Volume Receptors and the Mechanism of Postprandial Diuresis in the Harbor Seal (Phoca Vitulina L.)

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Seals have pronounced postprandial diuresis without dilute urine but with 2-5 fold increase in glomerular filtration rate (GFR) (Hiatt, 1942). This change in GFR is not reproduced by water, salt, mannitol or uprea diuresis. Studies were done on 6 female seals to evaluate this diuresis and the existence of volume receptors in the seal. There was no change in urine flow with 20 cm water continuous positive or negative pressure breathing. This implies no atrial stretch receptors or demonstrates the effectiveness of the seal's inferior vena caval valve in control of pulmonary blood volume, important since the seal breathes relative negative pressure when swimming. Dextran was filtered so freely by the glomeruli of the seal kidney that it could not be used in the study. Intravenous infusion of 500 ml 1% gelatin in saline or 500 ml fresh seal plasma caused up to 40 fold increase in urine flow, without proteinuria or dilute urine, but with a 2 fold increase in GFR. Control infusions of 500 ml 5% dextrose in water or normal saline did not cause a change in GFR or as marked a diuresis. It is concluded that the dramatic postprandial diuresis in the seal is due to increased blood volume and is mediated via volume receptors. Preliminary studies in the seal with I^{131} albumen support this. It is suggested that the inferior vena caval valve protects the seal from pulmonary congestion while swimming with head above water.

Renal Function During Diving in the Seal (Phoca Vitulina)

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Renal clearances were performed on young female harbor seals trained with a teeter board to allow the cephalad portion of their body to be submerged without struggling for 10 minutes. Exogenous creatinine clearance was used as a measure of glomerular filtration rate and para-aminohippurate clearance was used as an index of renl plasma flow. Urine flow stopped during this type of diving. Following the dive, glomerular filtration rate and renal plasma flow returned to normal in a step wise fashion over a period of 4 to 6 minutes. Urine concentration and urine sodium concentration decreased progressively during the first few minutes after the dive with the occurance of a brief diuresis. The data was consistant with cessation of glomerular filtration rate and renal blood flow during normal diving. Unlike the changes in renal clearance when respiration was interrupted by an occlusive nose cone, (Bradley, et al, J. Cell. and

Comp. Physiol., 1942) these changes could be prevented by the administration of atropine. These changes in renal clearances with diving reveal that the renal circulation is envolved in the arterial constrictor response instead of representing a separate phenomenon of diving in the seal.

Function of Inferior Vena Caval Valve of the Seal (Phoca Vitulina)

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Assumptions have been made in the past that the inferior vena caval valve (IVCV) of the seal constricts during diving to pool blood in the hepatic sinuses and abdominal veins as a reservoir to use while submerged. This would seem not to be an advantage to the seal since most of the cerebral blood flows must return via the extradural vein to the abdominal veins. In order to evaluate the timing of constriction of the IVCV a cannula was threaded up the extradural vein to the level of the neck of the seal. Albumin I-131 administered into a foreflipper vein appeared from the cannula in 20 to 25 seconds whether the seal was diving or not. When the isotope was administered into the renal venous plexus it appeared from the cannula in 25 seconds when diving but was delayed in appearance when the seal was out of water. The data indicate that the IVCV constricts when the seal is out of water. This would fit the suggestion that the IVCV serves to prevent pulmonary congestion when the head is above water.

Observations on the Arterial Constrictor Response To Diving in the Seal (Phoca Vitulina)

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Young harbor seals were trained to dive on a teeter board with the cephalad portion of the body submerged during the diving periods. They learned to dive in this manner for 10 minute periods without struggle or other evidence of fright. Changes in extradural vein blood lactic acid concentrations and oxygen content at 4 minutes of diving, and observations of small webb arteries were used as indices of function of the arterial constrictor response to diving. At 4 minutes of normal trained dive there was no increase in blood lactic acid concentration and little decrease in oxygen content. After I.M. atropine or I.V. tetraethylammonium chloride there was no reflex bradycardia to diving and blood lactic acid concentration increased with the blood oxygen content markedly reduced at 3 minutes of diving. Pilocarpine, given I.V. after the tetraethylammonium chloride but