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The main goal of my research at the University of Rochester during my year as Hunt Fellow was to determine the physical mechanisms of kidney and gallstone destruction by acoustic lithotripsy. I was fortunate in being able to collaborate with members of the Rochester Center for Biomedical Ultrasound such as Edwin Carstensen, Sally Child, Sheryl Gracewski, Stephen Burns, and Nimish Vakil. Dr. Vakil, a gastroenterologist and clinical gallstone lithotripsy expert, was instrumental in allowing me to have access to two clinical lithotripters in regular use at the Strong Memorial Hospital. In early studies of the destruction of model stone materials in the clinical lithotripters, we began to realize the importance of the material properties of the stone in determining the success of lithotripsy. A paper was submitted describing the importance of careful preparation of sample materials and the influence of stone properties on the mechanisms of destruction. These findings led us to seek experts who could help us determine the mechanical properties of gallstones. We were fortunate to enlist the aid of a materials scientist (Stephen Burns) and a geologist and specialist on crystal structure (Asish Basu). After an extensive experimental study of the microhardness of gallstones, we submitted another paper detailing some of our measurements. Much work is still ongoing to determine other properties such as the fracture toughness and dynamic critical fracture stress of gallstones; during 1989–1990 much of the groundwork (including construction of apparatus) was laid. Another fruitful line of investigation developed when we discovered that some of the gallstones we were using for in vitro lithotripsy experiments floated prior to the first shock wave application and sank afterward. A paper describing the gas content of human gallstones and its implications in favoring one mechanism over another in acoustic lithotripsy was submitted. During 1989–1990, aspects of the lithotripsy research were presented by my colleagues and me at various meetings.

During my year at Rochester, I designed and built prototypes of an inexpensive wide-bandwidth hydrophone for lithotripsy research. The resulting instrument represents a significant improvement over existing hydrophones commonly used by researchers to determine the acoustic field of a lithotripter. Currently, I am still refining the design of the piezoelectric copolymer that is the hydrophone's active element, and expect to submit soon for publication a complete description of the device. The basic design of the hydrophone was outlined in an abstract and accompanying presentation at the spring 1990 Acoustical Society meeting at Penn State.

Although the lithotripsy research was my main focus during my year as Hunt Fellow, I found the research environment at the University of Rochester so fertile that I participated in several other projects. I collaborated on an investigation of the properties of echo-contrast agents reported at meetings of cardiologists such as the American Heart Association. I also investigated the color of the stomach lining as an indicator of pathology, worked on improving the design of a device for removing bubbles from blood acoustically, and did preliminary experiments to determine the role of acoustic cavitation in the ablation of arterial plaque. Perhaps most important, however, was the long-term collaboration that I have established with the researchers at the University of Rochester. I accepted a nontenure appointment as an Adjunct Professor of Electrical Engineering at Rochester to allow me to spend my summers and sabbatical years from Swarthmore College at the University of Rochester. I look forward to years of productive research on topics of biomedical acoustics. None of this would have been possible without the help of the F. V. Hunt Fellowship, and I remain most grateful for the opportunities it afforded me.

The destruction of gallstones and the influence of stone properties on the mechanisms of gallstone destruction by shock waves was presented at the spring 1990 Acoustical Society meeting at Penn State.

2. T. D. Rossing, Teaching the Science of Sound (Rossing, De Kalb, IL, 1982); write to T. Rossing, Physics Department, Northern Illinois University, De Kalb, IL 60115.

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15E. Carr Everbach, "An inexpensive wide-bandwidth hydrophone for litho-

min and saccharide contrast agents,” Feinstein Meeting on Echocontrast, Chicago, IL (1990).


