Canad. M. A. J.
July 15, 1957, vol. 77

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RÉSUMÉ

On ne peut prétendre que l'infarctus dans le myocarde soit remplacé par du tissu de granulation sans donner une fausse impression de la réalité. A la région périphérique d'un infarctus se trouve une lisière de tissu dans laquelle le stroma survit. Dans cette lisière le muscle mort est rapidement absorbé, l'absorption étant complétée vers le dixième jour. L'endomysium qui reste se condense graduellement en perdant son liquide d'œdème, mais il n'y a aucune croissance de tissu conjonctif ou vasculaire qui pourrait former une cicatrice. La partie centrale de l'infarctus est absorbée plus lentement par les macrophages et sa trame d'endomysium est envahie par les fibroblastes et les cellules endothéliales. Les fibres réticulaires s'épaississent et deviennent hyalines avec le temps mais conservent leur orientation et peuvent être retracées dans les coupes transversales. Le tout illustre la remarquable capacité d'un organe à s'adapter à une infirmité avec relativement peu de perturbation dans sa structure et aucune interruption dans sa fonction. La période de repos devrait être déterminée beaucoup plus d'après l'évaluation clinique de la fonction cardiaque que d'après les altérations histologiques.

THE REVERSIBILITY OF ATHEROSCLEROSIS*

G. C. WILLIS, D.T.M.&H., M.R.C.P.(Edin.), F.R.C.P.[C.], *Montreal*

THE QUESTION whether or not it is possible for experimentally induced atherosclerotic plaques to be resorbed has been investigated several times. An early study by Anitschkow[1] indicated that withdrawal of cholesterol from cholesterol-fed rabbits was followed by a gradual disappearance of lipid from the plaques. In large plaques it took two to three years for this to occur. In a recent study by McMillan and his colleagues[2] along the same lines, histological reorganization of plaques was noted but no decrease in arterial lipid content was detected chemically in animals killed at intervals up to six months. Indeed, their work demonstrated that atherogenesis in the cholesterol-fed rabbit proceeds for some time after withdrawal of cholesterol from the diet. They attribute this to the persistence of hyperlipæmia, the etiological mechanism in this experimental procedure. This explanation would appear to be valid, as it is well recognized that atherosclerosis of cholesterol feeding is accompanied by extensive lipid deposits throughout the body, particularly in the reticulo-endothelial system. Except in such conditions as xanthomatosis, this state of cholesterol saturation has no counterpart in atherosclerosis in man.[3] It is not etiological in human atherosclerosis and does not offer a barrier to resorption of plaques in the way that it does in the cholesterol-fed rabbit.

Any approach to the study of resorption of atherosclerosis in experimental animals should thus have as its basis a method of inducing atherosclerosis without cholesterol feeding. As this has now become possible through the medium of scurvy in the guinea-pig,[4] ascorbic acid treatment of such animals forms an ideal means of studying the reversibility of atherosclerosis.

MATERIALS AND METHODS

A total of 77 male and female adult guinea-pigs was rendered scorbutic in the manner described in a previous communication.[4] After intervals of from 21 to 30 days, 50 of these animals were given ascorbic acid therapy and the remaining 27 were sacrificed. Ascorbic acid therapy consisted of a single intraperitoneal injection of 75 mg. of sodium ascorbate followed by the liberal addition of ascorbic acid powder to the basic scorbutogenic diet. The animals in this treated group were then sacrificed at intervals of time varying from 1 to 27 days.

Twelve additional animals employed as controls were placed on the scorbutogenic diet for 42 days, with powdered ascorbic acid liberally added from the beginning.

All animals were sacrificed by stunning and the thoracic aorta was dissected out and fixed in 10% formalin. Frozen sections were then made through the aortic arch and ascending and descending portions. As many sections as possible were obtained from each aorta and stained with Scharlach R for lipid.

The extent of deposition of stainable lipid in the aortic intima was graded as in the previous communication (i.e., + represented the earliest deposit of lipid, ++++ was solid filling of the intima with lipid, and ++ and +++ were intermediate). Careful note was made as to the morphology of the plaques under the different experimental circumstances.

RESULTS

No atherosclerosis was found in the controls