tr>
</thead>
<tbody>
<tr>
<td>KCl, 5 mM</td>
<td>K acetate, 5 mM</td>
<td>-7.3 ± 0.7</td>
<td>14</td>
<td>6.9 ± 1.4</td>
<td>13</td>
</tr>
<tr>
<td>KCl, 5 mM</td>
<td>K lactate, 5 mM</td>
<td>-7.9 ± 0.7</td>
<td>13</td>
<td>6.2 ± 0.8</td>
<td>13</td>
</tr>
<tr>
<td>NaCl, 5 mM</td>
<td>Na acetate, 5 mM</td>
<td>-8.5 ± 1.2</td>
<td>10</td>
<td>8.3 ± 1.2</td>
<td>9</td>
</tr>
<tr>
<td>NaCl, 5 mM</td>
<td>NH(_4)Cl, 5 mM</td>
<td>-8.3 ± 2.5</td>
<td>6</td>
<td>12.7 ± 3.0</td>
<td>5</td>
</tr>
<tr>
<td>KCl, 2 mM</td>
<td>K(_2) folate, 1 mM</td>
<td>-12.4 ± 3.6</td>
<td>6</td>
<td>10.1 ± 2.2</td>
<td>6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Repellents I</th>
<th>Test solution</th>
<th>( \Delta E_m ) change from control to test solution (mV)</th>
<th>( N )</th>
<th>( \Delta E_m ) change from test to control solution (mV)</th>
<th>( N )</th>
</tr>
</thead>
<tbody>
<tr>
<td>KCl, 5 mM</td>
<td>NaCl, 5 mM</td>
<td>1.6 ± 0.6</td>
<td>3</td>
<td>-2.6 ± 0.9</td>
<td>3</td>
</tr>
<tr>
<td>KCl, 0.1 mM</td>
<td>Quinidine-HCl, 0.1 mM</td>
<td>14.8 ± 2.7</td>
<td>10</td>
<td>-13.7 ± 4.6</td>
<td>6</td>
</tr>
<tr>
<td>KCl, 1 mM</td>
<td>Acetic acid, 1 mM</td>
<td>21.9 ± 1.6</td>
<td>11</td>
<td>-16.9 ± 1.6</td>
<td>9</td>
</tr>
<tr>
<td>KCl, 5 mM</td>
<td>BaC(_2)Cl(_2), 2.5 mM</td>
<td>25.3 ± 3.5</td>
<td>6</td>
<td>-24.0 ± 3.0</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Repellent II</th>
<th>Test solution</th>
<th>( \Delta E_m ) change from control to test solution (mV)</th>
<th>( N )</th>
<th>( \Delta E_m ) change from test to control solution (mV)</th>
<th>( N )</th>
</tr>
</thead>
<tbody>
<tr>
<td>KCl, 1 mM</td>
<td>KOH, 1 mM</td>
<td>-16.8 ± 1.9</td>
<td>10</td>
<td>14.1 ± 1.9</td>
<td>11</td>
</tr>
<tr>
<td>NaCl, 2 mM</td>
<td>BaC(_2)Cl(_2), 1 mM</td>
<td>31.6 ± 1.5</td>
<td>7</td>
<td>-31.0 ± 1.4</td>
<td>6</td>
</tr>
</tbody>
</table>
Experimental Allergic Neuritis Induced by Sensitization with Galactocerebroside

**Abstract.** Thirteen of 31 rabbits immunized repeatedly with bovine brain galactocerebroside developed experimental allergic neuritis, manifested by flaccid paresis and hypesthesia of four limbs, 2 to 11 months after the initial inoculation. Electrophysiological studies revealed multifocal conduction block of peripheral nerves. Perivenular demyelinating lesions associated with phagocytic mononuclear cells occurred in spinal ganglia, roots, and less frequently in distal nerves.

