TITLE: LOW DOSE VASOPRESSIN IN ORGAN DONORS

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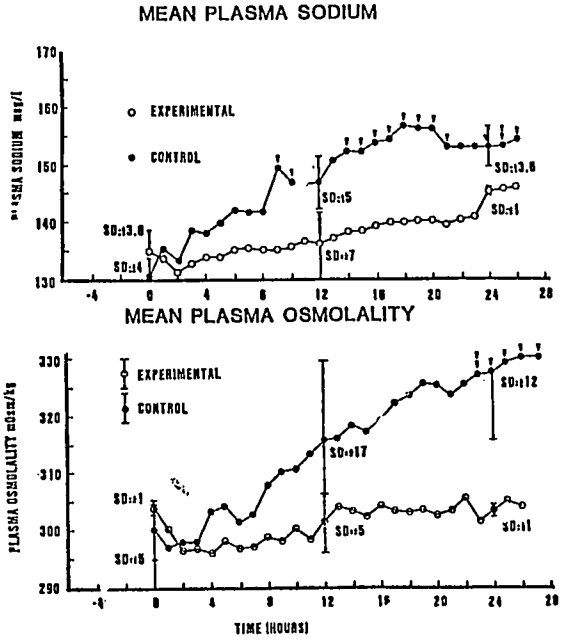
INTRODUCTION. The renewed enthusiasm in transplantation of organs other than kidneys requires improved donor management to ensure adequate cardiac function and organ perfusion. Diabetes insipidus, a feature of the brain dead organ donor, leads to gross derangements in fluid and electrolyte balance manifested by hyperosmolality, hypernatremia and fluid shifts.[1] The role of low dose (2-10 ug/kg/min) vasopressin supplementation in an animal model of an organ donor was investigated.

MATERIALS AND METHODS Six domestic pigs were used as the experimental model. They were anesthetised, intubated and ventilated to maintain PaO_2 and $PaCO_2$ normal. Monitoring lines were passed via the internal carotid arteries to measure aortic and left ventricular pressures and via the external jugular to measure pulmonary artery pressures and cardiac output by thermodilution. Urine output and fluid volume administered were monitored. Plasma sodium, potassium and osmolality were measured regularly. Specimens of plasma for vasopressin analysis by Roche Laboratories were taken according to accepted practice. A craniotomy was made and the cranial contents were removed, great care being taken to remove the Pituitary gland. Polyuria invariably ensued within three hours and the experiments continued for 24 hours. The animals were divided into control and experimental groups with the three experimental animals each receiving a vasopressin infusion (250 ug/ml) at a rate of 2-10 ug/kg/min. once polyuria had been clearly documented for one hour. Analysis of variance was performed between the two groups using a SAS program. Statistical significance was accepted at $p < 0.05$ value.

RESULTS. There were no significant differences in systemic arterial pressure, left ventricular pressure and pulse rates between the two groups. Cardiac output remained constant in all animals for the duration of the experiments. There was a gradual and sustained moderate elevation of pulmonary artery pressure in the experimental group. The control animals showed progressive rises in plasma sodium and osmolality. Urine sodium and osmolality fell to low values and remained low. Potassium supplementation was elevated. All the experimental animals maintained plasma sodium and osmolalities in the normal range. Potassium supplementation was minimal. Plasma Vasopressin was undetectable in all animals within three hours of brain removal. Vasopressin supplementation in the experimental animals maintained plasma vasopressin in the normal range.

DISCUSSION. The organ donor invariably develops cardiac failure with time and this may prevent the use of that particular heart for transplantation. What factors contribute to the onset and development of cardiac failure in a previously healthy person? Recent reports have shown that rats with Diabetes insipidus have altered myocardial electrophysiology and derangements in intra and extracellular electrolyte distribution.[2] This would apply to the brain dead organ donor with Diabetes insipidus and when coupled to aggressive fluid resuscitation, severe fluid shifts may occur resulting in interstitial and intracellular edema and promoting the development of myocardial dysfunction and cardiac failure. Vasopressin supplementation in organ donors is a controversial subject. It is a potent vasoconstrictor and if used as currently recommended its effects on the renal vasculature may indeed result in vasoconstriction, ischemia and impaired renal function in the post-transplantation period.[3] However administration of low dose vasopressin as a continuous infusion aimed at achieving a physiological plasma concentration prevented the sequelae of Diabetes insipidus in this study without any systemic vasoconstrictive effects. It would appear that further research in human organ donors is warranted.



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