differing in both intensity and spectral characteristics) suggest a general rule: If there is contextual evidence that a sound may be present at a given time, and if the peripheral units stimulated by a louder sound include those which would be stimulated by the anticipated fainter sound, then the fainter sound may be heard as present.

Auditory induction appears to be a quite useful perceptual phenomenon permitting a highly selective reinstatement of sounds which would otherwise be lost through masking. The listener can thus establish a simpler and more stable interpretation of his auditory environment than the intermittent extraneous sounds present in our noisy world would otherwise permit.

RICHARD M. WARREN
CHARLES J. OBUSEK
JOHN M. ACKROFF
*Department of Psychology, University of Wisconsin–Milwaukee, Milwaukee 53201*

**References and Notes**

1. R. M. Warren, *Science* **167**, 392 (1970); ——— and C. J. Obusek, *Percept. Psychophys.* **9**, 358 (1971).
2. G. A. Miller and J. C. R. Licklider, *J. Acoust. Soc. Amer.* **22**, 167 (1950); W. Thurlow, *Amer. J. Psychol.* **70**, 653 (1957); L. Elfner, *J. Acoust. Soc. Amer.* **49**, 447 (1971).
3. Listening was through matched TDH 49 headphones, and subjects controlled intensity by adjusting a visually shielded attenuator. Frequencies from separate oscillators were measured with the aid of quartz crystal time base, and the intensities were measured with an artificial ear connected to a Brüel and Kjaer model 2204 sound level meter. Timing was accomplished with a Grason-Stadler model 829E electronic switch set at a rise/decay time of 5 msec (which eliminated audible clicks).
4. The threshold of audibility in this and all other experiments corresponded to the minimum intensity at which the fainter tone (adjusted by the listener) could be heard while alternating with the fixed louder sound at the rate employed for measuring AI.
5. The same tone generator was used for the masked and the masking sounds when both were at 1000 hz to provide exact correspondence of frequency and phase. Hence, this measurement was equivalent to a just noticeable difference in intensity.
6. R. L. Wegel and C. E. Lane, *Phys. Rev.* **23**, 266 (1924).
7. Supported by NSF grant GB 26459 and by the University of Wisconsin–Milwaukee graduate school.

15 November 1971; revised 11 February 1972 ■

## Endothelial Projections

Smith *et al.* (*1*) describe endothelial projections which have been previously "overlooked." While not denying the presence of these structures in their photographs and the intriguing speculations which they have elicited, considerable caution is warranted lest Smith *et al.* also be guilty of having overlooked an important consideration. Their method described fixation of the pulmonary vessel *after* its removal; many artifacts can be introduced by loss of extension and distention of an elastic vessel such as this. Absence of distending pressure causes an increase in wall thickness of about 40 percent; an elastic vessel shrinks in length about 40 percent on removal which causes even further wall thickening and distortion of the organization of constituent elements (*2*). The endothelium is not spared, for others have shown a marked difference in its appearance when measures are taken to preserve distention and extension during fixation (*3*). The endothelium of distended arteries and veins is greatly thinned, and the published photographs of such vessels give little evidence for the presence of endothelial projections (*3*).

This is not to gainsay the findings of Smith *et al.* for the pulmonary artery; but it does seem premature to speculate about new structures seen at 14,000-fold magnification until attention is paid to the maintenance of gross functional architecture of the entire arterial wall.

HARVEY WOLINSKY
*Albert Einstein College of Medicine, Bronx, New York 10461*

**References**

1. U. Smith, J. W. Ryan, D. D. Michie, D. S. Smith, *Science* **173**, 925 (1971).
2. H. Wolinsky and S. Glagov, *Circ. Res.* **14**, 400 (1964).
3. D. C. Pease and W. J. Paule, *J. Ultrastruct. Res.* **3**, 469 (1960); J. A. Esterly and S. Glagov, *Amer. J. Pathol.* **43**, 619 (1963); J. A. G. Rhodin, *J. Ultrastruct. Res.* **18**, 181 (1967); *ibid.* **25**, 452 (1968).

15 September 1971

While there is no doubt that shrinkage and contraction occur in muscular arteries upon removal, we have been unable to correlate cellular contraction with the occurrence of endothelial projections. Indeed, transmission electron micrographs of the material that we used for scanning electron microscopy show areas of endothelial thickening [possibly due to contraction of the endothelial fibrillar material (*1*)]. However, the number of projections never appears large in thin sections and over the thickened portions is no greater than over the slender arms of the cells [figure 4 of Smith *et al.* (*2*)], making it unlikely that the projections result from gross "crinkling" of the vessel wall.

Furthermore, we have evidence of the existence of endothelial projections in pulmonary capillaries fixed in situ by perfusion at physiological pressures. These vessels appear to have little or no intrinsic capacity for contraction and are not surrounded by muscular or elastic elements.

Finally, once one has recognized the existence of endothelial projections, one finds them in a large number of published micrographs of vessels of a wide variety of sizes [for examples, see (*3*) and figure 4 of Esterly and Glagov (*4*), cited by Wolinsky].

The density of endothelial projections varies in different vessels. In some instances the degree of their proliferation suggests highly specialized functions, for example, in the pecten of the bird's eye (*5*) and in the rat Gasserian ganglion (*6*). The latter study, performed entirely by transmission electron microscopy, led the authors to postulate the existence of endothelial projections nearly 3 years ago.

UNA SMITH
*Howard Hughes Medical Institute, University of Miami, Miami, Florida 33152*

**References**

1. G. Majno, S. M. Shea, M. Leventhal, *J. Cell Biol.* **42**, 647 (1969).
2. U. Smith, J. W. Ryan, D. D. Michie, D. S. Smith, *Science* **173**, 925 (1971).
3. W. Bloom and D. W. Fawcett, *A Textbook of Histology* (Saunders, Philadelphia, 1968), pp. 364 and 370; K. R. Porter and M. Bonneville, *Fine Structure of Cells and Tissues* (Lea & Febiger, Philadelphia, ed. 3, 1968), p. 104.
4. J. A. Esterly and S. Glagov, *Amer. J. Pathol.* **43**, 619 (1963).
5. D. W. Fawcett, in *The Peripheral Blood Vessels*, J. L. Orbison and D. E. Smith, Eds. (Williams & Wilkins, Baltimore, 1963), p. 17.
6. G. Gabbiani and G. Majno, *Z. Zellforsch. Mikrosk. Anat.* **97**, 111 (1969).

26 November 1971; revised 5 January 1972