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Effect of Diet and Viosterol on Calcium Deposition in the Callus of Parathyroidectomized Rats.

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Canal[1] showed that calcium fails to be deposited in the callus following fracture in parathyroidectomized rats, and Morel[2] noticed a similar defect in cats. Erdheim[3] concluded that the deficient calcification in the callus throughout the skeleton of parathyroidectomized rats resembles that of rickets and osteomalacia. The above experiments were conducted when exact knowledge of dietary requirements were wanting and hence, there is a possibility that the diet may have contributed to the failure of calcification in the callus in the presence of a parathyroid deficiency. The following experiments were conducted to ascertain the effect of alterations in the calcium and phosphorus diet and of viosterol on callus calcification.

The procedure and diets have been briefly described.[4] The radii and ulnae of rats were fractured while under amytal anaesthesia. The rats were placed on a low calcium diet until tetany developed and then they were divided into groups according to the diet they were to receive. X-rays of the fractured bones were taken weekly until the rats were killed.

The results may be summarized as follows:

1. (a) *Low calcium diet.* In spite of the low calcium diet the calcification of the callus progressed moderately within 30 days as evidenced by X-ray. The serum calcium in this group remained low and the animals continued to have tetany.

(b) When 1% $CaCO_3$ was added to the diet, the calcification in the callus seemed somewhat denser.

(c) *Stock diet.* Very good calcification of the callus was observed within 20 days.

(d) *Steenbock rachitogenic diet.* In spite of its being a rickets-producing diet, the calcium deposition in the callus was marked.

(e) *High phosphorus and low calcium diet.* The deposition of calcium salts was very poor, even after a month.

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[2] Morel, L., *Compt. rend. Soc. de Biol.*, 1911, **63**, 749.

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2. (a) Low calcium diet with 5% viosterol. Callus formation fair, somewhat better than with low calcium diet alone.

(b) When 500% viosterol was added to the above diet the calcifying effect in the callus was very apparent.

(c) When 1% $CaCO_3$ and 500% viosterol were added to a high phosphorus and low calcium diet, the calcium deposition in the callus was definitely increased, but the degree of deposition did not approach that of a normal callus or a callus in the experiments in which less phosphorus and the same amounts of calcium and viosterol were used.

Unoperated animals kept on a stock diet showed nearly complete healing of fractures in 18 days.

The results seem to indicate again the importance of diet in parathyroidectomy experiments. They prove clearly that in the presence of a parathyroid deficiency calcification does not occur regularly, if calcium is also lacking in the diet. They also show that the property of viosterol in elevating the serum calcium level after parathyroidectomy is evident in its ability to promote callus calcification. In comparison with the normal animal, however, callus calcification is somewhat delayed.

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Role of the Parathyroids in Calcification, and Susceptibility of Parathyroidectomized Rats to Viosterol.

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The long continued administration of parathyroid extract to animals results in an increased excretion of calcium and phosphorus from the body.[1, 2, 3] Similarly, the presence of hyperfunctioning parathyroid glands results in a negative calcium and phosphorus balance and bone decalcification termed clinically, *osteitis fibrosa cystica.* Several observers noticed marked improvement when one or more of these glands was removed. Hence, in hypofunction a marked retention of calcium and phosphorus should result. Greenwald,[4] contrary to previous assumptions, demonstrated retention of

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[4] Greenwald, I., Gross, J. J., *J. Biol. Chem.*, 1925, **66**, 185.