tion and inhibition has also been reported in the spinal cord[3]. There is anatomical evidence[8] for the presence in the central nervous system of excitatory interneurons which synapse with presynaptic terminals as well as with the dendrites of the neurons on which these presynaptic terminals end. If pentylenetetrazol blocks both of these sites, its combined effects of facilitation and inhibition, as well as the lack of dose-response relationship, would be accounted for.

Résumé. Les effets du pentylènetetrazol sur la transmission de l'influx nerveux dans le noyau de Burdach furent étudiés. Il apparut que ce produit empêche l'augmentation de l'excitabilité des extrémités afférentes cunéaires et l'inhibition de la réaction lémnisque, ceci en conditionnant les décharges cutanées. Le pentylènetetrazol réduit aussi l'intensité de l'onde de surface positive. Le pentobarbital parut s'opposer à ces effets. En conclusion, le cardiazol bloque l'inhibition présynaptique.

N. R. BANNA and J. HAZBUN

Department of Pharmacology, School of Pharmacy, American University of Beirut, Beirut (Lebanon), 7 November 1968.

[8] F. I. KHATTAB, Experientia *24*, 690 (1968).

Vitamin A-Deficiency and Cartilage in Healing Skull Fractures of Rats

During the healing of skull vault defects in the rat fed adequately, cartilage is seen rarely[1] or none is reported[2,3], except in one experiment[4] involving 50 rats less than 7 days old of which 19 formed some cartilage. HOWELL and THOMPSON[5] have related an abnormal subperiosteal formation of cartilage in the lumbosacral vertebrae to a vitamin A-deficient state in chicks. This report concerns the incidence of cartilage development observed in repairing parietal bone defects in 32 albino rats made vitamin A-deficient[6] beginning at weaning and in 24 control litter-mates treated similarly but given orally in almond oil a supplement providing 50 IU of vitamin A palmitate per day. The bone lesions were made by scraping back the pericranium of one side, drilling through the skull and enlarging the hole by breaking off small bone pieces.

Operations were performed on animals between 12–18 weeks of age. At that time the vitamin A-deficient group alone showed most or all of the following symptoms: white fur, pale incisor teeth, inflamed eyelid margins, low weight and loss of appetite. Assays[7] performed at death on 10 deficient animals gave vitamin A concentrations between 0–9 μg/100 ml in the blood and 0–9 μg/g in the liver. All fractured skull vaults were prepared for histological examination.

Cartilage developed between the 9th and 28th postoperative day in 12 of the 21 vitamin A-deficient animals, but only in 1 of the 19 A-supplemented rats, surviving into that period. The cartilage occurred as

[1] J. J. PRITCHARD, J. Anat. *80*, 55 (1946).
[2] C. J. SUTRO and S. A. JACOBSON, Archs Path. *28*, 313 (1939).
[3] G. H. BOURNE, Proc. R. Soc. Med. *37*, 275 (1944).
[4] F. G. GIRGIS and J. J. PRITCHARD, J. Bone Jt Surg. *40-B*, 274 (1958).
[5] J. McC. HOWELL and J. N. THOMPSON, Br. J. Nutr. *21*, 741 (1967).
[6] By feeding the vitamin A test diet supplied by Nutritional Biochemicals Corp., Cleveland.
[7] The assistance of the Nutrition Laboratory of the American University of Beirut is gratefully acknowledged.

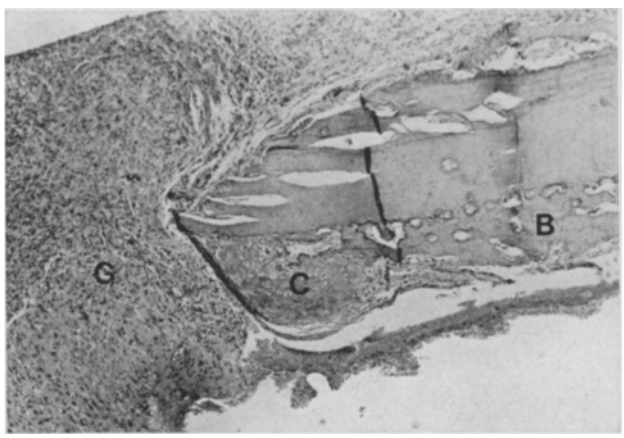


Fig. 1. The skull margin of a vitamin A-deficient rat surviving 21 days has a callus on its dural side comprising bone (B), and cartilage (C) near to the brain (G) herniating through the defect. Haematoxylin and eosin. × 80.

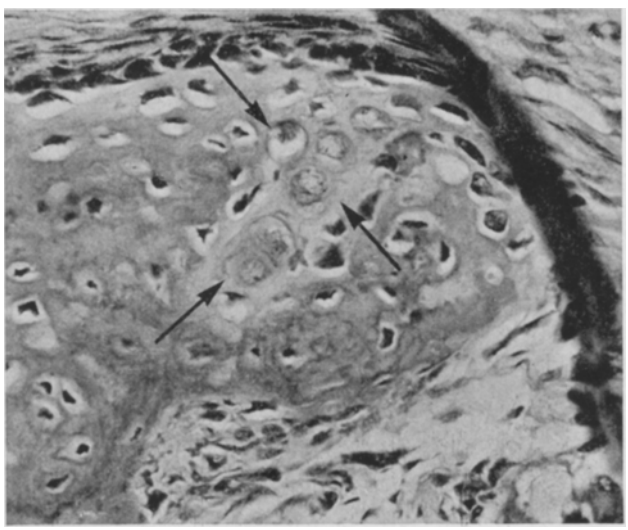


Fig. 2. The bony callus of a vitamin A-supplemented rat living 21 days post-operatively includes a small area of cartilage (arrowed). Haematoxylin and eosin. × 500.

small nodules comprising only a part of the callus and usually lay at the defect's margin on the dural side of the bone. The cartilage was then in close proximity to brain tissue herniating through the defect (Figure 1). A χ^2 test shows the difference in the occurrences of cartilage between deficient and supplemented groups to be significant at the 0.1% level. Moreover, the cartilage formed in the one vitamin A-supplemented rat was extremely scanty (Figure 2).

