optic nerve of the goldfish. Indeed, Jacobsen and Gaze have expressed surprise that in neither the frog nor in the optic lobe of the fish are there any units with different types of function from those in the optic nerve. While the commissural fibers from which we record do behave in a manner which is apparently very similar to this one class of optic nerve unit, we can at least be sure in our experiments that we dealt with the activity of tectal cells and not with the terminals of the primary optic nerve fibers. It seems quite probable that many "off" units of the tectum come from this class of tectal cells too. Are these cells all given over then to merely spreading across the brain one kind of apparently crude visual coding which has already been carried out by the retina? Or is the similarity between the "off" units of the optic nerve and commissural or tectal "off" units coincidental and are the commissural fibers carrying, perhaps in subtle variations of frequency or in the spatial distribution of discharge between different cells, some form of higher coding involved in visual perception?

Behavioral studies that demonstrate the importance of the tectal commissure for interocular transfer indicate that intertectal exchange over this pathway is somehow involved in perceptual mechanisms related to fine pattern vision. Therefore, in spite of the apparent insensitivity of isolated commissural fibers to localized or patterned visual input under the conditions of our experiments, one cannot discount the possibility that the cells which give rise to the tectal commissure may be involved in something much more subtle than might be supposed from the simple responses to light levels described here.

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Active Transport of 5,5-Dimethyl-2,4-Oxazolidinedione

T. C. Butler (*1*), commenting on our recent report (*2*), has suggested an explanation other than active transport for the movement of 5,5-dimethyl-2,4-oxazolidinedione (DMO) across the intestinal wall. As he points out, Hogben *et al.* (*3*) have postulated that a narrow zone of fluid adjacent to the mucosal cell surface has a higher hydrogen ion concentration than the remainder of the bulk mucosal perfusing solution. Such a microlayer of fluid with a low *p*H would lead to a relatively high concentration of un-ionized DMO next to the mucosa and, subsequently, to augmented mucosa-to-serosa movement of DMO by means of nonionic diffusion. By this mechanism, values for the ratio of $[DMO]_{serosa}$ to $[DMO]_{mucosa}$ greater than 1 could be obtained without postulating active DMO transport.

While this is a reasonable possibility, we have evidence which strongly suggests that this is not the explanation for the net movement of DMO we reported (*2*). For example, it can be shown that the net movement of another weak acid, cholic acid, which has a pK_a value (6.4) nearly identical to that of DMO, is from the serosal to the mucosal solution in everted gut sacs prepared from the jejunum. The *p*H of the bulk serosal fluid becomes acid relative to the bulk mucosal solution during the incubation of these sacs (*2*, *4*). Hence, cholic acid, which can cross the intestinal wall via passive nonionic diffusion (*4*), moves from the serosal media to the mucosal media in response to the hydrogen ion gradient which is measurable between the two bulk solutions.

Thus, it seems highly improbable that a hypothetical *p*H gradient adjacent to the mucosal cell surface could account for net mucosal-to-serosal movement to DMO while, at the same time, a second weak acid with a nearly identical pK_a value moves in the opposite direction, from the serosal to the mucosal solution, as would be expected from the relative *p*H values of the bulk perfusing solutions. In point of fact, we consider such data to support even more strongly the concept that DMO is actively transported by the small intestine of the rat, since this substance is moving not only against an electrochemical gradient but, as these data would indicate, against a *p*H gradient.

Furthermore, the distribution of DMO transport activity down the length of the small bowel (*2*, Fig. 1) is remarkably similar to that of α-aminoisobutyric acid, which clearly is actively transported and the movement of which, theroretically, is independent of any *p*H gradient effect. In addition, the active transport of DMO appears to be stimulated by the presence of glucose in the perfusing media—a finding which has been described in association with the active transport of numerous other substances.

Although we recognize the difficulties of extrapolating from the small intestine to the muscle cell, it seems reasonable to question the validity of the DMO muscle *p*H method on the basis that this substance may be transported into or out of the cell. The apparent "close agreement" of the HCO_3^- and DMO methods for intracellular *p*H determination commented on by Butler cannot be used as an independent validation of the DMO method for at least two reasons: (i) There is considerable scatter in the intracellular *p*H values given by either method (varying from 6.85 to 7.10); and (ii) the validity of the HCO_3^- method has been seriously challenged, and this challenge has not been adequately answered (*5*).

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A Piston Extractor for the Hughes Press

Simplified forms of the Hughes press (*1*) have been described which permit rapid and efficient breakage of microbial cells (*2*). Such a press consists essentially of a cylinder block in which the cells are placed and then frozen with dry ice. A piston is inserted into the cylinder, which is then mounted on a second block containing a suitable receiving vessel, and the frozen cells are forced through a small hole in the bottom of the cylinder by use of a hydraulic ram. A disadvantage of this technique is that the cylinder