of transport, provided the cell bodies are maintained in normal incubation medium, it follows that the energy supply within the axons on which transport depends is not affected by the altered ionic conditions.

The finding of a calcium requirement for axonal transport, localized at a site within the ganglia, suggested that calcium ions might serve as ionic links during the loading of proteins onto the transport system. To test the hypothesis that such calcium links might be involved during the transport process as well as during loading, the possible axonal transport of $^{45}Ca^{2+}$ was examined. Such transport was indicated 19 hours after a 1-hour exposure of ganglia to $^{45}Ca^{2+}$ by a pronounced buildup of the isotope proximal to ligatures placed on spinal nerves (Fig. 2A). The rate of $^{45}Ca^{2+}$ transport during 8- and 16-hour incubations at 18°C was similar to the fast transport rate of [^{3}H]protein, approximately 75 mm/day (*12*), as determined at the same temperature in the contralateral preparation (Fig. 2B).

Calcium movement along axons in association with mitochondria would be an expected observation since these organelles concentrate large quantities of calcium (*13*). However, studies have generally shown that mitochondria are transported at rates significantly slower than those observed for rapidly transported proteins (*14*). Calcium-binding proteins present in extracts of neural tissues (*15*) might also account for the axonal transport of calcium, but characteristics of the transport of these proteins have not yet been reported.

The present observations that calcium-free incubation conditions markedly depress the amount of radioactive protein transported from cell body to axon, and that exposure of the ganglia to calcium-free medium is essential for this effect to occur, indicate that calcium ions may be involved in the initiation of axonal transport. In addition, the transport of $^{45}Ca^{2+}$ along axons at a rate similar to that of rapidly transported proteins suggests a mechanism in which calcium ions couple the transported proteins to the transport system, a system that may involve the "sliding filaments" proposed in models of axonal transport (*5*).

RICHARD HAMMERSCHLAG
ANANT R. DRAVID
ARLENE Y. CHIU
Division of Neurosciences,
City of Hope National Medical Center,
Duarte, California 91010

References and Notes

1. R. J. Lasek, *Int. Rev. Neurobiol.* **13**, 289 (1970); W. O. McClure, *Adv. Pharmacol. Chemother.* **10**, 185 (1972); P. L. Jeffrey and L. Austin, *Prog. Neurobiol.* **2**, 207 (1973). For evidence of dendritic transport, see G. W. Kreutzberg, P. Schubert, L. Toth, E. Rieske, *Brain Res.* **62**, 399 (1973).
2. S. Ochs, *Science* **176**, 252 (1972).
3. ———, *Proc. Natl. Acad. Sci. U.S.A.* **68**, 1279 (1971).
4. F. O. Schmitt, in *Cellular Dynamics of the Neuron*, S. H. Barondes, Ed. (Academic Press, New York, 1969), p. 95; R. B. Wuerker and J. B. Kirkpatrick, *Int. Rev. Cytol.* **33**, 45 (1972); J. P. Paulson and W. O. McClure, *Brain Res.* **73**, 333 (1974). But, see also F. E. Samson [*J. Neurobiol.* **2**, 347 (1971)] for an example of one of several species that have no axonal neurofilaments but carry out axonal transport, and M. R. Byers [*Brain Res.* **75**, 97 (1974)] for autoradiographic evidence that microtubules may not be involved in axonal transport.
5. F. O. Schmitt, *Proc. Natl. Acad. Sci. U.S.A.* **60**, 1092 (1968); ——— and F. E. Samson, *Neurosci. Res. Program Bull.* **6**, 113 (1968); S. Ochs (*2*). Experimental support for a neural system analogous to that subserving muscle contraction is provided by the studies of an actomyosin-like complex, neurostenin, isolated from mammalian brain [S. Berl, S. Puszkin, W. J. Nicklas, *Science* **179**, 441 (1973)].
6. F. Fuchs, *Annu. Rev. Physiol.* **36**, 461 (1974).
7. A. R. Dravid and R. Hammerschlag, *J. Neurochem.*, in press.
8. P. Greengard, in *Methods of Enzymatic Analysis*, H. U. Bergmeyer, Ed. (Academic Press, New York, 1965), p. 551. Assay was modified by using 25 m*M* tris(hydroxymethyl)-aminomethane (tris) HCl buffer, *p*H 8.0, and a commercial preparation of hexokinase plus glucose-6-phosphate dehydrogenase (Sigma Chemical Co.).
9. S. Ochs, *Abstr. Commun. Soc. Neurosci.* **2**, 255 (1972); A. Edström, *J. Cell Biol.* **61**, 812 (1974).
10. Preparations incubated for 20 hours in either normal or calcium-free medium were fixed by immersion in an aldehyde solution, postfixed in OsO_4, dehydrated, and embedded in Epon-Araldite. ($CaCl_2$ was omitted from fixation and postfixation solutions.) Ultrathin sections were cut from both nerves and ganglia and viewed with a Hitachi HU 11B electron microscope. We thank J. E. Vaughn and J. A. Grieshaber for performing the ultrastructural examination and evaluation.
11. M. A. Khan and S. Ochs, *Trans. Am. Soc. Neurochem.* **3**, 93 (1972).
12. This transport rate for [^{3}H]protein of 75 mm/day at 18°C is comparable to that found in a similar in vitro preparation of frog sciatic nerve [A. Edström and H. Mattsson, *J. Neurochem.* **19**, 205 (1972)]. When corrections are made for temperature [A. Edström and M. Hanson, *Brain Res.* **58**, 345 (1973)] this rate is also comparable to the rates of axonal transport of protein observed in mammalian nerves.
13. A. L. Lehninger, *Biochem. J.* **55**, 119 (1970).
14. P. L. Jeffrey, K. A. James, A. D. Kidman, A. M. Richards, L. Austin, *J. Neurobiol.* **3**, 199 (1972); L. M. Partlow, C. D. Ross, R. Motwani, D. B. McDougal, *J. Gen. Physiol.* **60**, 388 (1972).
15. D. J. Wolff and F. L. Siegel, *J. Biol. Chem.* **247**, 4180 (1972); S. Alemà, P. Calissano, G. Rusca, A. Guiditta, *J. Neurochem.* **20**, 681 (1973).
16. We appreciate the skillful assistance of J. R. Slemmon. Supported by PHS grants NS-09885 and NS 09226.