Experimental allergic neuritis (EAN) and experimental allergic encephalomyelitis (EAE) are autoimmune, demyelinating diseases of the peripheral nervous system (PNS) and central nervous system (CNS); they are classically produced in animals by injection of homogenates of PNS or CNS tissue, respectively, with complete Freund's adjuvant (1). Experimental allergic neuritis can be induced by injection of P2 basic protein (BP) of peripheral nerve myelin. A peptide from myelin P2 BP is at least one of the neuritogenic determinants (2).

In EAE, the encephalitogen is CNS myelin BP (3, 4). Another major component of CNS and PNS myelin (5), galactocerebroside (β-β-galactotyrosyl ceramide) (GC), is a glycolipid hapten (6, 7) and binds specific antibodies in isolated myelin (7–9). Antiserum to GC binds GC in both central and peripheral myelin in sections of rat optic and sciatic nerve, as well as in CNS myelin of unfixed spinal cord cultures as demonstrated by indirect immunofluorescence or immunoperoxidase techniques (10). Galactocerebroside can serve as a cell-surface antigenic marker for oligodendrocytes in culture (10). Further, rabbit antiserum to GC demyelinates organotypic CNS cultures and inhibits myelination and sulfatide synthesis in immature CNS cultures (11–13). However, immunization with one or two injections of GC has not been encephalitogenic (6). We describe here the first successful production of EAN in rabbits by repeated immunization with GC. The distribution of demyelinating lesions seems to correspond to areas known to have a defective blood-nerve barrier (14, 15).

Thirty-one male New Zealand albino rabbits, weighing 2.3 to 2.7 kg, were sensitized with GC up to seven or eight times following one of three schedules (I to III) (Table 1). The immunizing inoculum contained 1 or 2 mg of bovine brain GC (lower spot cerebrosides, 98 percent with hydroxylated fatty acids) (Sigma) and bovine serum albumin or egg albumin (Sigma) as a carrier protein (5 mg per milligram of GC), in complete Freund's adjuvant (Difco) or, for booster injections in schedules II and III, without adjuvant. Galactocerebroside was checked for purity by thin-layer chromatography on silica gel G plate developed with chloroform, methanol, and water (65:25:4, by volume) using six standard sphingolipids and phosphoglycerides (16). Two spots were obtained. The major spot (> 99 percent) had an R F of 0.85 and the minor spot (< 1 percent) had an R F of 0.88, corresponding, respectively, to cerebrosides with longer (24 carbon) or shorter (16 or 18 carbon) length fatty acids. Analysis by thin-layer and gas chromatography after hydrolysis revealed that galactose was the only carbohydrate moiety detectable (> 99.9 percent). Sixteen control rabbits were similarly immunized but without GC. Since results with the three schedules did not vary significantly, we will describe the clinical, pathological, and serological results as grouped data.

Thirteen of 31 rabbits immunized with GC developed a neurological disorder, with onset ranging from day 44 to day 314 (135 ± 21 days; mean ± standard error) after the initial inoculation (Table 1). Rabbits were maintained for a maximum of 1 year. Subacute onset of weight loss, tremulousness, ataxia, flaccid paresis, and hypesthesia of four limbs were the main features of the clinical illness (Fig. 1A). Progress was sometimes rapid; quadriplegia and respiratory paresis were terminal events in three animals less than 2 weeks after onset of signs of illness. None of the control rabbits showed neurological abnormalities.

Animals immunized following schedule I were subjected to electrophysiological studies prior to terminal histological examination. The characteristic abnormality was multifocal conduction block (Fig. 1, E and F). In animals examined from 2 to 24 weeks after onset of weakness, motor conduction velocities were diffusely slowed (11 m/sec; normal is 50 m/sec), suggesting widespread peripheral nerve demyelination. These electrophysiological abnormalities were indistinguishable from those found in human multifocal demyelinating neuropathies such as Guillain-Barre syndrome (17). Twenty-three GC-immunized rabbits, including all 13 paralyzed rabbits at various clinical stages, and 12 control rabbits were killed at corresponding intervals between 1 month to 1 year after immuno-