Group 1 exhibited considerable peroxidation and marked brown discoloration of the adipose tissue, changes which were accompanied by inhibition of enamel pigment deposition. The non-saponifiable fraction (Group 2) totally failed to produce such changes. There was no essential difference in the growth curves of the two groups, although both were somewhat subnormal.

Growth in Groups 3 and 4 was essentially normal, in contrast to the marked retardation of growth in Group 5. All offspring in this group died between 10 and 46 days of age following the development of anemia and diarrhea. This is interpreted as a toxic manifestation of the highly unsaturated fraction when given separately. The fact that animals receiving the highly unsaturated fraction (Group 5) developed marked fat peroxidation and discoloration about the same time as those receiving the total fatty acid fraction (Group 1) leaves no doubt that the highly unsaturated fatty acids are responsible for these changes. In Group 4 (middle unsaturated fraction) only one animal showed low fat peroxidation without discoloration at the time marked peroxidation and discoloration appeared in Groups 1 and 5. These changes appeared simultaneously with the rapid increase in fat deposition which was apparent at the beginning of the second month of life.

The deposition of the dental pigment took place at a normal rate in Group 3, whereas in Group 4 the deposition of the pigment was slower and did not reach the same intensity as compared with that of Group 3. This could be interpreted as a slight inhibition of the full development of the pigment due to the middle unsaturated acids or more likely to a small amount of highly unsaturated acids in the middle fraction. In Group 5 the inhibition of the pigment deposition was not so complete as that in Group 1, which received the total acids, for there was a slight and retarded deposition of pigment from the 33rd day to the 42nd, at which time the pigment disappeared completely from the upper incisors. The mandibular incisors of the animals which reached the 46th day of age were still slightly pigmented, a fact which confirms previous observations of a prolonged ability of the mandibular enamel organ to deposit the pigment.[4,13] This experiment demonstrates that the highly unsaturated fatty acids are responsible for the enamel depigmentation. Qualitative differences in the fatty acids of different fats, namely cod liver oil and lard, may account for the fact that both fats produce enamel depigmentation but only cod liver oil causes discoloration of the adipose tissue.[5] The question whether the lack of the antioxidant effect of vitamin E is responsible not only for the peroxidation and discoloration of fat but also for the dental depigmentation is still not clear.

Our examination of the chemical nature of the enamel pigment has eliminated the possibility of its being a lipochrome, porphyrin or melanin. It does not fluoresce and contains iron in the ferric form, which dissolves readily in 10 per cent. HCl. When exposed to a weaker acid (2 per cent. HCl) the pigmented layer comes off as a finely granular and fibrous film or layer. Incineration of the tooth in a gas flame separates the enamel from the dentin and leaves the pigmented layer a black color. After burning away the carbon with ammonium nitrate the yellow-brown color of the outer surface of the ashes from the enamel reappears, indicating that inorganic iron alone accounts for the total color of the pigment, which in the intact tooth appears to be embedded in or loosely combined with an organic matrix. Depigmented enamel of vitamin E-deficient rats failed to give reactions for iron with potassium ferro- or ferric-cyanide; when subjected to incineration it developed only a slight gray color, which turned again white after burning away the carbon with ammonium nitrate. Exposure of depigmented teeth to 2 per cent. HCl resulted in the splitting off of a much thinner, less compact and colorless film from the outer surface of the enamel. It is, therefore, also demonstrated that the highly unsaturated fatty acids in absence of vitamin E act on the enamel organ in such a way as to inhibit the deposition of the normal iron-containing layer, resulting in white appearance of the enamel surface.

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[13] H. Granados, K. E. Mason and H. Dam, *Jour. Dental Research* (Proc. 23rd general meeting International Assoc. for Dental Research), 1945. In press.

ARTERIAL ANASTOMOSIS IN DOGS EMPLOYING VEIN GRAFTS FROM CHICKENS AND TURKEYS

As a part of extended studies directed towards devising a ready source of veins for arterial replacement in animals and humans, vein grafts from young healthy chickens and turkeys were transplanted to bridge defects in the arteries of dogs. These experiments were planned on the basis of the findings of Copley that chicken thromboplastin prepared from brain and skin is ineffective or only slightly effective in activating thrombin in recalcified oxalate plasma of mammals.[1] We have since found that chicken thromboplastin as contained in vein juice is effective with whole dog's blood in arterial segments *in vivo* for the production of coagulation thrombosis.[2] Since

[1] A. L. Copley, *Am. Jour. Physiol.*, 137: 178–186, 1942.
[2] A. L. Copley and P. L. Stefko. To be published.

our experiments in utilizing vein segments from a living, heterologous source of a lower class exhibited unexpected results, they are being reported.