5 December 1974

Development of Specific Sensory-Evoked Synaptic Networks in Fetal Mouse Cord-Brainstem Cultures

Abstract. *Neurites of nerve growth factor–enhanced fetal mouse dorsal root ganglion cells can not only establish characteristic sensory synaptic network functions in dorsal regions of attached spinal cord explants, but some of the neurites may grow through the cord tissue in these cultures and make similar functional synaptic connections with specific types of "target" neurons in localized zones within nearby medulla explants.*

In a recent study of fetal rodent spinal cord explants with attached sensory dorsal root ganglia (DRG's), we observed marked augmentation of characteristic sensory-evoked synaptic network discharges in dorsal cord regions following DRG hypertrophy induced by nerve growth factor (NGF) (*1*). The present report demonstrates, for the first time, that some of these NGF-enhanced DRG neurites may grow through the cord tissue and establish similar functional synaptic connections with specific types of "target" neurons in nearby brainstem explants. Earlier electrophysiologic studies showed that complex synaptic interactions can, indeed, develop between neurons in explants of fetal rodent spinal cord and brainstem tissues after growth of neurites across gaps of 0.5 to 1 mm (*2*), but no signs of selective synaptic connections between specific central nervous system (CNS) neurons were detected.

Spinal cord with attached DRG's and meningeal covering from 13- to 14-day fetal mice was cut into cross sections 0.5 to 1 mm thick (Fig. 1). Prior to explantation, a midline section of the spinal cord fragment was made from the central canal through the dorsal cord and meninges; in the younger fetuses only the meninges required cutting since dorsal closure of the cord was not yet complete. This ensured outgrowth of CNS neurites and glial cells, including DRG fibers which have passed through the cord, comparable to dorsal column axons. Previous studies showed that "peripheral-type" neurites (invested by Schwann cells) will not invade separate CNS explants (*3*), and functional bridging of neural explants has so far only been achieved through growth of "CNS-type"

neurites (*2*). Fetal mouse brainstem explants were carefully positioned near the dorsal edge of the cord cross sections, on collagen-coated cover glasses (Fig. 1). Tissues from various regions of the medulla and midbrain were presented to the cord explant, including complete cross sections through the medulla at the level of the cuneate and gracilis nuclei (Fig. 2). The cultures were incubated in Maximow depression-slide chambers at 34° to 35°C, in a serum–embryo extract medium which was replenished three times a week (*1–3*).

