

normal. Group III. Four rabbits. Alcohol was injected into the periosteum of the femoral neck; the periosteum was stripped from the entire neck of the femur. Microscopic sections showed nothing of importance except for a few small areas of aseptic necrosis of the bone. Apparently, the circulatory embarrassment caused only a limited necrosis, reparable without gross changes in the head. Group IV. Eleven rabbits. The round ligament ligated with a silk suture; the periosteum stripped from the entire femoral neck; silk suture placed around neck. In one dog (10 weeks) the femoral head appeared normal grossly but in all others it was greatly roughened and flattened. Microscopically in all cases, the bone and cartilage of the head were uniformly and markedly atrophied and, in many areas, necrotic. Of particular interest were well defined cystic spaces which represented areas of degeneration of the medullary bone and of the marrow tissues. As early as 2 weeks after operation, new vessels were seen growing from the shaft along the surface of the neck and the necrotic areas were found to be undergoing repair.

It is concluded that the circulatory deficiencies of the head of the femur produced in Group IV caused gross and microscopic changes similar essentially to those of the human lesions of osteochondritis. It seems probable that the dog in Group IV without flattening of the femoral head would have developed that deformity if it had been kept alive a little longer, since the collapse in the other cases was very probably due to weakening of the internal structure and weight-bearing.

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Acidosis in Cholera. I. Path of Displacement of Serum Acid Base Equilibrium.

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During the outbreak of cholera in the summer of 1932 we had the opportunity of studying 28 cases from the standpoint of their serum acid-base equilibrium. With the micro-acid-base method of Shock and Hastings,¹ cell volume, pH, and total CO₂ of blood were

¹ Shock, N. W., and Hastings, A. B., *PROC. SOC. EXP. BIOL. AND MED.*, 1929, **26**, 780.

determined and from these values serum bicarbonate content in millimols per liter and carbon dioxide tension in millimeters of mercury were calculated. These observations were made on admission before treatment and repeated thereafter almost daily throughout their hospital sojourn. Altogether 222 observations were made.

During the acute stage of cholera there was invariably acidosis. Serum pH obtained from this series of cases on admission averaged 7.28, bicarbonate content 14.1, and CO₂ tension 30.7. The lowest pH recorded in this series was 7.07, the lowest bicarbonate content 8.2, and the lowest carbon dioxide tension 16.6, as compared with the normal average values of 7.40, 26.6, and 44.9, respectively.

With recovery following intravenous therapy with either saline or alkali or both, pH returned to normal fairly rapidly and was followed by the return of bicarbonate. Soon after these values returned to normal they were often shifted to the alkaline side. Fourteen cases in this series which were followed for a relatively long period during their convalescence showed a tendency to alkalosis, irrespective of whether or not they had received alkali. During the stage of alkalosis these cases showed on the average pH 7.51, bicarbonate content 31.3 and CO₂ tension 42.3.

In the 4 cases of death in this series the intravenous treatment was capable of bringing the serum pH and bicarbonate content to normal, but this was only temporary.

When all the observations on the 28 cases are grouped according to their pH ranges, and the average values of pH, bicarbonate and CO₂ tension of each group are plotted on triaxial coordinate paper, the general trend of displacement of serum acid-base balance may be described as follows: A change in bicarbonate involves a change in both pH and CO₂ tension. Similar to experimental displacements of acid-base balance by the administration of acids or alkali,² the ratio of change in bicarbonate:pH:CO₂ tension is approximately 3:2:1, when bicarbonate and CO₂ tension are also expressed logarithmically.

² Liu, S. H., and Hastings, A. B., *Proc. Soc. Exp. Biol. and Med.*, 1931, **28**, 781.