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Previously, during my Ph.D. thesis, research with Dr. Peter Dallos (Northwestern University, Evanston, IL), I recorded intracellularly from cells in the organ of Corti and attempted to determine the origins of sound-induced potential changes recordable in the supporting cells of the organ. Findings demonstrated the existence of potentials that ap-
peared to emanate from the supporting cells themselves and were reminiscent of slow electrical potential changes that occur in central nervous system (CNS) glial cells [E. C. Oesterle and P. Dallos, “Intracellular recordings from supporting cells in the guinea pig cochlea: AC potentials,” J. Acoust. Soc. Am. 86, 1013–1032 (1989); E. C. Oesterle and P. Dallos, “Intracellular recordings from supporting cells in the guinea pig cochlea: DC potentials,” J. Neurophysiology 64, 617–636 (1990)]. The results were consistent with the hypothesis that some types of inner-ear supporting cells may function similarly to the glial cells of the CNS—operating to maintain the homeostatic environment surrounding the hair cells and eighth-nerve endings by participating in the uptake and inactivation of chemical transmitters from the extracellu-
lar spaces and/or buffering levels of extracellular ions.

The F. V. Hunt postdoctoral fellowship gave me the opportunity to continue research in this area under the su-
pervision of Dr. Edwin W. Rubel (Dept. of Otolaryngology, University of Washington, Seattle, WA). Research explori-
g possible similarities between inner-ear supporting cells and the better studied neuroglia was conducted during the fellowship period, June 1988 to August 1989. More specifically, the hypothesis that proteins known to be present in CNS glia are also present in inner-ear supporting cells was investigated.

Antibody labeling techniques were used to look for the existence of two classes of intermediate filaments in inner-
ear supporting cells which are uniquely associated with glial cells in the CNS. Vimentin and glial fibrillar acidic protein (GFAP) are intermediate filament proteins present normal-
ly in many types of neuroglia. During glial reactivity (when glial cells form scars in response to neural or sensory cell damage) dramatic increases in intermediate filament content are observed, and increased GFAP and vimentin immuno-

staining are evident. In response to photoreceptor degeneration, retinal Muller cells express GFAP immunoactivity even though they do not express this protein normally. Since auditory support cells form phalangeal scars in response to hair-cell degeneration, it appeared plau-
sible that GFAP and vimentin immunoactivity might be expressed in auditory support cells in response to hair-cell injury.

Studies of GFAP and vimentin expression in normal and experimentally damaged mammalian cochleas were completed. Findings were presented at several meetings [Oesterle et al., “Distribution of vimentin in the guinea-pig cochlea,” Soc. Neurosci. Abstr. 14, 488 (1988); Y. Raphael and E. C. Oesterle, “The distribution of intermediate fila-

Findings regarding the normal organ of Corti in adoles-
cent and adult guinea pigs indicated that vimentin is present in two types of supporting cells, Deiters’ cells and inner pil-
lar cells. Our observations also suggest the existence of a differential distribution of vimentin immunoreactivity among Deiters’ cells in the mature guinea-pig organ of Corti and raise questions regarding the possible existence of sub-
classes of Deiters’ cells. GFAP immunoreactivity was not detected in any supporting-cell type in the normal organ of Corti. Cochlear hair cells were unlabeled for either GFAP or vimentin. The expression of vimentin and GFAP in end or-
gans, where hair-cell degeneration was experimentally in-
duced, was not observed in phalangeal scars replacing lost hair cells. These findings concerning the absence (or pres-
ence) of GFAP and vimentin in the auditory support cells suggest that auditory support cells do not use the same cyto-
skeletal proteins as CNS neuroglia, thereby contributing to an understanding of the roles inner-ear support cells play in end-organ functioning.

The F. V. Hunt fellowship provided the opportunity to conduct this research, and for this I am most grateful. In answering the experimental question, I acquired expertise with anatomical and immunocytochemical techniques and was exposed to a new research environment. I am presently applying the techniques learned during the fellowship year in my current studies of regeneration in the inner ear. I would like to offer a big “thank you” to the Hunt family and the Acoustical Society of America for making this possible.