Introduction of NGF at 100 to 1000 biological units per milliliter (*1, 4*) to the nutrient medium (at explantation) led to vastly increased survival of DRG neurons concomitant with more abundant neuritic outgrowth from these cells. Massive DRG's containing many hundreds of neuron perikarya developed during the first week in vitro (Figs. 1 and 2), similar to the remarkable NGF-induced selective hypertrophy of DRG's and sympathetic ganglia demonstrated by Levi-Montalcini (*5*) and Levi-Montalcini and Cohen (*6*) in 7- to 10-day chick embryos in ovo. In addition to NGF-induced DRG hypertrophy, the associated spinal cord segment developed unusual dorsal enlargements apparently related to profuse arrays of axons entering from the dorsal roots (*1, 7*). Abundant growth of neurites (and glia) also occurred between the spinal cord and brainstem explants, often filling much of the gap between the apposed tissues (Figs. 1 and 2).

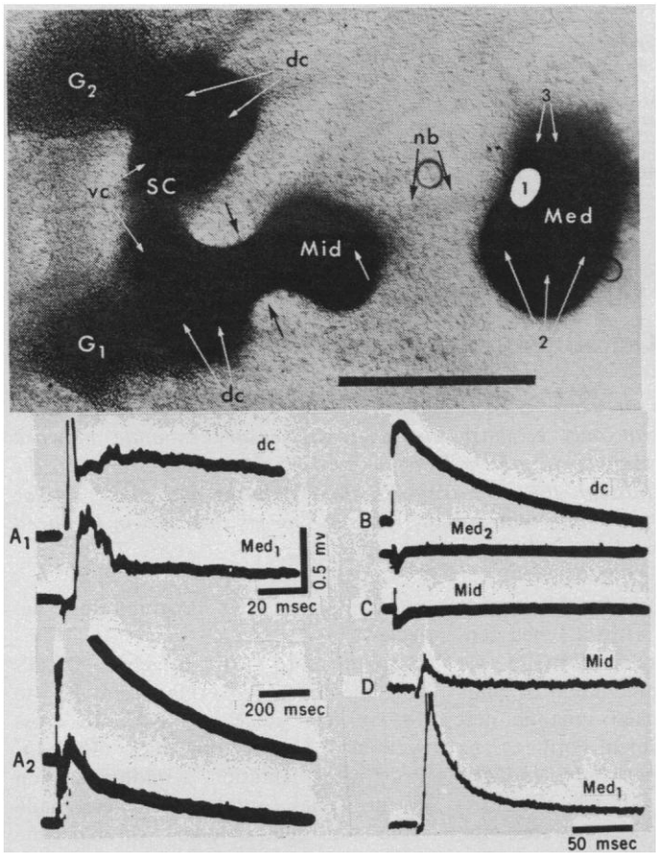

Fig. 1. Negative slow-wave potentials resembling PAD's (see text) evoked in spinal cord and brainstem explants by DRG stimuli (14-day fetal mouse tissues; 14 days in culture). Photomicrograph shows spinal cord cross section (*SC*) with attached DRG's (G_1 and G_3); dorsal edge of one half of cord is "fused" to midbrain (*Mid*) explant (black arrows); thinly spread array of neurites and glial cells have formed a bridge (*nb*) to the medulla (*Med*) explant (barely visible at this low magnification). Midline section of dorsal cord (see text) resulted in lateral displacement of dorsal horns and DRG's at either end of ventral cord (*vc*). Scale bar, 1 mm. (A_1) Simultaneous recordings of PAD's in dorsal cord (*dc*, lower left arrow) and medulla (*Med*, site 1) in response to single DRG stimulus (G_1). Note abrupt onset and long duration of these potentials, and also longer latency of medulla response. (A_2) Brief (100 per second) DRG volley (at smaller, near-threshold stimulus strength) elicits much larger and longer-lasting PAD's in both cord and medulla. (B) Large G_1 stimulus evokes only small positive slow-wave responses in other regions of medulla explant (Med_2); dorsal cord PAD is still large. Microelectrode mapping showed no signs of medulla PAD's in response to DRG stimuli, except in zone indicated in white around site 1, and none in entire midbrain (*Mid*) explant (for example, record C was taken at site of white arrow). (D) After addition of $10^{-5}M$ GABA, PAD in medula (Med_1 shows marked increase in amplitude and duration (as in Fig. 2B), and it can now also be evoked in adjacent region 3; polarity of midbrain response (*Mid*) has now become negative, although still relatively small. Time and amplitude calibrations and specification of recording sites apply to succeeding records, until otherwise noted; upward deflection indicates negativity at active recording electrode, and stimulus onset is indicated by first sharp pulse or break in baseline of each sweep.

