A Study on the Effect Brain Death has on organs

D. TABROSKA, J. BONAVENTURA, V. DIVISOVA.

Summary

The effect of brain damage and brain death on organ pathology has been studied both in the patient donor and also in the experimental model using the dog. The morphological and functional changes occurring where studied after the loss of central vascular and nervous regulation. The serum creatinine level is used as a test of function of the kidneys of the potential donor.

Introduction

Tracing the effect brain death has on organs in the clinical material is very difficult. Many hours pass between the beginning of brain damage and between brain death. During this time, the organs are influenced by:

a) Isolated brain damage and brain death which in case of a trauma leads to a conspicuous gradation of sympatho-adrenal activity. The kidneys are insufficiently supplied with blood, the liver looses its reserves of energy, in direct correlation with the length of time after injury.

b) Traumatic damage to brain cells, often connected with polytrauma, leads to release of thromboplastine and an increased incidence of coagulopathy with further changes in microcirculation.

c) Brain death causes failure of control over water resorption in kidney tubules. The loss of liquids, when not recompensed sufficiently leads to hypovolemia and to a further loss of volume of blood available to the organs. This central polyuria leads to hypokalemia which may cause further changes in morphology and function of the kidneys. The destructive changes after polytrauma, connected with loss of blood are much greater than those of isolated brain damage.

The loss of central brain regulation on peripheral organs after brain death is caused by the block of ADH secretions so that the autoregulation of peripheral organs is then dependent on changes in blood flow and on changes of acid base concentration. In such case a minimum stimulus which is usually no problem for a normal organism with an integrated CNS may lead to damages in organs.

The Canine Model

We tried to clear up the matter in our experimental model by causing brain trauma in dogs. We biopsied the liver and the kidneys in the 3rd, 6th and 12th hour after the experimental brain death. A very important premise of the experiment was the fact that no disorders in haemodynamics occurred, as the loss of liquids after the beginning of polyuria was measured and replaced carefully. After three hours we found in the liver of dogs oedematous changes in hepatocytes and accumulation of leukocytes in the centrilobular zone.

In the kidney the lumen of proximal tubules was dilated due to flattening of proximal tubular epithelium. Monocellular necroses of tubular epithelial cells are rare. These histological findings demonstrate the fact that isolated brain death can cause a state similar to shock in the organs. These changes are usually reversible. On the other hand, when hypovolemia and other changes are caused by central polyuria after polytrauma, the damage of organs may be irreversible.

The Human Model

A 28 year old patient was treated after a short time of arrest of blood circulation and was successfully resuscitated. Samples were obtained by means of kidney biopsy and showed shock kidney with hydropic dystrophy of proximal tubular epithelial cells and monocellular necrosis (acidophilic necrosis) (Figure 1).

After 6 hours a second biopsy showed the kidney with advanced changes in tubular epithelial cells, and focal tubular necrosis. Necrotic acidophilic cells are present in tubular lumina necrosis of cells are more numerous. This is a picture of an advanced shock of the kidney (Figure 2).

Prof. D. Tabroska M.D., Dr. J. Bonaventura M.D., Dr. V. Divisova M.D., Department of Anaesthesiology and Resuscitation, Medical Faculty – brno Czechoslovakia. *Head* Ass. Prof. D. Tabroska M.D.

