TABLE 1. Observed and predicted chloride (extracellular) and non-chloride (cell) spaces in skate telencephalon

Condition	n	Chloride space, ml. g^{-1} dry wt.		Non-Cl-space, ml. g^{-1} dry wt.	
		Observed	Predicted	Observed	Predicted
Isotonic	7	1.45±0.06	-	3.26±0.09	-
Hypertonic 2 hr	5	1.52±0.03	1.23	2.89±0.10	2.77
Hypertonic 4 hr	5	1.40±0.03	1.20	2.89±0.05	2.69
Hypotonic 24 hr	6	1.62±0.03	1.79	3.95±0.08	4.02

Predicted spaces are calculated on supposition that they behave as perfect osmometers in relation to plasma [Na^+ + Cl']. Values are means ±SE.

TABLE 2. Chloride, sodium and potassium in skate telencephalon, mmol. $(100\ g)^{-1}$ dry wt., with % change in plasma [Na^+ + Cl']

Condition	n	Plasma, %Δ in [Na^+ + Cl']	Telencephalon		
			Chloride	Sodium	Potassium
Isotonic	7	-	38.5±0.8	42.2±0.8	55.3±0.3
Hypertonic 2 hr	5	+17.6	46.5±1.4	51.1±1.8	57.6±2.4
Hypertonic 4 hr	5	+21.2	45.5±2.2	49.9±1.9	58.7±1.8
Hypotonic 24 hr	6	-18.9	34.0±0.7	38.2±1.2	53.6±1.1

Values are means ±SE.

It is concluded that there is a rapid and considerable regulation of the extracellular fluid volume in skate telencephalon. Cell volume does not appear to be so well controlled over these relatively shoft time periods. Preliminary experiments with ^{22}Na suggest that the source of the sodium entry in hypertonic conditions is not extradural fluid.

H INTRACRANIAL PRESSURE DYNAMICS IN SKATES.

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In mammals, osmotic disturbances are associated with marked changes in intracranial pressure (ICP). The change in pressure is caused by the osmotic flow of water either into or out of the rigid cranial cavity. As part of a general study of the CNS response to osmotic disturbances in skates, Raja erinacea and Raja ocellata, we found that ICP failed to change in hypernatremia. In an attempt to explain this observation, we have analyzed factors related both to the initial perturbation (change in intracranial volume) and to compensatory mechanisms for alleviating changes in volume and pressure (compliance of the neural axis, resistance to fluid outflow).

In lower vertebrates the brain is surrounded by extradural fluid (EDF) rather than cerebrospinal fluid (CSF). In control skates EDF pressure (±SD), measured relative to the ventral surface of the skate, was 2.1 ± 0.2 cm H_2O compared to central venous pressure of 2.2 ± 0.4 cm H_2O. Measurement of brain water content in control and hypernatremic skates confirmed that water is withdrawn from brain tissue with hypernatremia (see abstract by Cserr et al., This Bulletin); there was no change in ICP, however, in skates monitored continuously for 2 hours (N=5) or 6 hours (N=1) following the increase in plasma osmolality. Conceivably, differences between EDF and CSF could contribute to the different pressure responses to osmotic disturbances in skates and mammals. Analysis of the size of the brain and EDF compartments as a function of body mass (Table 1) shows that as the size of the cranial cavity increases, EDF weight increases more rapidly than brain size. For a typical 1 kg skate, weights of EDF and brain are 3.4 g and 1.6 g, respectively. The large volume of intracranial fluid relative to brain tissue in the skate is in marked contrast to the typical vertebrate pattern in which CSF occupies a much smaller fraction of intracranial volume (e.g., CSF weight is roughly 10% of brain weight in man, 25% in goats, and 15% in rats). Determination of the osmotic gradient between plasma and EDF during hypernatremia revealed that the osmolality of EDF was slow to follow changes in plasma, consistent with the slow turnover of EDF constituents generally (Fenstermacher & Patlak, Am. J. Physiol., 232:R 45-R53, 1977). For example, 2 hours after producing hypernatremia EDF osmolality had risen by only 2.8% as compared to an increase in plasma of 9.9% (N=5). Based on these results - namely, that fluid occupies a larger fraction of intracranial volume in the skate than in a typical mammal and, further, that this fluid participates only slowly in osmotic exchanges with plasma - we predict that the fractional water loss from the cranium associated with hypernatremia (i.e., the total osmotic water loss from brain plus surrounding fluid) is smaller for a skate than for a mammal.

TABLE 1. Relationships for skates between intracranial parameters and body mass (M_b in grams)

Brain weight (g) = $0.057 \times M_b^{0.48}$
EDF weight (g) = $0.0045 \times M_b^{0.96}$
PVI (µl) = 29 (Brain weight + EDF weight) + 49

The compliance of the EDF-brain system was measured by observing the initial rise in ICP induced by bolus injection of varying, small volumes of fluid into the EDF. Results (Table 1) are expressed in terms of a pressure volume index, or PVI, where the PVI is the volume required to raise ICP tenfold. For animals of similar intracranial volume (2 ml) the PVI is roughly the same for a skate (107 µl) as for a rat (80 µl) (Melton. PhD thesis, Dartmouth College, 1981).

The resistance to EDF outflow was determined from the steady-state ICP response to a constant infusion of fluid into the EDF cavity over the pressure range 4 to 24 cm H_2O. Resistance, which decreased with skate size, averaged 370 cm $H_2O \cdot min \cdot Ml^{-1}$ for 1 kg skates. This falls within the range of values for resistance to CSF outflow for other vertebrates (Roomet & Fenstermacher, Comp. Biochem. Physiol. 51A:897-901, 1975). The pathways of EDF outflow are not known.

In summary, our analysis of ICP dynamics and of the EDF-brain system suggests that the lack of ICP response to hypernatremia in skates may be related to a smaller perturbation in skates relative to mammals (i.e., to a smaller intracranial water loss) but not to an increased compliance of the neural axis.