Extracellular bioelectric recordings were made in these cultures (*8*) with Ag-AgCl electrodes via micropipettes (3- to 5-μm tips) filled with isotonic saline, using high input impedance preamplifiers and an oscilloscope (pass-band, 0.2 hertz to 10 khz). Electric stimuli (0.2 to 0.5 msec; up to 50 μa) were applied through pairs of similar pipettes with 10-μm tips. The culture was transferred from the Maximow slide to a larger closed chamber mounted on an inverted microscope. Electrodes were positioned in the tissue with micromanipulators and recordings were made in 0.5 ml of balanced salt solution (*p*H about 7.2) at 35°C. Application of focal stimuli to NGF-hypertrophied DRG's (or dorsal roots) evoked unusually prominent negative slow-wave responses restricted to dorsal regions of the spinal cord (*1*), arising abruptly after latencies of 2 to 3 msec, with amplitudes up to 2 mv, and lasting more than 500 msec (Fig. 1A). Marked temporal facilitation occurred after brief (100 per second) DRG volleys, at low stimulus strength (Fig. $1A_2$). Simultaneous recordings in ventral cord regions generally showed small positive or polyphasic slow-wave potentials and spike barrages, after latencies of 5 to 10 msec, similar to those observed in cultures without NGF exposure (*1*).

The temporal patterns of the dorsal cord potentials evoked by DRG stimuli in vitro contain components that are remarkably similar to those characteristic of the primary afferent depolarization (PAD) response in spinal cord in situ (*9*). The PAD-like component recorded in cord explants appears to be a field potential produced by summated excitatory postsynaptic potentials (EPSP's) generated after DRG activation of dorsal cord circuits. Bicuculline and picrotoxin ($10^{-5}M$) produce marked attenuation of the "PAD's" in cord explants as in situ (*1*), concomitant with onset of convulsive discharges, especially in ventral cord regions. On the other hand, the PAD responses in dorsal cord are maintained or even augmented in $10^{-3}M$ γ-aminobutyric acid (GABA) (Fig. 2B), in contrast to the rapid and sustained depression of synaptically mediated discharges in ventral cord regions, as well as long-latency discharges in dorsal cord (*1, 10*). These and related pharmacologic data suggest that the PAD's in cord-DRG explants

involve specific sensory-evoked synaptic circuits leading to GABA-mediated depolarization, possibly at DRG terminals as in situ (*1, 11*; however, see *12*), thereby mediating presynaptic inhibitory functions (*9*).

Similar PAD's have been detected in small regions of medulla explants connected to cord explants with NGF-hypertrophied DRG's. The medulla PAD's evoked by single DRG stimuli ranged up to 1 mv and arose after longer latencies (about 3 to 10 msec) (Figs. 1 and 2). The large amplitude of these PAD's indicates that relatively large numbers of DRG terminals probably make synaptic connections with target neurons in the medulla explants. [In cord-DRG cultures without added NGF, where only a few dozen DRG neurons may survive, dorsal cord PAD's are often much smaller than these medulla PAD's, in spite of the abundant dorsal cord neurons available for establishing sensory synaptic networks with ingrowing DRG neurites (*1*).] Introduction of $10^{-3}M$ GABA generally augmented the brainstem and dorsal cord PAD's (Figs. 1D and 2B), whereas various cord-evoked brainstem network discharges were seriously depressed, as were ventral cord responses. Moreover, in cases where a midbrain explant was positioned between the cord and medulla, prominent DRG-evoked PAD's were detected only in the latter explant, even when it was located more than 1 mm distal to the interposed midbrain tissue (Fig. 1A). Weak PAD's could, however, be detected in some midbrain regions, especially when medulla target neurons were relatively distant (Fig. 1, C and D) or absent. By use of this pharmacologic marker technique, we have been able to carefully map more than 12 DRG-cord-brainstem cultures, and in most cases PAD's were sharply localized to one or two small zones (about 100 to 300 μm) in each medulla explant. In six cultures where cross sections of the entire medulla at the level of the cuneate and gracilis nuclei were presented to the DRG-cord explant, with controlled orientation, prominent PAD's were evoked only in the dorsal medulla regions (Fig. 2), precisely where dorsal column sensory fibers normally terminate and lead to PAD's in situ (*9, 13*). Similar results were obtained in cultures where the medulla cross section was rotated 90° or 180° with respect to its orientation to the cord explant in Fig. 2.

