When gradients in excess of the physiological values are imposed on the xylem the conductivity decreases markedly (*2*); the shutdown is probably due to closure of bordered pits.

R. C. PLUMB
W. B. BRIDGMAN

References

1. M. H. Zimmerman and C. L. Brown, *Trees: Structure and Function* (Springer-Verlag, New York, 1971), p. 206.
2. B. Huber and W. Merz, *Planta* **51**, 645 (1958).

15 January 1973

The report by Plumb and Bridgman (*1*) appears to be inconsistent with the quantitative physiochemical basis for constructing such a theory (*2*). We would like to make two points of criticism.

1) The authors "suggest that the xylem conduit contains a filamentous gel-like structure having a concentration gradient sufficient to support the column without pressure gradients." There is no evidence that the hairlike gel structures the authors refer to extend across the lumen. The volume of the fibrils would have to be a small fraction of the flowing liquid in order not to impede the flow by frictional resistance. "The xylem of a tree at 100 m of height would contain about 10 percent of this gel," or about 10 g of dry gel per 100 ml of total water in the xylem. These fibrils will be in essential equilibrium with the water flowing past them and have a water content of about 90 percent (in order to have a water potential of about 10 atm). This would impede the water flows, which have been observed at 75 cm/min. Lower gel concentrations in dry matter per total volume of water would not provide the activity gradient required by the authors' hypothesis.

2) They suggest that, "the transport of water is hydrodynamic and not diffusion controlled." Experiments have shown that the actual flow rates are several orders of magnitude too large to be accounted for by diffusion. The authors then propose a mechanism of a "dynamic balance of the gravitational potential with an activity gradient," and this statement appears to contradict the one above. First, if there is an activity gradient, the flow must proceed because there is a concentration gradient (induced by temperature or pressure gradients) which must be relaxed by diffusional processes. Second, the thermodynamic description of a system involving a mixture in a uniform gravitational field (we assume that a 100-m change in elevation will not significantly change the local acceleration of gravity) with two phases present (a water phase and a gel phase) is controlled by the total potential for mass flow between the phases (*3*). Therefore, the activity defined by the authors is related to the adsorption and desorption of water by the gel at a particular elevation in the tree. This steady state (or equilibrium) does not involve the motion of the fluid through the xylem.

These points, which can be discussed in more detail, lead us to reject the hypothesis proposed by these authors.

J. LEVITT
Division of Biological Sciences, University of Missouri, Columbia 65201

T. S. STORVICK
College of Engineering, University of Missouri

References

1. R. C. Plumb and W. B. Bridgman, *Science* **176**, 1129 (1972).
2. A. T. J. Haywood, *Amer. Sci.* **59**, 434 (1971).
3. A. E. Guggenheim, *Thermodynamics* (North-Holland, Amsterdam, 1967), chap. 9.

3 July 1972; revised 20 November 1972

A reasonably complete discussion of the thermodynamics of columns of liquids bearing a constrained chemical activity gradient appears in the *Journal of Physical Chemistry* (*1*). We do not feel that the abridged discussion in *Science* (*2*) constitutes an adequate basis for a discussion of the validity of the thermodynamic analysis of the columns.

It is possible that the alternative theory we have proposed may be shown to be unrealistic and unacceptable on the grounds that the substance imposing the chemical activity gradient would introduce too high an impedance to flow. However, cell wall and cortical gel effects on water structure are not well understood at this time. It has been established by microscopic observation that gels and sols near cell walls play a role in cytoplasmic streaming in plants (*3*) and in amoeba locomotion (*4*). The physical chemistry of the mechanisms by which these motile systems operate is not known. Until these phenomena are better understood we prefer to withhold judgment on an argument which assumes a particular concentration, volume, and mode of action for the gel structure. Some recent nuclear magnetic resonance spectroscopic measurements (*5*) are pertinent. They indicate that agar (the major component of which is agarose, a polysaccharide similar to that which we proposed) imprints a structure on all the water in a gel, not just on the water molecules adjoining the macromolecular chains.

