exogenous and endogenous parathyroid hormone. In Tashjian's studies(5), Actinomycin-treated rats were parathyroidectomized and given exogenous parathyroid hormone 3 hours after administration of the drug. Hypocalcemia was noted 7 hours after Actinomycin (4 hours after parathyroid hormone injections). In our studies, hypocalcemia did not occur until 10 hours after administration of the drug to rats with intact parathyroids. It must be noted also that Tashjian *et al*(5) did not obtain hypocalcemia 7 hours after administration of Actinomycin-D to rats with intact parathyroid glands. They were unable, as we are unable at the present time, to explain the difference between the effect of the drug on exogenous and endogenous parathyroid hormone.

From present available data, no definite conclusion may be reached as to the exact mechanism of action whereby Actinomycin-D produced hypocalcemia. It is possible that the drug, by antagonizing the calcium mobilizing effect of endogenous parathyroid hormone, causes a fall in serum calcium. However, another possibility must be considered. Since parathyroid hormone is a protein or polypeptide and by definition may be affected by agents blocking synthesis of proteins, does Actinomycin-D inhibit synthesis and/or secretion of parathyroid hormone, and by this mechanism affect calcium homeostasis? This question was not answered by our experiments. Further studies are indicated.

Summary. Actinomycin-D produces hypocalcemia in rats 10 to 15 hours after administration. This effect can be produced in the absence of kidneys, adrenals and thyroid glands. Possible mechanisms of action are discussed.

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Effect of Hemodialysis on Serum and Parotid Fluid Phosphate Levels. (30344)

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Mechanisms controlling the inorganic phosphate concentration of parotid fluid are not understood. Studies by Langley *et al*(1) have suggested that parotid fluid phosphate levels in the dog are influenced by alterations in serum values. A 7-fold increase in plasma phosphate doubled parotid fluid concentration. Studies with human subjects have produced conflicting results in this regard(2-10).

The high serum phosphate levels encountered in azotemic renal disease are subject to rapid readjustment when hemodialysis is employed. The present study was designed to determine the effect of decreasing abnormally high serum phosphate concentrations by hemodialysis on parotid fluid phosphate levels.

TABLE I. Effect of Hemodialysis on Phosphate Levels.

Subject identification	Serum phosphate (mg/100 ml) Hr of dialysis 0	1.5	3.5	5.5	Parotid fluid phosphate (mg/100 ml) Hr of dialysis 0	1.5	3.5	5.5
1 (34 yr ♂)	7.3	5.3	4.9	4.7	16.2	18.0	18.2	18.5
2 (40 yr ♀)	10.8	8.3	5.0	3.8	10.7	10.5	9.9	9.5
3 (14 yr ♀)	8.8	6.4	5.6	5.2	11.8	9.4	10.2	10.7
4 (41 yr ♂)	9.7	6.5	4.5	4.1	9.5	10.5	8.5	12.8
5 (24 yr ♂)	9.5	8.0	8.0	7.5	13.0	13.7	13.9	17.5
Mean	9.2	6.9	5.6	5.1	12.2	12.4	12.1	13.8

Materials and methods. Three of the 5 patients studied (Nos. 1, 2, 3) underwent hemodialysis* in treatment of acute renal tubular necrosis, while the other 2 (Nos. 4, 5) had chronic azotemic renal disease. Initial blood and parotid fluid collections were made prior to treatment and at 1.5, 3.5 and 5.5 hours after initiation of hemodialysis. Parotid fluid was collected in graduated centrifuge tubes by means of a small metal vacuum device(11). Sour cherry drops were used to elicit flow and collection intervals were carefully timed. Inorganic phosphate was measured by the method of Fiske and Subbarrow (12) as adapted to the AutoAnalyzer(13).

Results and discussion. Table I presents individual observations as well as means for phosphate levels in the two fluids. The anticipated decrease in serum phosphate concentration was observed in all instances; a definite decrease was apparent for each patient at the first sampling interval after dialysis began. The means for parotid fluid phosphate were not markedly altered over the period of dialysis. The glandular management of phosphate during dialysis was thus distinctly different from that previously reported for urea nitrogen(14).

Parotid fluid flow rate means for the 5 subjects at the 4 testing intervals were 0.82, 0.77, 0.79, and 0.83 m/min. Hemodialysis did not exert a significant influence on this variable.

Available evidence indicates that there is apparently no direct relationship between serum and parotid fluid inorganic phosphate levels in humans. Bates(10) has shown in one apparently normal human subject, that methods directed toward altering plasma phosphate levels (phosphate infusion, glucose ingestion) did not influence parotid fluid phosphate concentration. Freeman and Welt(15) found, in a group of patients with hypercalcemia of varying etiology, a definite tendency toward increased parotid fluid phosphate, even though the majority of these patients were hypophosphatemic. The results of the present study are in accord with these observations in that marked acute decreases in serum phosphate levels produced by hemodialysis were not accompanied by alterations in parotid fluid phosphate concentration.

Summary. In patients with acute renal tubular necrosis or chronic azotemic renal disease, marked decreases in serum inorganic phosphate levels produced by hemodialysis were not associated with changes in parotid fluid phosphate concentration.

The opinions expressed herein are those of the authors and do not necessarily reflect official USAR or USAF policy.

* All dialyses performed on the Travenol twin-coil (disposable artificial kidney).

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