(1.6 molar or greater). We have found barrier opening with the more moderate increases in plasma osmolarity associated with systemic administration of hyperosmolar solutions.

Cerebrovascular permeability to ^{14}C-mannitol was measured in control and hypernatremic skates as the product of permeability x surface area (PS) using the integral technique of Ohno et al (Am. J. Physiol. 235:H229-H307, 1978). Radiolabeled test compound was injected intravenously and the arterial concentration followed for 30 min by analyzing serial blood samples collected from an A-V bypass, between the anterior mesenteric artery and posterior intestinal vein. The arterial concentrations define a time integral, I, and PS is calculated as PS = C/I where C is the brain parenchymal tracer concentration. Measurements were made over two experimental periods (5 to 35 min and 90 to 120 min) following evaluation of plasma osmolarity (42 or 56 milliosmoles NaCl im/kg body weight plus transfer to salt water made hypertonic with 62 or 83 mM NaCl, respectively).

During the earlier experimental period, 5 to 35 min after the hypersomotic stress, the permeability of the blood-brain barrier to mannitol increased by a maximum of 4-fold. Results in 5 control and 13 hypernatremic skates over the range in plasma osmolarity of 932 mosm (control) to 1060 mosm yielded the relationship: $PS = 3.73 \times 10^{-5} e^{.011y}$ where PS, the permeability x surface area product, is in min^{-1} and y, the osmolarity of plasma, is in mosm. By 90 to 120 min the permeability had returned toward control values (1.43 times normal, N=5) suggesting that the permeability increase in skates, as in mammals, is transitory. Supported by PHS NS 11050.

MOVEMENT OF ^{22}Na FROM EXTRADURAL FLUID TO BRAIN IN THE LITTLE SKATE (RAJA erinacea) DURING HYPERTONIC STRESS

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The brain of the little skate (Raja erinacea) is capable of a marked degree of volume regulation when exposed to hypertonic stress. This volume regulation is largely achieved by uptake of sodium chloride and accompanying water into the brain during this stress (Cserr et al., Bulletin MDIBL, 21:4, 1981). Since these ions are largely extracellular, the net result of this process is to maintain the extracellular fluid volume of the brain during periods when marked dehydration might otherwise occur.

The source of the volume regulatory influx of sodium and chloride into the brain during exposure to hypertonicity is unknown. Potential sources include plasma, cerebrospinal fluid and extradural fluid (EDF), the fluid which surrounds the brain of these elasmobranchs. The present study was undertaken to investigate the role of EDF as an ion source during hypernatremia.

Experimental animals were divided into two groups, control and hypertonic. All animals were anesthetized with 15 mg/kg sodium pentobarbital iv. The extradural space of control animals was perfused using a Harvard Apparatus perfusion pump with elasmobranch Ringers to which ^{22}Na or ^{125}I-radiolabeled serum albumin (RISA) had been added to a final concentration of 0.3-0.5 μCi/ml. Pump speed was set such that the outflow concentration of isotope reached a steady-state within ten minutes and outflow height was adjusted to maintain EDF pressure at pre-infusion control levels. After two hours of EDF perfusion the skate was sacrificed, a blood sample taken for electrolyte analysis and a sample of brain tissue from the telencephalon blocked, frozen, and sliced horizontally as previously described (Cserr et al., Bulletin MDIBL 21:4, 1981). Each slice was placed in a preweighed vial, reweighed to determine slice weight and counted in a gamma counter. Skates made hypertonic were perfused and treated in exactly the same manner as controls but prior to EDF perfusion these fish were given 56 mmole NaCl/kg body weight im and placed in sea water to which 78 mM NaCl had been added, raising plasma osmolality (mean $\pm$ SD) from control values of 942 $\pm$ 7 mosm to 1015 $\pm$ 35 mosm.

