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

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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

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Paul Tasch

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