R. C. PLUMB
W. B. BRIDGMAN

References

1. R. C. Plumb and W. B. Bridgman, *J. Phys. Chem.* **76**, 1637 (1972).
2. ———, *Science* **176**, 1129 (1972).
3. T. Hayoshi, in *Primitive Motile Systems in Cell Biology*, R. D. Allen and N. Kamiya, Eds. (Academic Press, New York, 1964), pp. 19–29.
4. D. Marsland, in *ibid.*, pp. 173–189.
5. M. S. Micciancio-Giammarinaro, S. M. Micciancio, M. B. Palma-Vitterelli, M. U. Palma, M. L. Marino, *Ann. N.Y. Acad. Sci.*, in press.

22 January 1973

On Habituation in the Cochlear Nucleus

Humphrey and Buchwald (*1*) recorded neural response decrements in the cochlear nucleus of decerebrate cats during a series of tonal or white noise stimulations. These decrements were interpreted as evidence of neuronal habituation. Simplified mammalian preparations that exhibit true habituation would aid in understanding the plasticity of the mammalian central nervous system (CNS). Neural plasticity that can be shown to develop under controlled procedures with a time course of minutes or tens of minutes may form the basis for explanations of the information storage capabilities of the CNS. For these reasons, we wish to question several methodological points in Humphrey and Buchwald's report.

We contest the intended implications in their statement, "Thus the development of cochlear nucleus response decrements in the absence of changes in the microphonic potential indicated that receptor adaptation was not responsible for the phenomenon." On the contrary, the cochlear microphonic (CM) potential recorded from the round window (or anywhere near the cochlea) in a healthy preparation will not diminish in amplitude under continuous stimulation (*2*, *3*). The cochlear microphonic potential is literally a microphonic potential, and reflects the transformation of the mechanical disturbance at the hair-

bearing end of the cochlear hair cells into electrical potentials that mimic the waveform input to the cochlea. A recording of the microphonic potential cannot directly reflect changes in receptor responses (*4–6*). For example, the CM is present in both a normal cochlea and one whose eighth nerve has been sectioned, but the neural component is absent when the nerve is sectioned (*5*). Tetrodotoxin abolishes the N1 component of the eighth nerve response leaving the CM unaffected (*6*). The CM, unlike an action potential, is resistant to anesthesia and death of the animal (*2*, p. 311).

Apparently, Humphrey and Buchwald felt that they had sufficient control conditions for receptor adaptation, and therefore they do not mention that this phenomenon could explain the response decrements that occur *during* stimulus presentation. Adaptation [which can be readily seen in recordings from primary afferent neurons (*7*)] is undoubtedly occurring within the time courses of the stimuli presented. A "significant" response decrement that appeared after 45 to 50 presentations, with controls for the amount of concurrent adaptation, and that required a 10-minute rest period for recovery, would imply something akin to habituation. But, significant decrements occurred in only 54 percent of all the habituation series (or 82 percent of those series analyzed, as the authors report). As there is no control for adaptation, it is no longer clear with what response level the response decrements ought to be compared. All response levels shown (even at the end of "habituation") are far above baseline. With the confusion over what response levels should be compared it is unfortunate (i) that the authors did not, as a routine control, present an enduring stimulus of several minutes, in order to get some notion of how much of the response decrement might reflect diminished firing in the primary afferent neurons, and hence probably be a measure of receptor adaptation, without having to postulate additional mechanisms; and (ii) that successive time plots of the course of recovery were not presented.

It is also unclear whether at least some of their stimuli—presented at 2 dyne/cm^2—were not intense enough to exceed the interaural air or bone conduction levels of a cat at the frequencies of sound used. This could possibly be a factor because it has been demonstrated (*8*) that relatively small amounts (0.015 to 0.002 dyne/cm^2) of contralateral stimulation can inhibit spontaneous activity of neurons in the cochlear nucleus of the chinchilla.

Had more stringent controls been used, and response decrements to repeated acoustic stimuli still demonstrated in the cochlear nucleus, then the evidence would point more convincingly to central habituation factors. The data as presented can most easily be interpreted as reflecting primarily receptor adaptation, and this is not to be confused with the habituation that has been found in the CNS of both mammals (*9*) and invertebrates (*10*).