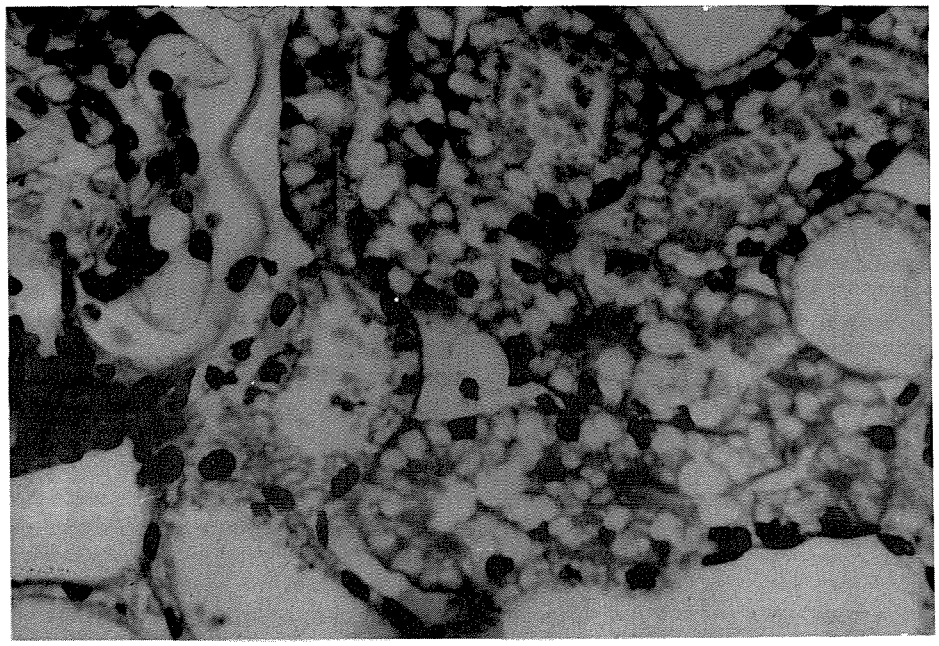

FIG. 1 Biopsy of kidney taken soon after circulatory arrest. Hydrophic dystrophy of proximal tubular epithelial cells. Few necrotic cells noted.

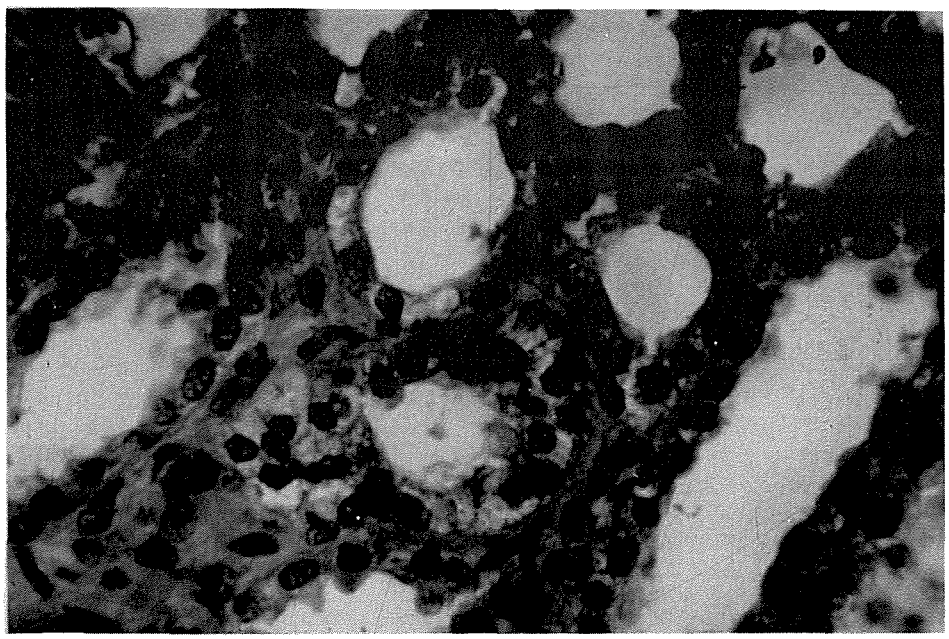

FIG. 2 Biopsy of kidney taken 6 hours after circulatory arrest. Advanced changes in tubular epithelial cells. Focal tubular necrosis. Necrotic acidophilic cells are numerous.

Canine Creatinine Studies

Changes in filtration and resorption of creatinine in the bodies of our experimental animals was studied. In the course of 12 hours the filtration went down by 50 – 100%, the resorption decreased by 90%.[1] The value of creatinine in the serum was found to be twice normal possibly due to release of creatinine from another source besides the body musculature. Can it be creatinine phosphate from the brain tissues? The level of creatinine in the venous cerebral drainage was studied and compared to the level in mixed venous blood. The curve of creatinine in venous cerebral blood shows two rises a) after brain damage, b) after arrest of cerebral circulation. This is a significant finding. To clear the problem we studied two canine models having a) a sustained cerebral circulation after brain death resulting in an increased level of creatinine in venous cerebral blood and b) an arrested cerebral circulation and brain death resulting in a stable creatinine level in venous cerebral blood.

The difference in creatinine level depends on the time between brain damage and arrest of cerebral circulation.[2] While the changes in filtrations are approximately the same the value of creatinine in cases with a delayed arrest of brain function is higher. It appears that serum creatinine level is not a good enough parameter for assaying kidney function.[3]

Conclusion

The canine model provides a basis for studying the differential effect brain death alone or combined with arrested cerebral circulation has on other organs. The application of these findings to the human model with brain death will clear problems associated with efficiency of the kidneys for transplantation.

References

1. **Brotchner-Mortensen, J. Tougard, L.,** etc. Individual Determination of Glomerular Filtration Rate from Plasma Creatinine. Scand. Chir. Lab. Invest., 1976, 36: 389 – 393.

2. **Mitch, W.E., Collier, V.V., Walser, M.:** Creatinine Metabolism in Chronic Renal Failure. Clin. Sci., 1980, 58: 327 – 335.

3. **Preuss, H.G.:** Tubular Function in Experimental Acute Tubular Necrosis in Rat. Kidney Int., 1976, 105: 51 – 57.