Most deficient rats did not eat and lost weight postoperatively. Although their deficiency thus was probably multiple, adequate materials were recruited to form a bony callus which included the islands of cartilage.

Possible factors promoting the development of this cranial cartilage could be: (1) an ischaemia[8] resulting from a pressure by the herniating brain; (2) a mechanical action by the mis-sized brain[9] on osteoprogenitor cells of the callus; (3) a chemical inductive effect by the brain tissue[10]; (4) a regressive return to the cartilage-forming tendencies of neonatal development at sutures[11] or neonatal repair[4]; or (5) a direct action on the callus-forming cells of the low level of vitamin A. Excessively high levels of vitamin A cause cartilage destruction in vitro[12]; low levels in vivo might provide a permissive environment in which cartilage could be formed and survive[13].

Zusammenfassung. Die Heilungsvorgänge bei experimentell erzeugten Defekten der Parietalknochen junger Ratten wurden untersucht: Knorpel entwickelt sich im Kallus nach 9–28 Tagen bei Tieren mit einem Vitamin-A-Defizit, jedoch nur in einem von 19 Kontrolltieren, welche Vitamin A zusätzlich erhalten haben.

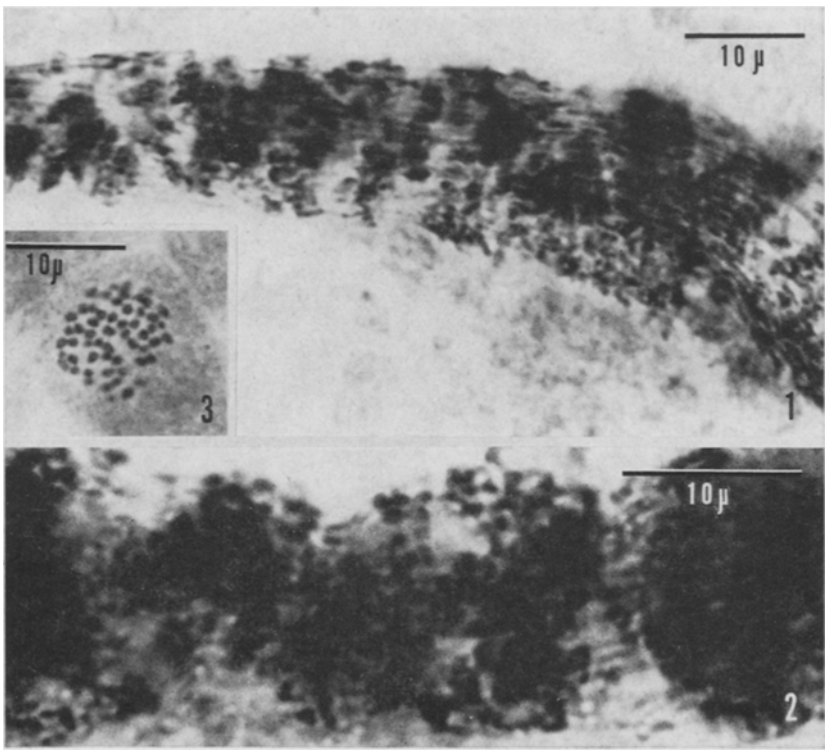
W. A. Beresford[14]

Department of Anatomy, School of Medicine, American University of Beirut, Beirut (Lebanon), 7 October 1968.

[8] A. W. Ham, J. Bone Jt Surg. *12*, 837 (1930).
[9] R. W. Young, Am. J. Anat. *105*, 383 (1959).
[10] E. B. Siqueira and P. C. Bucy, J. Neuropath. exp. Neurol. *25*, 667 (1966).
[11] J. J. Pritchard, J. H. Scott and F. G. Girgis, J. Anat. *90*, 73 (1956).
[12] J. T. Dingle, J. A. Lucy and H. B. Fell, Biochem. J. *79*, 497 (1961).
[13] Supported by an N.I.H. grant of the Columbia University – American University of Beirut Nutrition Research Program. Miss Ardemis Khatcherian is thanked for her technical assistance.
[14] Present address: Department of Anatomy, Medical Center, West Virginia University, Morgantown, W. Va. 26506, USA.

Polytene Chromosomes in Silk Gland Cells of the Silkworm, *Bombyx mori*

It is well-known that the silk gland of the silkworm becomes bigger without increase in the number of cells, or without division of cells, in the advance of growth after hatching[1,2]. On the basis of the results obtained by electron microscopic autoradiography, Akai and Kobayashi[3] have suggested that a function of the chromatin in the nucleus of the silk gland is like that of the polytene chromosomes in the salivary gland of Diptera. However, the polytene chromosomes in the silk gland nucleus have not been observed by them. So far as we are aware, no adequate literature pertaining to this subject is accessible.



Figs. 1 and 2. Thick and distinct strands are seen running nearly parallel to the long axis of the silk gland nucleus of the silkworm. The strands, presumably polytene chromosomes, are twisted at many loci, showing differential coils along their length. Fig. 3. Metaphase chromosomes in a spermatogonium of the silkworm.