A. Barnebey
E. C. Carterette
Perceptual Systems Laboratory, Department of Psychology, University of California, Los Angeles 90024

References

1. G. L. Humphrey and J. S. Buchwald, *Science* **175**, 1488 (1972).
2. S. S. Stevens and H. Davis, *Hearing* (Wiley, New York, 1938).
3. "The diminution is a function of the intensity of the stimulation and also of the length of time which it continues. With submaximal stimulation there is no suggestion of fatigue—that is to say no diminution of activity from prolonged stimulation" (*2*, p. 325).
4. H. Davis, *Physiol. Rev.* **37**, 1 (1957); E. G. Wever, *ibid.* **46**, 102 (1966); A. D. Grinnell, *Amer. Rev. Physiol.* **31**, 345 (1969); A. J. Derbyshire and H. Davis, *Amer. J. Physiol.* **113**, 476 (1935).
5. T. F. Weiss, W. T. Peake, H. S. Sohmer, *J. Acoust. Soc. Amer.* **50**, 587 (1971).
6. P. E. Stopp, in *Sensorineural Hearing Loss*, G. E. W. Wolstenholme and J. Knight, Eds. (Churchill, London, 1970), p. 225.
7. N. Y.-S. Kiang, *Discharge Patterns of Single Fibers in the Cat's Auditory Nerve* (M.I.T. Press, Cambridge, Mass., 1965).
8. T. E. Mast, *J. Neurophysiol.* **33**, 108 (1970).
9. W. A. Spencer, R. F. Thompson, D. R. Neilson, Jr., *ibid.* **29**, 221 (1966).
10. F. B. Krasne, *J. Exp. Biol.* **50**, 29 (1969).

19 April 1972

Barnebey and Carterette suggest that the cochlear microphonic (CM) is not an adequate control for receptor adaptation, because it persists after surgical or pharmacological damage to the eighth nerve, and it is resistant to anesthesia and death of the organism. Because the CM does persist after degeneration of the eighth nerve (*1*), its origin is clearly in the cochlea, and not in the primary afferent fibers of the nerve. Although the CM is somewhat more resistant to anesthesia than is the acoustic nerve potential, it shows marked decrements during anesthesia or anoxia, and completely disappears when the organism dies (*2*). Experimental data closely links the CM with excitation of the cochlear hair cells, the receptors of the auditory pathway. When isotonic KCl or a polarizing direct current is passed across the basilar membrane in one turn of the cochlea (to block local excitation of the hair cells), the CM is selectively abolished or depressed in that cochlear turn; control infusions of saline across the same area of the basilar membrane have no effect on the CM (*3*). No CM can be recorded during acoustic stimulation of congenitally deaf animals (for example, albinotic cats, waltzing guinea pigs) that histologically have an absence of cochlear hair cells, although the cochlear endolymphatic potential of such animals is of normal amplitude (*4*). As a result of exposures to loud noise (for example, jet-engine noise), the CM recorded at the round window of the ear shows a significant, permanent reduction in responsiveness, the magnitude of which is directly related to the number of cochlear hair cells destroyed by the excessive acoustic stimulation (*5*). During excessive acoustic stimulation that does not produce permanent hair cell damage, recordings of the CM at the round window show a reversible reduction in responsiveness; subsequent recovery of the CM occurs over a period from seconds to minutes, depending upon the intensity of stimulation (*6*). Below the level of acoustic overloading, the CM is stable as long as the physiological condition of the animal is unchanged, and the middle ear muscles are quiescent (*6*). To summarize, the cochlear hair cells are the receptor cells of the auditory pathway, the CM is primarily a representation of hair cell excitation, and recordings at the round window can reveal changes in CM responsiveness due to excessive stimulation. Thus, we feel that round window recordings of the CM in our experiments provide an acceptable control for receptor adaptation.