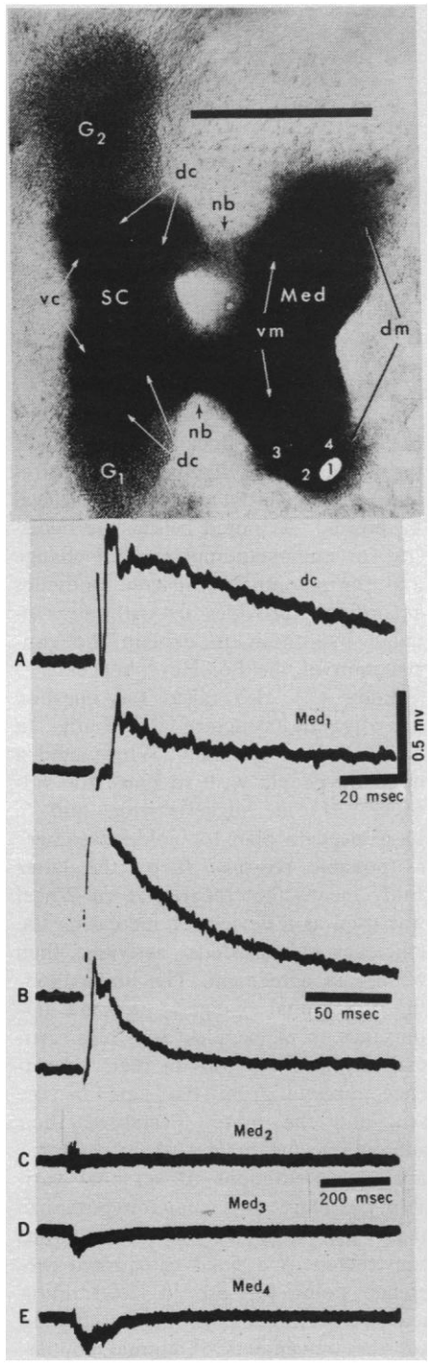

Fig. 2. PAD-like responses evoked in dorsal regions of spinal cord (*SC*) and medulla (*Med*) explants (complete cross sections) by DRG stimuli (14-day fetal mouse tissues; 14 days in culture). Medulla cross section is at level of cuneate and gracilis nuclei [dorsal closure has not yet occurred at this fetal stage so that the dorsal medulla tissues (*dm*) are laterally displaced]. Note bridges (*nb*) (neurites and glia) which have formed between dorsal edge of cord (*dc*) and ventral edge of medulla (*vm*) explants in two regions; DRG's (G_1 and G_2) are located more laterally in this explant (compare Fig. 1), further away from ventral cord (*vc*). Scale bar, 1 mm. (A) Simultaneous recordings of PAD's in dorsal cord (*dc*, lower left arrow) and dorsal medulla (*Med*, site 1) in response to single G_1 stimulus. (B) After addition of $10^{-3}M$ GABA, PAD's at this site in medulla (Med_1) and in dorsal cord are augmented (as in Fig. 1D). (C) Large G_1 stimulus evokes only spike burst at site 2 in medulla (Med_2) and small positive slow-wave responses at Med_3 and Med_4 (D and E). Systematic mapping of entire medulla explant showed no signs of PAD's in response to DRG stimuli except in small zone indicated in white around Med_1 (about 0.1 by 0.2 mm), even during GABA exposure. (Mapping was not attempted in this culture with stimuli to G_2.)

