ally this furrow is shallow, and on the left side where it approaches the distal end of the genal cæcum (Fig. *B*) a curvilinear raised ridge originates which runs back and then bends inward slightly to meet the posterior border. Can this conceivably mark the position of an early lateral or posterior suture at a stage when *Scotoharpes*, at least ancestrally, showed affinities with a genus like *Aulacopleura*? The possibility of such a relationship is supported by the recent work of Přibyl[4], in the general background of that by Stubblefield[5]. The definite suture close to the sides of the glabella suggests the condition in Olenidæ and Prœtidæ. *Prœtus loredensis* Delgado, in particular, has recently been compared with the Harpedidæ[6].

Ruedemann has suggested that the evolution of a genus like *Cryptolithus* has been marked by great lateral development of the cheeks. It is to this he attributes the length and parallelism of what he calls the "lateral facial sutures". While this may be true, one must also consider the likelihood of eyes migrating inwards and upwards on to the highest parts of the cheeks in a mud-dweller. Even if functional eyes were afterwards lost, the tendency for the migration towards the glabella appears to be supported by the course of the sutural traces in *Trinucleus fimbriatus*. Migration of eyes inward, possibly from the position of slight depressions behind the outer ends of the genal cæca in *Scotoharpes domina*, also may be arguable.

It is not easy to answer the question as to why traces of what may be facial sutures should remain on the inside of a cephalon of *Trinucleus fimbriatus*, unless they continued to serve as lines of easy splitting. In *Scotoharpes* the lateral and posterior sutures were evidently functional, and their presence on the alæ may mean that the latter had a sensory function during ecdysis and perhaps at other times.

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Changes in the Incisor Teeth and Incisal Alveolar Bone of Rats in Hypervitaminosis A and Avitaminosis A

THE changes in bone in avitaminosis A have been reported by Mellanby[1], Wolbach and Bessey[2], and Wolbach[3], who consider that the vitamin is concerned with the control of bone modelling. Mellanby found excessive formation of bone in certain areas during avitaminosis and that the osteoblasts and osteoclasts could reverse their positions at the effective bony surfaces. Wolbach[3] considered that appositional bone formation continued in the normal site, but that concurrent resorption of bone ceased. Excessive doses of vitamin A have been found by many writers to cause spontaneous fractures of bones and internal hæmorrhages (see Moore and Wang[4] for a review of the literature). Wolbach[3] considered that bone remodelling was greatly accelerated and that a defective tissue was laid down.

The effects of vitamin A deficiency on the teeth are already well known[5]. A defective dentin is laid down by the odontoblasts, especially on the labial side. Pohto[5] and Wolbach[3] both reported that hypervitaminosis A has no effect upon the rat's incisor teeth.

Through the kindness of Dr. T. Moore and Mrs. S. E. Walker, of the Dunn Nutritional Laboratory, Cambridge, I obtained the heads of animals suffering from hypervitaminosis A. These were young rats and had been given 40,000 I.U. of vitamin A daily for 10–42 days. Their incisal alveolar bone and upper incisor teeth were examined microscopically. In the alveolus, the rate of bone formation was greatly reduced, many cementing lines were seen and active osteoblasts were much less prominent than usual. Osteoclasts appeared to be unaffected, so that the bone became abnormally thin and disappeared in places. In the teeth, only dentin formation was affected. This became decreased in appositional rate, the interfibrillar cementing substance was gradually reduced in amount, and the lingual odontoblasts had begun to atrophy.

The avitaminotic material studied was from rats previously investigated (Irving and Richards[5,6]). The animals had been on the vitamin-free diet used by us 28–53 days from weaning. The alveolar bones showed considerable over-production of new bone. This occurred in areas where apposition was usually seen and also in situations where resorption normally occurred. In the early stages of the deficiency, the osteoclasts endeavoured to overcome the latter abnormal apposition, while working alongside invading osteoblasts. Later they became displaced by osteoblasts and some osteoclasts were found on the opposite side of the bone. On faces where excessive apposition occurred in the usual site for apposition, osteoclasts were never found. The changes in the teeth in avitaminosis were the same as those reported by earlier workers.

It is concluded from these results that the primary action of vitamin A is upon the osteoblasts. Excessive dosage with vitamin A depresses the action of these cells, and, when the vitamin is lacking from the diet, these cells engage in disorderly overactivity. The reactions of the osteoclasts in avitaminosis are purely secondary, in an attempt to prevent excessive bone formation. In hypervitaminosis A these cells continue to act as usual. The odontoblasts, cells in the teeth comparable to the osteoblasts, react in a way similar to that of the bone-forming cells, producing less dentin in hypervitaminosis and excessive amounts in avitaminosis.

The expenses of this work were defrayed by grants from the South African Council for Scientific and Industrial Research and from the Staff Research Fund, University of Cape Town.

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April 27.

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