Previous analyses of net uptake of cold sodium by the brain during hypernatremia have shown that the process is relatively rapid, being essentially complete within two hours (Cserr et al., Bulletin MDIBL 21:4, 1981). These

data allow prediction of the brain space which ^{22}Na would occupy in all brain slices (cpm/mg telencephalon ÷ cpm/ul EDF) were the EDF to supply all of the sodium gained by the brain during this stress. As seen in Table 1,

Penetration of ^{22}Na and ^{125}I-HSA From EDF Into Skate Brain

Experiment	Isotope	Slice#	Tissue ÷ EDF distribution ratio, cpm/mg tissue ÷ cpm/ul EDF									
			1	2	3	4	5	6	7	8	9	10
Control	^{22}Na		.023	.011	.007	.007	.005	.004	.004	.003	.003	.002
Hypertonic	^{22}Na		.026	.018	.017	.016	.012	.017	.021	.028	.026	.016
Control + Hypertonic	^{125}I-HSA		.006	.003	.003	.002	.001	.002	.002	.003	.004	.004
Predicted Value of ^{22}Na space			.095	.095	.095	.095	.095	.095	.095	.095	.095	.095

All values are means of five to seven animals. Brain slices are numbered dorsal to ventral.

Although ^{22}Na values are consistently higher in hypertonic skates, differences are insignificant with the exception of the 10th slice due to the large SD in the hypertonic group.

the predicted distribution ratio (0.095) is 4-20 times greater than that measured in any brain slice under hypertonic conditions. There appears to be a tendency for skates exposed to hypernatremia to have more ^{22}Na penetration into the brain than controls, but differences between the two groups were not significant in all but one slice due to large standard deviations, particularly in the hypernatremic data. For RISA, penetration into the brain was identical in control and hypernatremic skates and, for the purposes of Table 1, the values from both RISA groups have been averaged. The results of these experiments clearly show that EDF is not the major source of the sodium taken up by the brain during hypernatremia. Supported by PHS NS 11050.

CHLORIDE EFFLUXES FROM THE PERFUSED DOGFISH HEAD

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Our recent studies with the intact pup of Squalus acanthias have indicated that the rectal gland plays an important but not unique role in NaCl extrusion. This conclusion is prompted by our finding that blood Na and Cl concentrations rise, after removal of the rectal gland, at rates significantly below those which can be accounted for by loss of this salt extrusion mechanism (Evans & Mansburger, Bull. MDIBL 19:101-103, 1979; Evans et al., J. Exp. Biol. in press, 1982). Similar results have been reported for the same species by Burger (Physiol. Zool. 38:191-196, 1965) and Forrest et al (Bull. MDIBL 13:41-42, 1973), for the lip shark, Hemiscyllium plagiosum (Chan et al., Comp. Biochem. Physiol. 23:185-198, 1967), and for the striped dogfish, Poroderma africanum (Haywood, J. Exp. Zool., 193:167-176, 1973). It has therefore been proposed that elasmobranchs may excrete salts via branchial transport mechanisms (Evans, Am. J. Physiol. 238:R224-R230, 1980). We therefore decided to initiate studies of the mechanisms of transport of Cl across the dogfish gill epithelium utilizing the perfused pup head which we recently described (Evans and Claiborne, Bull. MDIBL 20:9-11, 1981; Evans and Claiborne, J. Exp. Biol., in press, 1983).

The perfused pup head was prepared as described previously (Evans & Claiborne, ibid) except that in some experiments the plastic collar was not utilized and the head was suspended by plastic strips so that irrigation fluid drained into another container. No significant differences were seen in the pressure/flow or flux characteristics of heads suspended in the two different manners. Heads were perfused at approximately 750 µl/min (resulting in afferent pressures of 15 to 30 torr), and irrigated at 40 ml/min. Cl effluxes were measured by adding 3-5 uCi of 36 Cl to the recirculated perfusate (10-20 mls), and removing 1 ml samples of the irrigation bath (50 - 100 mls at various times thereafter and measuring the radioactivity via liquid scintillation counting. Drugs (ouabain or furosemide) were added directly to the perfusate after a 20 minute control period. Thus, each perfused head served as its own control. All data are expressed as mean ± s.e. (N).