These in vitro experiments demonstrate that DRG neurites, after passing through spinal cord tissue, can grow across a homogeneous collagen-film substrate and, in mimicry of dorsal column fibers in situ, establish characteristic functional synaptic networks with programmed target neurons in brainstem explants, even in the presence of a variety of alternative CNS neurons with abundant synaptogenic receptor sites (see also *14*). Furthermore, although the initial neuritic and glial spinal cord outgrowth in relation to these nearby medulla explants was comparable to that extending toward nontarget CNS tissues, preliminary analyses in suitably arrayed cultures suggest that prominent fascicles of "dorsal column fibers" may become organized toward the target neuron zones in the medulla (*15*). The remarkable degree of characteristic pharmacologic sensitivity and regional specificity of these sensory-evoked spinal cord and brainstem networks provides the basis for a powerful new model system. With this system it should be possible to analyze the mechanisms underlying the formation and development of specific synaptic connections in the mammalian CNS (*16*), which are now accessible for more direct studies under rigidly controlled physicochemical conditions in culture. Experimental manipulation of these programmed neuronal interactions in vitro may also shed light on the more general problems of cell recognition leading to joint function between cells of different types (*17*).

STANLEY M. CRAIN
EDITH R. PETERSON
Departments of Neuroscience and *Physiology* and *Rose F. Kennedy Center for Research in Mental Retardation and Human Development, Albert Einstein College of Medicine, Bronx, New York 10461*

References and Notes

1. S. M. Crain and E. R. Peterson, *Brain Res.* **79**, 145 (1974); see also *Science* **141**, 427 (1963).
2. ———, M. B. Bornstein, in *Growth of the Nervous System*, G. E. W. Wolstenholme and M. O'Connor, Eds. (Churchill, London, 1968), pp. 13–31; S. M. Crain and M. B. Bronstein, *Science* **176**, 182 (1972); S. M. Crain, in *Studies on the Development of Behavior and the Nervous System*, G. Gottlieb, Ed. (Academic Press, New York, 1974), vol. 2, pp. 69–114; in *'Simple' Nervous Systems*, P. N. R. Usherwoood and D. R. Newth, Eds. (Arnold, London, 1975), pp. 67–117.
3. E. R. Peterson, S. M. Crain, M. R. Murray, *Z. Zellforsch. Mikrosk. Anat.* **66**, 130 (1965);
4. R. P. Bunge and P. Wood, *Brain Res.* **57**, 261 (1973).
5. Purified NGF (7*S* form) was kindly provided by E. M. Shoot [S. Varon, J. Nomura, E. M. Shooter, *Biochemistry* **7**, 1296 (1968)], and our assay with DRG cultures (*5*, *6*) indicated a potency of about 1 biological unit per 10^8 g.
6. R. Levi-Montalcini, in *Symposium on the Chemical Basis of Development*, W. D. McElroy and B. Glass, Eds. (Johns Hopkins Press, Baltimore, 1958), pp. 646–664.
7. —— and S. Cohen, *Ann. N.Y. Acad. Sci.* **85**, 324 (1960).
8. E. R. Peterson, E. B. Masurovsky, S. M. Crain, *J. Cell Biol.* **63**, 265a (1974); in preparation.
9. S. M. Crain, in *Methods in Physiological Psychology*, R. F. Thompson and M. M. Patterson, Eds. (Academic Press, New York, 1973), vol. 1, pp. 39–75.
10. J. C. Eccles, *The Physiology of Synapses* (Academic Press, New York, 1964); P. D. Wall, *Prog. Brain Res.* **12**, 92 (1964).
11. S. M. Crain, in *Drugs and the Developing Brain*, A. Vernadakis and N. Weiner, Eds. (Plenum, New York, 1974), pp. 29–57.
12. J. L. Barker and R. A. Nicoll, *Science* **176**, 1043 (1972); *J. Physiol. (Lond.)* **228**, 259 (197 J. M. Benoist, J. M. Besson, J. R. Boissier, *Brain Res.* **71**, 172 (1974): R. A. Davidoff, *Science* **175**, 331 (197 *Exp. Neurol.* **35**, 179 (1972).
13. D. R. Curtis, A. W. Duggan, D. Felix, G. A. R. Johnston, *Brain Res.* **32**, 69 (1971).
14. P. Andersen, J. C. Eccles, R. F. Schmidt, T. Yokota, *J. Neurophysiol.* **27**, 78 (1964); N. Davidson and C. A. P. Southwick, *J. Physiol. (Lond.)* **219**, 689 (1971).
15. M. J. Olson and R. P. Bunge, *Brain Res.* **59**, 19 (1973).
16. S. M. Crain and E. R. Peterson, in preparation.
17. R. W. Sperry, in *Organogenesis*, R. L. DeHaan and H. Ursprung, Eds. (Holt, Rinehart and Winston, New York, 1965), pp. 161–186.
18. G. B. Kolata, *Science* **186**, 1013 (1974).