Chickens weighing 2.2 to 3.6 kgm were anesthetized by intraperitoneal injection of 2 cc Veterinary Nembutal—Abbott (sodium pentobarbital-alcohol mixture); turkeys, weighing 5 to 9 kgm, were injected intraperitoneally with 0.45 cc Veterinary Nembutal per kgm. After securing the fowl to the operating table, the neck was plucked, shaved and the skin prepared with a disinfectant. Following sterile draping of the neck, the midportion was exposed. Then the right jugular vein was excised following ligation with fine silk of venous branches, usually from 7 to 15 in number. The vein segment was irrigated thoroughly with physiologic saline, sometimes containing heparin.

The dogs used were healthy mongrels weighing from 15 to 25 kgm. They were anesthetized by intravenous injection of 0.45 cc Veterinary Nembutal per kgm. The femoral and carotid arteries of the dogs—in one case the aorta—were severed and bridged with the fowl's vein, using the one-tube and two-tube non-suture methods of Blakemore, Lord and Stefko.[3] Presumptive patency or occlusion was detected by palpation[3] of the vessel distal to the graft on various days following operation. Conclusive evidence was obtained by exploratory operation which terminated the experiment.

In Table 1 a brief summary of our findings is presented. In 37 heteroplastic grafts, 9 anastomoses were patent upon exploratory operation after 2 to 12 days, while partial or complete occlusion occurred in 24 instances and disappearance of the graft in 4 cases. Sloughing of the graft from one or both arterial ends was observed in 7 instances, 6 being associated with thrombosis, and one with disappearance of the graft. These results are significant, as in only a small number (10 out of 37 transplants) either sloughing or disappearance of the graft occurred. Thrombosis was observed only after several days of presumptive patency.

TABLE 1

SUMMARY OF EXPLORATORY FINDINGS OF VEIN TRANSPLANTS FROM CHICKENS AND TURKEYS IN THIRTY-SEVEN ARTERIAL ANASTOMOSES IN DOGS

Exploratory finding	Artery of dog	Jugular vein of fowl	Number of transplants	Day of exploration
Patency	Femoral	Chicken	5	2,6,7,8,12
	Carotid	"	3	2,4,5
	Aorta	Turkey	1	6
Occlusion (partial or complete)	Femoral	Chicken	11	5,7,7,7,9,10,12,14, 14,14,18
	Carotid	"	8	2,5,5,6,6,8,10,67
	"	Turkey	5	5,5,6,10,14
Slough from arterial end(s)*	Femoral	Chicken	4	7,8,12,14
	Carotid	"	2	10,67
	"	Turkey	1	14
Disappearance of graft	Femoral	Chicken	4	8,14,20,71

* The 7 transplants are recorded twice, since in 6 cases sloughing occurred with occlusion and in 1 case with disappearance of the graft.

Bacterial cultures of the vein segment made immediately after removal from the fowl and of the anastomotic site in the dog during the original operation were negative. All cultures of the vein graft, the anastomotic site and the thrombus made after exploratory operation showed bacterial contamination. It is quite possible that thrombosis in the majority of anastomoses was enhanced because of bacterial influences, and that the few cases of disappearance of the vein graft may also have been effected by bacteria. The precaution of employing aseptic technique may not be sufficient because of the common occurrence of transient bacteriaemias in animals in the absence of a traumatic lesion. This problem has been discussed by Haines.[4] It is possible that the infection of our vein transplants may have resulted from such a bacteriaemia.

In spite of the generally accepted contention that heterotransplants can not be expected to survive, the period of continued function of these transplants compares favorably with the work on autotransplants in dogs by Blakemore, Lord and Stefko.[3] These authors in 25 arterial anastomoses found 12 to be patent and 13 to have complete or partial occlusion on final exploration 2 to 69 days later. Further experimentation on heterotransplants in animals should be made before vein transplantation from the fowl to the human being can be considered. Such experimental work should include the combined use of sulfonamides or penicillin with heparin or other anticoagulants.

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[3] A. H. Blakemore, J. W. Lord, Jr. and P. L. Stefko, *Surgery*, 12: 488–508, 1942.

THE EFFECT OF PENICILLIN IN EXPERIMENTAL RABBIT SYPHILIS[1, 2, 3]

IN the November 3, 1944, issue of SCIENCE[4] I presented experiments dealing with the therapeutic effect of penicillin sodium dissolved in water and the same substance suspended in peanut oil. At that time my

[4] R. B. Haines, "Microbiology in the Preservation of Animal Tissues." Food Investigation, Special Report No. 45. London, His Majesty's Stationery Office, 1937.

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