A problem separate from that of receptor adaptation is the possible change in acoustic nerve transmission as a function of repeated stimulation—a distinction that Barnebey and Carterette do not make. The response of some acoustic nerve fibers may decrease during continuous, prolonged (for example, 13 minutes) stimulation (*7*). However, decreases in unit responses in the cochlear nucleus and trapezoid body, which also develop during *continuous*, prolonged tone stimulation, have been associated with local changes in neuronal excitability, rather than with a decreased input from the acoustic nerve (*8*). In our experiments, we used a qualitatively different kind of acoustic stimulation, that is, a *discontinuous*

acoustic stimulus. During repeated presentations of a discontinuous acoustic stimulus at one stimulus per second for 1 hour or one stimulus per 3 seconds for 5 hours, there was no change in acoustic nerve potentials (*9, 10*). After even more prolonged discontinuous stimulation, decrements in acoustic nerve potentials appeared, but were subsequently abolished by midline section of the olivocochlear bundle (*9*), or by barbiturate dosages sufficient to depress the olivocochlear system (*10*). Thus, these decrements were interpreted as being a reflection of peripheral inhibition imposed by the olivocochlear system, and not as being a reflection of decreased acoustic fiber transmission per se. In our experiments, discontinuous acoustic stimuli were presented more slowly (at 5-second intervals) than in the other studies (*9, 10*), the decrements in the cochlear nucleus response developed more rapidly (within 4 minutes) than did the changes in acoustic nerve potentials (*9, 10*), and the decrements in the cochlear nucleus responses were independent of olivocochlear efferent influences (*11, 12*). Thus, our data do not support the idea that changes in acoustic nerve transmission, or peripheral effects of the olivocochlear system, are primary causes of decrements in cochlear nucleus response. Furthermore, in other experiments with the same acoustic stimulus parameters, we have found that shock to the paw of an animal restores (dishabituates) previously decremented acoustic responses to control or near-control levels (*13*), an effect which could not occur if the response decrements were simply reflecting decreased acoustic nerve transmission.

The preceding data indicate that decrements in cochlear nucleus responses, which result from acoustic stimuli repeated at 5-second intervals, are not passive reflections of decreased peripheral input but, rather, reflect centrally mediated changes in neural excitability. The control experiment suggested by Barnebey and Carterette, that is, presenting an enduring stimulus of several minutes to provide a measure of receptor adaptation, would not really provide us with information on receptor or primary afferent changes during a procedure utilizing discontinuous acoustic stimuli. We have reported the time course of recovery of cochlear nucleus response as occurring over a 10-minute period, with the most rapid phase of recovery in the first 5 minutes after stimulus cessation (*11–13*). This is comparable to the recovery periods of habituated spinal reflex responses after repeated cutaneous nerve stimulation (*14*).

Barnebey and Carterette incorrectly calculated that significant decrements occurred in only 54 percent of all habituation series. Forty-three habituation series were reported, 36 of which (84 percent) showed obvious decrements with computer analysis (Computer of Average Transients, Mnemotron); when 28 of these 43 series were subjected to statistical analyses, 82 percent of them showed significant response decrements (*11*). The phenomenon was, in our mind, clearly established, and we did not subject the remaining series to the same analysis. We have subsequently made a more extensive report of decrements in cochlear nucleus responses in a larger number of subjects (*12*).

In agreement with Mast's data on the chinchilla (*15*), we have found that contralateral tone stimulation produces inhibition of unit discharge in the ipsilateral dorsal cochlear nucleus, with no effect on the ipsilateral ventral cochlear nucleus (*12*). If physical spread to the opposite ear had occurred in our experiments, any resultant stimulation from the contralateral side back to the ipsilateral cochlear nucleus would then be confined to inhibition of the dorsal cochlear nucleus. It would not account for response decrements in the ventral cochlear nucleus, which developed with approximately the same time course as those in the dorsal cochlear nucleus (*12*). Moreover, inhibition of unit discharge in the dorsal nucleus, induced by contralateral stimulation, became progressively less with repeated contralateral stimulation (*12*). These data suggest that physical spread of the acoustic stimulus to the contralateral ear was not a factor in the development of decrements in the ipsilateral cochlear nucleus responses.