Supported by grants from the National Institute of Neurological Diseases and Stroke (NS-06545 and NS-08770) and from the Alfred P. Sloan Foundation, and by a Kennedy Scholar Award to S.M.C. The cultures were prepared in the tissue culture laboratory at the Rose F. Kennedy Center (supported in part by NINDS grant NS-06735 to M. Bornstein). Thanks are due to B. Crain for skillful technical assistance in carrying out the electrophysiologic experiments.

13 November 1974; revised 24 December 1974 ■

Big Horn Medicine Wheel: Why Was It Built?

Eddy (*1*, p. 1042) offers two hypotheses to explain how the Big Horn Wheel could have been built: First, the construction can be viewed as the next step in the expansion of the indigenous astronomical system; second, the technique of astronomically aligning cairns could have been learned from Pueblo people to the south.

As the author of the paper to which Eddy refers for support of his second hypothesis (*2*), I think that his *first* suggestion is more plausible; that is, the construction followed "naturally" from the astronomical knowledge already possessed by the northern plains people. In other words, granted that the builders of the Big Horn Wheel were making celestial observations and had the technical skill necessary to incorporate these observations into the construction at the time the Wheel was built, the structure is one way of permanently recording these observances for year-to-year use (*3*). A major problem, of course, is that, because of precession, the three stellar alignments (Aldebaran, Rigel, and Sirius) would be inaccurate and therefore useless within a few hundred years after they had been set; solar alignments would remain accurate. The main point, however, is that, in this case, no diffusion of either astronomical knowledge or the techniques of aligning architectural features to celestial rise-set points need be posited. As noted below, the necessity for understanding seasonal change and for planning subsistence activities accordingly provides us with an adequate hypothesis to explain the construction of the Big Horn Wheel.

Eddy (*1*) also raises the question of why the structure was built. In answer to the question "Why would a nomadic people wish to mark the solstice?" (*1*), he suggests ritual and "a basic need to plan for colder weather" as possible reasons. If by the latter Eddy means that the Big Horn Wheel was used as a device for increasing the efficiency of subsistence activities, then we are in agreement. The understanding of seasonal change is of prime importance to all peoples; it is most crucial to those who obtain their subsistence *directly* from the land or the sea, and the more specialized their adaptation, the greater their need for accurate predictions of seasonal variability. Thus one can hypothesize that the Big Horn Wheel was constructed as a fixed calendrical reference point for use in determining seasonal changes and for predicting (i) the movements of animal populations upon which subsistence partially depended (this may account for the possible lunar count as represented in the 28 spokes of the Wheel, although, if these spokes do represent a lunar count, then one should reasonably expect to find lunar alignments of the cairns; Eddy does not indicate the presence of any lunar alignment), and (ii) the availability of important plant foods at various locales within the group's econiche. Furthermore, as Lowie (*4*), among others, has pointed out, many Plains groups, particularly in the Historic period, relied on agriculture as a significant part of their subsistence base; thus the Big Horn Wheel may have functioned, in part, as a calendrical device in the implementation of the agricultural cycle. [Another possible function, suggested by Kehoe (*5*), is that medicine wheels were used to mark the graves or places of death of important war chiefs and medicine men.]

Indeed, the lack of a specific, testable hypothesis to explain the adaptive functions of the Big Horn Wheel is the major weakness in Eddy's article; this shortcoming is also evident in much of the current archaeoastronomical research (*3*). Yet it is clear that, among most cultures, astronomical observations fundamentally serve in the planning and execution of subsistence activities (*6*). Therefore, the rituals usually associated with the observations (*6*) can be understood to be part of this adaptation (*7*). Moreover, it is precisely at the point when such ritual, for whatever reason, is diverted from its original adaptive function that the greatest threats to the survival of the system are seen to arise (*3*).

One final point needs to be made. If one argues, as I have, that the Big Horn Wheel served this adaptive function, then it is necessary to demonstrate that the energy expenditure (in calories) required to build the structure resulted in increased energy production: energy production *after* construction must exceed energy production *before* construction. There must be a marked increase in the efficiency of subsistence techniques in order for the construction to be worthwhile. If not, then that part of the system, predictably, should fall into disuse. Perhaps this is what happened in the case of the Big Horn Wheel; it did not function as expected. In addition, as the specific stellar alignments became unreliable, the entire system was eventu-