vasoconstriction somewhere in the gill vasculature; this is the only plausible explanation for the set of circumstances where $\overline{VAP}$ rises with a fall in flow and dorsal aortic pressure. Since in mammals anoxia causes local vasoconstriction in the lungs, it is an attractive hypothesis that the active increase in gill resistance results from an intrinsic, local vasomotion. If this were the mechanism, however, gill resistance would be expected to show an even greater increase when cardiac output increase is an added feature of the response as in the preparations exposed to CO_2 after vagotomy and atropine. This is not the case; both pharmacological and surgical vagotomy abolish the rise in gill resistance altogether. It must be concluded that hypercapnia and anoxia have no direct local effect on gill vasculature, but that the change in blood gas does evoke a vagally mediated vasoconstriction. In report #28 this issue, external chemoreceptors are shown not to be a necessary sensory input, and pre-gill chemoreceptors are excluded as possible afferent inputs. Chemoreceptors in the efferent gill vasculature are a possibility and certainly hypercapnia and hypoxia may affect medullary centers in such a way as to increase motor vagal firing to the gills.

Central chemoreceptors have, in fact, been demonstrated in dogfish with the cardiac vagus intact and with section of the glossopharyngeal and the part of the vagus related to gill structures (J. Exptl. Biol. 38:531, 1961). In such a preparation anoxia caused bradycardia, but when the cardiac vagus was cut, no bradycardia was seen. Undoubtedly, cerebral anoxia plays a role in the heart rate response. Heart rate is also controlled by a baroreceptor reflex which slows the heart via the vagus in response to pressure increases in the ventral aorta (Biol. Bull. Woods Hole, 62:10, 1932). Such a reflex would become operative in our preparation secondary to the rise in gill resistance. Neither hypoxia nor hypercapnia produced bradycardia after removal of the nervous control of the heart. It is possible that a local stimulatory effect on heart rate could be masked by the inability of the heart to beat faster. An inotropic stimulation may be reflected in the increase in stroke volume. From measurements of max dp/dt, however, we were not able to demonstrate inotropic changes in three fish after atropine and 5% CO_2 in air. The stroke volume changes might also result from more complete filling if venous return were increased.

The idea of a vagally mediated vasoconstriction and bradycardia in the presence of hypercapnia and anoxia is not without precedent. The changes demonstrated in the dogfish are similar to those evoked by the diving reflex in seals and other mammals. Studies of further similarities would be interesting.

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1969 #14

TRANSPORT OF SUGARS INTO FLOUNDER TUBULES

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In a study program concerning the mechanism of sugar transport by renal tubular cells (cf. A. Kleinzeller; J. Kolínská; I. Beneš; Biochemical J. 104:843-60, 1967) the transport processes at the peritubular face of these cells were deemed to be of considerable interest. Therefore, the mechanism of transport of some sugars into teased tubules of flounder, Pseudopleuronectes americanus, kidney was studied. 0.5 - 1.0 mM D-galactose ^{14}C was transported into this prepa-

ration by an equilibrating system. The maximum T/M of 0.8 was practically reached within 30 min of aerobic incubation in a balanced saline at 15° C (air as gaseous phase). This galactose transport system was markedly inhibited by phlorhizin (0.5mM) but not by 0.1mM dinitrophenol, 0.5mM ouabain or some sugars (D-glucose,2-deoxy-galactose) at a molar ratio 1:10.

1mM 2-deoxy-D-glucose-^{14}C was accumulated to a slight degree (maximum T/M 1.3). The transport of this sugar was inhibited by 0.5mM D-glucose but not affected by ouabain or D-galactose.

The transport of 2-deoxy-D-galactose was investigated using a chemical assay specific for 2-deoxy-sugars. High tissue blanks prevented a detailed analysis of this transport system. However, evidence for an equilibrating transport system and its inhibition by 0.5mM phlorhizin was obtained.

The inulin-^{3}H space at 15° C was found to be 0.4 ml/g tissue.

These preliminary data suggest that the above three sugars are transported across the basal membrane of the flounder tubular cells by at least two systems of the facilitated diffusion type.

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1969 #15

ASPARTATE METABOLISM IN TELEOST LIVER

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In teleosts, ammonia is produced in the liver and excreted passively by the gills. Interestingly enough, L-aspartate is a major precursor for ammonia in teleost liver homogenates (Salvatore et al, Comp. Biochem. Physiol. 16:303, 1965). These authors speculated ammonia arose from L-aspartate by direct deamination. It seemed worthwhile to investigate this; particularly since enzymes for direct L-aspartate deamination are thought to occur only in plants and microorganisms.

Teleost liver homogenates (20%) were prepared in o.1M K_2HPO_4-KH_2PO_4 at pH 7.4. The incubation medium contained: 10 μmoles $MgCl_2$, 10 μmoles $Na^+AsO_2^-$, and 100 μmoles K^+L-aspartate or K^+L-glutamate, in a final volume of 3.0 ml. Ammonia production was calculated as: μmoles NH_3/gram tissue/hr, at 25° C. Values are the Mean ± SE, with 4 fish in each group.

Ammonia production from L-aspartate was 14.0 ± 9.7 in the eel (Anguilla rostrata), 5.8 ± 1.2 in the short-horned sculpin (Myoxocephalus scorpius), 4.2 ± 0.4 in the long-horned sculpin (Myoxocephalus octodecimspinosus), and 2.7 ± 0.8 in the flounder (Pseudopleuronectes americanus). In all species, L-glutamate proved to be a more active substrate for ammonia production; it was 29.6 ± 10.8 in A. rostrata, 8.5 ± 1.1 in M. scorpius, 8.5 ± 1.1 in M. octodecimspinosus, and 10.0 ± 0.9 in P. americanus.

Transaminase inhibitors were studied in homogenates of M. scorpius. Isonicotinoyl hydrazide (5×10^{-3}M), semicarbizide (4×10^{-3}M), and hydroxylamine (4×10^{-3}M), completely inhibited ammonia production from L-aspartate.

Finally, if a single enzyme deaminates L-aspartate, it should be demonstrable in at least one subcellular fraction. Subcellular fractions from P. americanus and M. scorpius were isolated in 0.25 M sucrose/0.02 M Tris, at pH 7.4, by differential centrifugation. No ammonia was