J. S. BUCHWALD
Departments of Physiology and *Psychiatry, Mental Retardation Center, University of California Medical Center, Los Angeles 90024*

G. L. HUMPHREY
Department of Physiology, University of Illinois Medical Center, Chicago 60612

References

1. T. F. Weiss, W. T. Peake, H. S. Sohmer, *J. Acoust. Soc. Amer.* **50**, 587 (1971).
2. D. H. Eldridge and J. D. Miller, *Annu. Rev. Physiol.* **33**, 281 (1971); I. C. Whitfield, *The Auditory Pathway* (Arnold, London, 1967), p. 26; E. G. Wever, *Physiol. Rev.* **46**, 102 (1966); T. Konishi, R. A. Butler, C. Fernandez, *J. Acoust. Soc. Amer.* **33**, 349 (1961); G. von Békésy, *ibid.* **22**, 576 (1950).
3. I. Tasaki and C. Fernandez, *J. Neurophysiol.* **15**, 497 (1952).
4. K. F. Brown, B. Gordon, C. R. Cazonius, *Nature* **228**, 1212 (1970); H. A. Howe, *Amer. J. Physiol.* **111**, 187 (1935); H. Davis, A. J. Derbyshire, M. H. Lurie, L. J. Saul, *ibid.* **107**, 311 (1934); M. H. Lurie, H. Davis, A. J. Derbyshire, *Ann. Otol. Rhinol. Laryngol.* **43**, 321 (1934).
5. I. E. Alexander and F. J. Githler, *J. Comp. Physiol. Psychol.* **45**, 381 (1952); *ibid.* **44**, 513 (1951); M. H. Lurie, H. Davis, J. E. Hawkins, *Laryngoscope* **54**, 375 (1944).
6. E. G. Wever, *Physiol. Rev.* **46**, 102 (1966); ——— and M. Lawrence, *J. Acoust. Soc. Amer.* **27**, 853 (1955); I. E. Alexander and F. J. Githler, *J. Comp. Physiol. Psychol.* **42**, 517 (1949).
7. N. Y.-S. Kiang, *Discharge Patterns of Single Fibers in the Cat's Auditory Nerve* (M.I.T. Press, Cambridge, Mass., 1965).
8. A. Starr, *J. Neurophysiol.* **28**, 850 (1965).
9. W. Buno, Jr., R. Vellute, P. Handler, E. Garcia Austt, *Physiol. Behav.* **1**, 23 (1966).
10. I. A. Altman, *Sechenov. Physiol. J. USSR* **46**, 617 (1960).
11. G. L. Humphrey and J. S. Buchwald, *Science* **175**, 1488 (1972).
12. J. S. Buchwald and G. L. Humphrey, *J. Neurophysiol.* **35**, 864 (1972).
13. ———, in *Progress in Physiological Psychology*, E. Stellar and J. Sprauge, Eds. (Academic Press, New York, in press), vol. 5; K. A. Brown and J. S. Buchwald, in preparation.
14. W. A. Spencer, R. F. Thompson, D. R. Neilson, Jr., *J. Neurophysiol.* **29**, 221 (1966).
15. T. E. Mast, *ibid.* **33**, 108 (1970).

8 December 1972

Silicon: Its Role in Vital Processes

The instructive paper by Carlisle (*1*) contains the observation that there had been no previous proof of a silicon metabolism role in "vital processes in animals or man." Vinogradov (*2*) cited data on the gastropod *Oncidium planatum*, showing that its liver contained 11.3 percent silica. Would not this be one kind of proof of a silicon role in vital processes, especially so since 10 percent of its weight consists of siliceous spicules?

PAUL TASCH
Wichita State University, Wichita, Kansas 67208

References

1. E. M. Carlisle, *Science* **178**, 619 (1972).
2. A. P. Vinogradov, *The Elementary Chemical Composition of Marine Organisms* [Sears Foundation for Marine Research Mem. No. 2 (Yale University, New Haven, Conn., 1953)], p. 295.

11 December 1972